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RESEARCH ARTICLE

Influence of Obesity and Type 2 Diabetes on Chemotherapy Response in Ovarian Cancer Patients Receiving Carboplatin–Paclitaxel

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Abstract

Background and Aim: Ovarian cancer is one of the leading causes of cancer death in the gynecologic sector across the globe. There is growing evidence to indicate that obese patients and those with Type 2 diabetes mellitus (T2DM) could have a biological effect on tumors and chemotherapy outcomes. The aim of the study was to determine how obesity and T2DM affect inflammatory, glycemic, and tumor markers of women whose ovarian cancer has Stage II and who are treated with Carboplatin-Paclitaxel chemotherapy. **Methods:** A total of 105 women were recruited and classified into three groups: Group 1 (normal BMI, non-diabetic), Group 2 (obese and have Type 2 diabetes mellitus), and Group 3 (overweight and have Type 2 diabetes mellitus). Serum measurements made in the hs-CRP, IL-1β, CA-125, fasting plasma glucose (FPG), and HbA1c were measured once during the third chemotherapy cycle. The results were presented as Mean ± SE and compared across groups. **Results:** Group 2 and Group 3 had significantly higher levels of inflammatory markers (hs-CRP and IL-1β) and glycemic markers (FPG and HbA1c) than Group 1. Group 3 also depicted much greater glycemic values in comparison to Group 2. The groups were also much higher in CA-125 in the diabetic groups in comparison with Group 1. No statistically significant difference in CA-125 was, however, found between Groups 2 through 3, although both had higher values compared to Group 1. This trend could indicate a less significant decrease in tumor marker levels in the patients with a metabolic poor health state, which could indicate a less potent biochemical response to chemotherapy. Conversely, the CA-125 levels of Group 1 were nearer to the normal range, which means that the chemotherapy response was more promising. **Conclusion:** Systemic inflammation and poor glycemic control associated with obesity and T2DM in patients with ovarian cancer undergoing chemotherapy. The consistent increase of CA-125 in the diabetic populations regardless of the general treatment may indicate decreased chemotherapy response, which will validate the arising correlation between metabolic dysfunction and chemoresistance. An essential supplement to oncologic management may thus be metabolic health optimization.

Keywords: Ovarian cancer- Carboplatin–Paclitaxel- Obesity- CA-125- Inflammation

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Introduction

Ovarian cancer is considered to be among the leading causes of death associated with cancer in the gynecologic sphere, and the most frequently diagnosed form of ovarian cancer is epithelial [1]. Despite the development of surgical methods and systemic treatment, the majority of patients are diagnosed at a rather late stage of the disease, and the general prognosis is still not optimal [2]. The typical first-line chemotherapy regimen of ovarian cancer is usually a platinum-based regimen involving carboplatin and paclitaxel, which has been proven to enhance disease management and survival [3]. Nevertheless, chemotherapy response is extremely inconsistent in patients, and there is increasing evidence that host-linked metabolic factors, including obesity and Type 2 diabetes mellitus (T2DM),

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can have a big impact on treatment tolerance and oncologic findings [4].

Metabolic disorders can have an impact on the microenvironment of the tumor, platinum sensitivity, and survival outcomes in ovarian cancer. Inflammation and hyperglycemia caused by obesity may alter the signaling of cytokines and apoptosis, which may have an impact on chemotherapy. Nevertheless, the correlation between metabolic status and biochemical response after Carboplatin-Paclitaxel therapy on Stage II ovarian cancer is still not clearly defined [4].

Obesity is a significant health issue in the world and is a risk factor contributing to the occurrence and further development of a number of malignancies this including ovarian cancer. The adipose tissue has been identified as an active endocrine organ that can release adipokines and other inflammatory agents that promote chronic low-grade systemic inflammation, hormonal disequilibrium, and insulin resistance [5]. These changes could be the causes of ovarian carcinogenesis as well as tumor growth and resistance to therapy. Likewise, T2DM has been linked to more cancer and worse survival prognosis, which may be mediated through hyperinsulinemia and hyperglycemia and oxidative stress, and dysfunctional immune responses [6]. High levels of glucose and insulin in the circulation could stimulate tumor growth signaling pathways, and comorbidities associated with diabetes are also likely to complicate cancer treatment and recovery [7, 8].

Other clinical investigations have indicated that obese and diabetic cancer patients could have a worse chemotherapy response, high levels of treatment toxicity, and poor progression or overall survival relative to metabolically healthy patients [9, 10]. Moreover, obesity and T2DM are associated with metabolic inflammation, which is demonstrated by high levels of inflammatory biomarkers, including high-sensitivity C-reactive protein (hs-CRP) and pro-inflammatory cytokines like interleukin-1 beta (IL-1β) [11]. These indicators have been associated with unfavorable cancer outcomes and could be mechanistic intersections between metabolic inefficiency and tumor aggressiveness. CA-125 is a proven tumor marker used in the monitoring of disease burden syndrome and treatment response in ovarian cancer, though the interplay between CA-125 dynamics and metabolic conditions has not been well elucidated [12].

Thus, this research is expected to compare the effects of obesity and Type 2 diabetes mellitus on the inflammatory (hs-CRP and IL-1β) and glycemic (FPG and HbA1c) and tumor markers (CA 125) in women with Stage II ovarian cancer under Carboplatin-Paclitaxel chemotherapy. The present study aimed to shed more light on the metabolic-oncologic interface and its possible applicability in the prediction of chemotherapy response and clinical outcomes by analyzing the biomarker variation of normal-weight patients, overweight patients, and obese patients with diabetes.

Materials and Methods

Subjects and study design

Study design

This prospective observational study included 150 women aged 42–65 years with histologically confirmed FIGO Stage II ovarian cancer, all receiving standard Carboplatin–Paclitaxel chemotherapy, 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 [13]. Blood samples were obtained during the third chemotherapy cycle. Patients were recruited from specialized oncology centers in Baghdad, Iraq, between October 2024 and October 2025. Among the total cohort, 35 women with normal BMI (18.5–24.9 kg/m²) and without Type 2 diabetes mellitus were enrolled as the first study group. From the remaining patients with abnormal BMI values, 70 women with Type 2 diabetes mellitus were selected and divided into two additional groups: the second group consisted of 35 obese women (BMI ≥30 kg/m²), while the third group consisted of 35 overweight women (BMI 25.0–29.9 kg/m²). All enrolled women had Stage II ovarian cancer and were receiving the same chemotherapy regimen at the same treatment stage to ensure clinical homogeneity. The remaining 45 patients were excluded because they had ovarian cancer without Type 2 diabetes mellitus and with abnormal BMI values that did not fit the predefined study groups. Peripheral venous blood samples were collected to measure inflammatory markers [high-sensitivity C-reactive protein (hs-CRP) and interleukin-1 beta (IL-1β)], the ovarian tumor marker CA-125, and glycemic markers [fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c)]. All of the procedures, methodologies, and ways of sampling are proposed and were officially approved by the Central Scientific Committee in the College of Dentistry, Ibn Sina University of Medical and Pharmaceutical Sciences.

Sample Collection

Peripheral venous blood samples of 10 mL were obtained from all participants during the third cycle of Carboplatin–Paclitaxel chemotherapy, following an overnight fast of at least 8–10 hours. A total of approximately 10 mL of blood was collected under aseptic conditions. For glycemic markers, 3 mL of whole blood was collected into EDTA tubes for the determination of glycated hemoglobin (HbA1c), and 2 mL blood was collected into fluoride-oxalate tubes for the measurement of fasting plasma glucose (FPG). For inflammatory and tumor markers, 7 mL of blood was collected into plain gel tubes and allowed to clot at room temperature; samples were then centrifuged at 3000 rpm for 10 minutes to separate the serum. The serum obtained was aliquoted in sterile microtubes and stored at -20 °C awaiting analysis [14].

Inflammatory and Tumor Markers

The Architect i2000sr was used to measure the levels of serum High-sensitivity C-reactive protein (hs-CRP) and Cancer Antigen 125 (CA-125) with the help of the Chemiluminescent Microparticle Immunoassay (CMIA) technology. The concentration of Serum Interleukin-1 beta (IL-1β) was measured by using Human Interleukin IL-1β ELISA Kit (Bioassay Technology Laboratory, China). All immunoassays were done following the protocols of the manufacturers strictly.

Metabolic Parameters

Whole blood samples were used to determine the glycemic control. The High-Performance Liquid Chromatography (HPLC) method of measuring Glycated Hemoglobin (HbA1c) involved the D-10 Hemoglobin Testing System (Bio-Rad Laboratories, USA). The Architect c8000 clinical chemistry analyzer was used to assess the Fasting Plasma Glucose (FPG) level using the enzymatic hexokinase methodology (Abbott Laboratories, USA). All biochemical tests had internal quality controls that were upheld to guarantee the accuracy of data.

Statistical Analysis

The Statistical Analysis System (SAS, version 2018) was used to examine the statistical data. Continuous variables were 2-tested on their normal distribution and given in form of mean + standard error (SE). The Welch independent samples t-test was used to compare inter group data to allow for possible variance differences between the groups. Group 1 and Group 2, Group 1 and Group 3, and Group 2 and Group 3 were compared on a pairwise basis when comparing each biomarker (hs-CRP, I--1β, CA-125, FPG, and HbA1c). A p-value of less than < 0.05 was taken as statistically significant by two-tailed.

Results

A total of 105 women enrolled in the study with Stage II ovarian cancer undergoing chemotherapy with Carboplatin provided with the combination of Paclitaxel were used and divided into three groups of the study based on BMI and diabetic status: Group 1 (normal BMI, non-diabetic), Group 2 (obese with Type 2 diabetes), and Group 3 (overweight with Type 2 diabetes). The mean serum concentrations of inflammatory, glycemic, and tumor markers expressed as Mean ± SE, together with the results of independent-samples t-tests, are summarized in Tables 1-3

As shown in Table 1, women in Group 2 demonstrated significantly higher levels of hs-CRP and IL-1β compared with Group 1 ($p < 0.0001$ for both markers). Similarly, glycemic markers were markedly elevated in Group 2, with both FPG and HbA1c showing statistically significant differences relative to Group 1 ($p < 0.0001$). CA-125 levels were also significantly higher in Group 2 compared with Group 1 ($p < 0.0001$).

A comparable pattern was observed when Group 1 was compared with Group 3 (Table 2). Both inflammatory markers (hs-CRP and IL-1β) and glycemic markers (FPG and HbA1c) were significantly higher in Group 3 than in Group 1 ($p < 0.0001$ for all comparisons). Likewise, CA-125 levels were significantly elevated in Group 3 relative to Group 1 ($p < 0.0001$).

Direct comparison between the two diabetic groups (Group 2 vs Group 3) is presented in Table 3. Obese diabetic women (Group 2) had significantly higher hs-CRP and IL-1β levels than overweight diabetic women (Group 3) ($p < 0.0001$), indicating a stronger inflammatory state in association with higher BMI. In contrast, glycemic control was poorer in Group 3, as reflected by significantly higher FPG and HbA1c values compared with Group 2 ($p < 0.0001$). Importantly, there was no statistically significant difference in CA-125 levels between Group 2 and Group 3 ($p = 0.108$), despite both groups showing higher CA-125 levels than Group 1.

On the whole, the data described in Tables 1-3 suggest

Table 1. Comparison between Group 1 (Normal BMI, Non-Diabetic) and Group 2 (Obese, Type 2 Diabetes).

Marker	Group 1 (Mean ± SE)	Group 2 (Mean ± SE)	t-value	p-value
hs-CRP (mg/L)	1.79 ± 0.09	7.40 ± 0.21	−25.14	<0.0001
IL-1β (pg/mL)	3.73 ± 0.12	9.20 ± 0.18	−25.41	<0.0001
CA-125 (U/mL)	29.94 ± 1.15	85.06 ± 1.15	−33.89	<0.0001
FPG (mg/dL)	92.05 ± 0.67	164.32 ± 1.27	−50.25	<0.0001
HbA1c (%)	5.36 ± 0.03	8.04 ± 0.05	−46.66	<0.0001

Table 2. Comparison between Group 1 (Normal BMI, Non-Diabetic) and Group 3 (Overweight, Type 2 Diabetes).

Marker	Group 1 (Mean ± SE)	Group 3 (Mean ± SE)	t-value	p-value
hs-CRP (mg/L)	1.79 ± 0.09	5.05 ± 0.11	−23.76	<0.0001
IL-1β (pg/mL)	3.73 ± 0.12	6.82 ± 0.11	−18.77	<0.0001
CA-125 (U/mL)	29.94 ± 1.15	71.68 ± 0.99	−27.53	<0.0001
FPG (mg/dL)	92.05 ± 0.67	175.42 ± 1.61	−47.94	<0.0001
HbA1c (%)	5.36 ± 0.03	8.55 ± 0.06	−45.93	<0.0001

Table 3. Comparison between Group 2 (Obese, Type 2 Diabetes) and Group 3 (Overweight, Type 2 Diabetes)

Marker	Group 2 (Mean ± SE)	Group 3 (Mean ± SE)	t-value	p-value
hs-CRP (mg/L)	7.40 ± 0.21	5.05 ± 0.11	10.15	<0.0001
IL-1β (pg/mL)	9.20 ± 0.18	6.82 ± 0.11	11.19	<0.0001
CA-125 (U/mL)	85.06 ± 1.15	71.68 ± 0.99	1.63	0.108 (NS)
FPG (mg/dL)	164.32 ± 1.27	175.42 ± 1.61	−5.42	<0.0001
HbA1c (%)	8.04 ± 0.05	8.55 ± 0.06	−6.50	<0.0001

that obesity and Type 2 are linked to improved systemic inflammation and poor glycemic control among patients of ovarian cancer undergoing chemotherapy. Nonetheless, the levels of tumor markers seemed to be level between metabolically unhealthy patients, and no significant differences were observed between CA-125 in obese and overweight patients with diabetes.

Discussion

The current research paper examined how obesity and Type 2 diabetes impact the inflammatory, glycemic, and tumor marker outcomes among Stage II ovarian cancer patients receiving Carboplatin-Paclitaxel chemotherapy. The results showed that the levels of systemic inflammatory and glycemic indicators were significantly increased in both obese and overweight diabetic patients than those in normal-weight non-diabetic women, as indicated in Tables 1–3. According to these findings, metabolic impairment is significant in exacerbating inflammation and glycemic variability among patients undergoing oncologic treatment.

Obesity is progressively being identified as a condition of chronic low-grade inflammation, which is mediated by adipose tissue-derived cytokines and adipokines [15]. The levels of hs-CRP and IL-1β were significantly higher in both diabetic groups compared to normal-weight controls in the current cohort, which is in line with the idea that adiposity and insulin resistance are pro-inflammatory signaling events [16]. Further, the levels of inflammatory markers were considerably greater in the obese diabetic women than in the overweight diabetic women, suggesting a dose-response relationship between the BMI and the systemic inflammatory load [17]. This finding confirms previous studies indicating that obesity increases tumor-related inflammation, which could, in turn, affect aggression and treatment tolerance of the diseases [18].

Glycemic markers had a different but the same direction. Fasting glucose and HbA1c were both found to be significantly higher in diabetic women relative to non-diabetic patients, but overweight diabetic women were seen to have worse glycemic control as compared to obese diabetic women. This observation indicates that other factors other than BMI such as the duration of the disease, adherence to medication, and metabolic heterogeneity, are possible causes of glycemic instability among ovarian cancer patients undergoing chemotherapy [19, 20]. The lack of glycemic control has been linked to suppressed immune activity, augmented oxidative stress, as well as the abnormal growth and development of cells, which can all affect the conduct of tumors [6, 21].

A significant clinical finding in the current research is associated with the tumor marker CA-125, which is commonly known in the diagnosis of ovarian cancer, and also in monitoring the treatment response in chemotherapy [22]. As anticipated, the level of CA-125 was much lower and nearer to the normal range in normal-weight non-diabetic women, and this, with respect to Carboplatin-Paclitaxel therapy, would indicate a more positive biochemical outcome among normal-weight non-diabetic women. Conversely, despite the statistical significance of the difference in CA-125 levels in both diabetic groups relative to Group 1, there was no statistically significant difference in obese and overweight diabetic women. This absence of CA-125 difference between the two metabolically unhealthy groups can be a result of the fact that, after diabetes and metabolic hampering have been achieved, the tumor marker inhibition to chemotherapy is diluted [23, 24]. That is, the continuously high level of CA-125 in both diabetic groups would indicate a lesser tumor response to chemotherapy, and the approach to a close-to-normal level in Group 1 is in line with a more effective treatment response [25]. Such results indicate the hypothesis that the hyperglycemic, hyperinsulinemic, and chronically inflammatory conditions linked to Type 2 diabetes and obesity may be the cause of chemoresistance but not tumor regression [26]. Combined, the findings suggest that unregulated diabetes and adiposity can result in an impaired anticipated drop in CA-125 under chemotherapy, confirming the emerging literature in which metabolic dysregulation correlates with decreased sensitivity to chemotherapy in ovarian cancer [19, 27].

All these findings support the multifaceted interactions between metabolic health, systemic inflammation, and cancer biology. The fact that, at this stage of treatment, inflammatory and glycemic, but not CA-125, levels are significantly different between the two diabetic groups, indicates that metabolic stress can influence the host physiology to a greater extent than tumor marker kinetics [28]. Clinically, these findings highlight the significance of metabolic surveillance and optimization, especially glycemic regulation in the course of chemotherapy since inadequate metabolic state can negatively impact treatment outcomes, quality of life, and risk of complications.

This research is limited in a number of ways. The relatively small sample size (n = 35 per group) could have compromised the statistical power, and high the possibilities of type II error especially when there are no significant differences between groups like CA-125 levels

among obese and overweight diabetic patients.

Moreover, the observational design is single-centered and, therefore, does not lead to an easy generalization. To establish these, larger, multicenter longitudinal studies are required to establish the relationship between metabolic dysfunction and chemotherapy response in ovarian cancer.

Lastly, there was no assessment of clinical outcomes like the progression-free survival, overall survival, and radiologic response. The assessment of chemotherapy response was largely based on the CA-125 dynamics, which made it impossible to make conclusive results on the outcome of survival or chemoresistance. Further research that includes survival and imaging data should be implemented.

In conclusion, to summarize, the current evidence shows that Type 2 diabetes and obesity correlate with increased systemic inflammation and the deterioration of glycemic parameters in the patient with ovarian cancer under chemotherapy, and the levels of CA-125 do not differ significantly in diabetic subgroups with various BMI. These findings indicate the possible importance of incorporating metabolic evaluation in oncologic treatment to manage disease better.

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Ethics Statement

The protocol used in the research was approved by the Central Scientific Committee in the College of Dentistry, Ibn Sina University of Medical and Pharmaceutical Sciences.

Competing Interests

There are no financial and non-financial conflicts of interest between the authors.

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