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The effect of obesity on prognosis in patients with endometrioid type endometrial adenocarcinoma

DÖzcan Gül¹, DGüzin Demirağ²

¹Division of Immunology and Allergy, Department of Internal Medicine, Erzurum City Hospital, Erzurum, Turkiye ²Division of Medical Oncology, Department of Internal Medicine, Faculty of Medicine, Ondokuz Mayıs University, Samsun, Turkiye

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ABSTRACT

Aims: This study sought to investigate the relationship between pre-treatment body-mass index (BMI) and established prognostic factors associated with endometrial cancer, as well as to assess its impact on survival in patients diagnosed with this malignancy. **Methods:** Patients diagnosed with endometrioid-type endometrial cancer between January 2000 and June 2010 at the medical oncology clinic, and who received treatment and follow-up at our clinic, were included in the study. The patient files and the hospital electronic database were retrospectively reviewed. The patients were divided into non-obese (BMI <30 kg/m²) and obese (BMI \geq 30 kg/m²) groups, then compared in terms of clinical characteristics, pathological results, and survival outcomes. **Results:** A total of 126 patients, 44 in the non-obese group and 82 in the obese group, were included in the study. The two groups were statistically similar when classified according to demographic and clinicopathological data. Stage, tumour grade, cytology, depth of myometrial or lymphovascular invasion and lymph node metastasis were significantly associated with 5-year survival. The 5-year disease-free survival rate was 86.4% and 90.2%, respectively and no statistically significant association was observed between 5-years survival outcomes between both groups.

Conclusion: This study demonstrated that there was no significant relationship between obesity and the defined prognostic factors of the disease and 5-year survival results.

Keywords: Endometrial cancer, obesity, survival

INTRODUCTION

Endometrial cancer is one of the most prevalent types of gynecological cancer globally.¹ According to Globocan data, 417,000 new cases were diagnosed globally in 2020, with 97,000 deaths occurring.² According to the same data set, endometrial cancer ranks fourth among the most prevalent cancer types among Turkish women (incidence rate: 11.1 per 100,000). The World Health Organization also reported 5,463 new cases of endometrial cancer among Turkish women in 2018, with 1,051 deaths.²

Hormones are a significant factor in endometrial cancer, which occurs predominantly during the postmenopausal period.³ Obesity contributes to endometrial carcinogenesis through mechanisms such as inflammation, hyperinsulinemiainsulin resistance, and unopposed estrogen.⁴ In excessive adipose tissue, which has high aromatase activity, estrone is formed from androstenedione, and estradiol is formed from testosterone. Moreover, in obese women, the presence of hyperinsulinemia results in a decline in sex hormone-binding globulin (SHBG) levels. This, in turn, leads to an increase in free hormone levels, consequently exposing the endometrium to elevated estrogenic effects.^{5,6} Hyperestrogenemia and insulin resistance, in conjunction with hyperinsulinemia, have been demonstrated to enhance the systemic availability of insulin like growth factor-1 (IGF-1). This, in turn, triggers the activation of specific pro-oncogenic pathways that affect the endometrium.^{6,7} Moreover, obesity has been demonstrated to induce a state of chronic inflammation within the endometrium, featured by high levels of pro-inflammatory cytokines (IL-6, IL-8), thereby engendering a carcinogenic environment.⁷

Obesity, through the aforementioned mechanisms, has been identified as a serious threat for endometrial cancer development, with the incidence of this condition being 2-5 times higher in obese women.⁵ Each 5 kg/m² increase in bodymass index (BMI), the risk of endometrial cancer has been demonstrated to increase by 60%.⁸ Women with a BMI over 25 kg/m² face double the endometrial cancer risk, while those with a BMI exceeding 30 kg/m² can experience a threefold increase in risk.⁹ Despite the clear link between elevated BMI and a heightened likelihood of endometrial cancer, research

Corresponding Author: Özcan Gül, dr.ozcangul@gmail.com



reveals conflicting information regarding how BMI impacts survival rates for women after diagnosis.⁹⁻²¹ With the global increase in overweight and obesity, there is an urgent need for further research and awareness regarding the critical relationship between obesity and cancer outcomes. In view of the data presented, our study has two main aims. First, it aims to analyze the relationship between pre-treatment BMI and various established prognostic factors of the disease. The other is to investigate how pre-treatment BMI impacts survival outcomes for these patients.

METHODS

Approval for the present study was obtained from the Ondokuz Mayıs University Clinical Researches Ethics Committee (Date: 12.02.2016, Decision No: OMÜ-KAEK-2016/68). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. The study population comprised patients diagnosed with endometrioid-type endometrial cancer at the internal medicine medical oncology clinic from January 2000 to June 2010 and whose treatment and follow-up were performed in our center. The patient files and the hospital electronic database were retrospectively reviewed. We excluded patients who had not undergone hysterectomy. Data collected from patient files included age, BMI, parity, menopause status, International Federation of Gynecology and Obstetrics (FIGO) stage, tumour grade, size, myometrial and lymphovascular invasion, cytology, pathology results (metastasis to lymph nodes etc.), and details on surgical and adjuvant treatments. Information on recurrence and mortality was also obtained.

A total of 126 patients participated in the study, which divided them into two groups as non-obese (BMI <30 kg/m²) and obese (BMI \geq 30 kg/m²) based on their pre-treatment BMI. A comprehensive comparison was conducted between the groups concerning clinical characteristics, pathology results, and survival outcomes.

Statistical Analysis

The study used SPSS version 20 software for statistical analysis. We compared the average values of the groups using the student's T test and looked at frequency data with the Chi-square test. We calculated survival data using the Kaplan-Meier method and compared survival rates with the log-rank test. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

A total of 126 patients participated in the study, 44 were classified as non-obese, while 82 were classified as obese. The mean BMI for the non-obese group was 25.98 kg/m², with a range from 18.7 to 29.7 kg/m². In contrast, the mean BMI for the obese group was 36.75 kg/m², with a range from 30 to 56.6 kg/m².

The study examined various clinical characteristics of the patients, including age, BMI, parity, and menopausal status. In addition, we evaluated the pathological results that were determined to be important for the prognosis of the disease. The surgery performed and the subsequent treatment modalities were categorized for both groups. The analysis revealed no statistically significant difference between the groups (Table 1).

The average follow-up duration for the cohort was 46.7 months, with a range of 3 to 118 months. Throughout the follow-up period, 109 patients (86.5%) survived, while 17 patients (13.5%) passed away. Of these 17 patients, 14 (11.1%) died within the first 5 years following diagnosis. Additionally, 6 patients (4.8%) experienced recurrence, with 3 patients (2.4%) having a recurrence within the first 5 years. Of the 6 patients who had recurrences, 4 died, with one of these deaths occurring within the first 5 years post-diagnosis. The cohort analysis indicated that the 5-year disease-free survival rate was 87.3%, while the cause-specific survival rate was 88.9%.

When evaluating patients' clinical and pathological data with respect to 5-year survival; stage, tumour grade, cytology, myometrial invasion deeper than half, lymphovascular invasion, and metastasis to lymph nodes were statistically significant for survival, while no statistical significance was found for other parameters (Table 2).

When evaluating the effect of patients' BMI values and their classification into non-obese and obese groups on 5-year survival rates, the findings were as follows: In the non-obese group, which consisted of 44 patients, the 5-year disease-free survival rate was 86.4%. In contrast, the obese group, composed of 82 patients, had a survival rate of 87.8% and no statistically significant difference between the groups (p-value: 0.833) (**Figure A**). In terms of cause-specific survival, the rates were 86.4% for the non-obese group and 90.2% for the obese group, again showing no statistically significant difference (p-value: 0.545) (**Figure B**).



Figure. Kaplan-Meier curves comparing disease-free survival (A) and cause-specific survival (B) in patients with endometrioid-type endometrial cancer according to body-mass index

J Health Sci Med. 2025;8(3):383-388

| Table 1. Patients features | | | | | |
|---|---------------|-----------------------|------------------------------------|-------------------|---------|
| | | Case n (%) | Non-obese (<30 kg/m ²) | Obese (≥30 kg/m²) | p-value |
| | | 126 | 44 | 82 | |
| Age | ≤50 | 36 (28.6) | 12 (27.3) | 24 (29.3) | 0.813 |
| | >50 | 90 (71.4) | 32 (72.7) | 58 (70.7) | _ |
| Menopause | Premenopause | 27 (21.4) | 11 (25.0) | 16 (19.5) | 0.474 |
| | Postmenopause | 99 (78.6) | 33 (75.0) | 66 (80.5) | _ |
| Parity | 0 | 18 (16.2) | 6 (15.4) | 12 (16.7) | 0.861 |
| | ≥1 | 93 (83.8) | 33 (84.6) | 60 (83.3) | _ |
| Stage | 1-2 | 97 (77.0) | 30 (68.2) | 67 (81.7) | 0.086 |
| | 3-4 | 29 (23.0) | 14 (31.8) | 15 (18.3) | _ |
| Tumour grade | 1 | 44 (36.4) | 16 (36.4) | 28 (36.4) | 0.972 |
| | 2 | 65 (53.7) | 24 (54.5) | 41 (53.2) | _ |
| | 3 | 12 (9.9) | 4 (9.1) | 8 (10.4) | — |
| — • () | ≤20 | 12 (12.4) | 4 (12.1) | 8 (12.5) | 0.616 |
| Tumour size (mm) | >20 | 85 (87.6) | 29 (87.9) | 56 (87.5) | _ |
| Myometrial invasion | <1/2 | 64 (51.2) | 19 (43.2) | 45 (55.6) | 0.128 |
| | >1/2 | 61 (48.8) | 25 (56.8) | 36 (44.4) | — |
| | Negative | 78 (75.7) | 24 (66.7) | 54 (80.6) | 0.116 |
| Lymphovascular invasion | Positive | 25 (24.3) | 12 (33.3) | 13 (19.4) | _ |
| Cytology | Negative | 102 (95.3) | 36 (94.7) | 66 (95.7) | 0.585 |
| | Positive | 5 (4.7) | 2 (5.3) | 3 (4.3) | — |
| Lymph node metastasis | No | 96 (80.7) | 33 (76.7) | 63 (82.9) | 0.414 |
| | Yes | 23 (19.3) | 10 (23.3) | 13 (17.1) | — |
| PLND | Not done | 31 (25.6) | 10 (23.3) | 21 (26.9) | 0.658 |
| | Done | 90 (74.4) | 33 (76.7) | 57 (73.1) | _ |
| PALND | Not done | 51 (42.1) | 18 (41.9) | 33 (42.3) | 0.962 |
| | Done | 70 (57.9) | 25 (58.1) | 45 (57.7) | _ |
| | Not done | 50 (39.7) | 13 (29.5) | 37 (45.1) | 0.117 |
| | Radiotherapy | 47 (37.3) | 17 (38.6) | 30 (36.6) | _ |
| Adjuvant therapy | Chemotherapy | 16 (12.7) | 6 (13.6) | 10 (12.2) | _ |
| | CT+RT | 13 (10.3) | 8 (18.2) | 5 (6.1) | _ |
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DISCUSSION

Obesity is a prevalent and significant concern among women diagnosed with endometrial cancer, as numerous studies have consistently demonstrated that obesity substantially increases the risk of developing endometrial cancer and underscored the importance of managing this risk factor.²²⁻²⁴ However, the impact of obesity on the prognosis of the disease-specifically how it interacts with other critical prognostic factors-remains less clear, despite the large body of research available in the literature.^{9-21,25} Undoubtedly, identifying and accurately evaluating the risk factors and clinicopathological features that adversely affect the prognosis of endometrial cancer are crucial. A deeper understanding of these factors will be critical for tailoring more effective treatment strategies and follow-up protocols, ultimately improving survival rates and patient outcomes in this common cancer type.

In our study, we evaluated clinical characteristics, such as age, parity, and menopausal status. Our findings indicated

that there were no statistically significant difference between non-obese individuals (BMI <30 kg/m²) and those classified as obese (BMI ≥30 kg/m²). We conducted an assessment of pathological outcomes, which included FIGO staging, tumour grading, tumour dimensions, myometrial or lymphovascular invasion, cytological findings, lymph node metastasis, and the surgical as well as subsequent adjuvant treatment strategies employed. When each prognostic factor was assessed independently, survival outcomes were consistent with those reported in the literatüre.²⁶⁻²⁸ Notably, however, our multivariate analysis revealed that there were no significant difference in 5-year disease-free survival or cause-specific survival rates between the groups in relation to obesity.

A thorough review of existing literature highlights a notable disparity in the findings concerning the relationship between obesity and survival outcomes. Some studies support our

| Table 2. Prognostic factors and survival of p | patients | | | | | | |
|---|----------------------------|------------------------|----------------------------|------------------|----------------|-------------------------|--|
| | | Disease-free survival | | | Cause spesific | Cause spesific survival | |
| | | Case n (%) | 5-year DFS (%) | p-value | 5-year CSS (%) | p-value | |
| Age | ≤50 | 36 (28.6) | 94.4 | 0.114 | 94.4 | 0.191 | |
| | >50 | 90 (71.4) | 84.4 | | 86.7 | | |
| Menopause | Premenopause | 27 (21.4) | 92.6 | 0.346 | 92.6 | 0.480 | |
| Wenopause | Postmenopause | 99 (78.6) | 85.9 | | 87.9 | | |
| Darity | 0 | 18 (16.2) | 83.3 | 0.640 | 88.9 | 0.967 | |
| ranty | ≥1 | 93 (83.8) | 89.2 | | 89.2 | | |
| $PMI(lra/m^2)$ | <30 | 44 (34.9) | 86.4 | 0.833 | 86.4 | 0.545 | |
| bivii (kg/iii) | ≥30 | 82 (65.1) | 87.8 | | 90.2 | | |
| Store | 1-2 | 97 (77.0) | 91.8 | 0.005 | 93.8 | 0.001 | |
| Stage | 3-4 | 29 (23.0) | 72.4 | | 72.4 | | |
| | 1 | 44 (36.4) | 93.2 | 0.018 | 95.5 | 0.006 | |
| Tumour grade | 2 | 65 (53.7) | 87.7 | | 89.2 | | |
| | 3 | 12 (9.9) | 66.7 | | 66.7 | | |
| There are the (mark) | ≤20 | 12 (12.4) | 100 | 0.178 | 100 | 0.209 | |
| i uniour size (mm) | >20 | 85 (87.6) | 85.9 | | 88.2 | | |
| Maron strict investign | <1/2 | 64 (51.2) | 95.3 | 0.004 | 95.3 | 0.015 | |
| Myometriai mvasion | >1/2 | 61 (48.8) | 78.7 | | 82.0 | | |
| | Negative | 78 (75.7) | 89.7 | 0.019 | 92.3 | 0.004 | |
| Lymphovascular invasion | Positive | 25 (24.3) | 72.0 | | 72.0 | | |
| | Negative | 102 (95.3) | 90.2 | 0.073 | 92.2 | 0.023 | |
| Cytology | Positive | 5 (4.7) | 60.0 | | 60.0 | | |
| | No | 96 (80.7) | 91.7 | 0.009 | 93.8 | 0.002 | |
| Lymph node metastasis | Yes | 23 (19.3) | 73.9 | | 73.9 | | |
| | Not done | 31 (25.6) | 83.9 | 0.413 | 87.1 | 0.502 | |
| PLND | Done | 90 (74.4) | 91.1 | | 92.2 | | |
| | Not done | 51 (42.1) | 84.3 | 0.290 | 86.3 | 0.221 | |
| PALND | Done | 70 (57.9) | 92.9 | | 94.3 | | |
| | Not done | 50 (39.7) | 92.0 | 0.162 | 94.0 | 0.113 | |
| Adjuvant therapy | Done | 76 (60.3) | 84.2 | | 85.5 | | |
| DFS: Disease-free survival, CSS: Cause spesific survival, B | MI: Body-mass index, PLND: | Pelvic lymph node diss | ection, PALND: Para-aortic | lymph node disse | ection | | |

conclusions, demonstrating no significant association between obesity and survival rates.¹⁵⁻¹⁹ Conversely, some studies suggest a troubling correlation between increased BMI and heightened mortality risk.^{9-14,25} For example, Secord et al.²⁵ reported that a 10% rise in BMI was linked to a 9.2% increase in mortality.

While numerous studies have associated obesity with poorer survival outcomes, some studies have highlighted the so-called 'obesity paradox,' where higher BMI appears to be associated with better survival in specific patient populations.^{20,21} Temkin et al.,²¹ in a multicenter retrospective study, found that women with a BMI \geq 30 had an average survival of 117 months, whereas those with a BMI <25 had a survival of 85 months (p: 0.003). The authors proposed several potential explanations for this paradoxical finding. One key factor is the younger age at diagnosis often seen in obese patients, which may result in a longer overall survival period. Additionally, obese individuals

tend to present with lower tumour grades and earlier stages of cancer, both of which are associated with better prognoses. Another factor worth considering is the clinical tendency to administer reduced doses of postoperative chemotherapy to obese patients, which could, paradoxically, lead to improved survival outcomes due to reduced toxicity.

The findings from studies investigating the effect of obesity on survival outcomes have been inconsistent, likely due to differences in study design, patient selection, and the BMI cutoffs used for categorization. In a study by Gates et al.,²⁹ which involved 165 patients diagnosed with endometrioidtype endometrial carcinoma, those with a BMI greater than 25 kg/m^2 were found to have more favorable prognostic factors than those with a lower BMI. On the other hand, when the BMI cutoff was set at \geq 35 kg/m² (class 2 obesity) or \geq 40 kg/m² (morbid obesity), the more obese groups demonstrated worse prognostic factors and lower survival rates.

Limitations

Similarly, the findings of our study may have influenced by the BMI cutoff (\geq 30 kg/m²) employed to delineate the patient groups. Had the cutoff been set according to class 2 obesity or morbid obesity, the results might have shown a different relationship. This represents an inherent limitation of our study. Other limitations of the study include its retrospective nature, which inherently restricts the ability to draw definitive conclusions about causality; a small sample size, particularly in subgroups; a lack of data on BMI changes following treatment; the absence of data about metabolic parameters or obesity-related comorbidities; and the inclusion of singlecenter data, which may reduce generalizability.

CONCLUSION

In conclusion, the study demonstrates that pre-diagnosis obesity does not impact the survival of Turkish women with endometrioid-type endometrial adenocarcinoma. However, the role of obesity in the prognosis of non-endometrioid subtypes remains unclear and warrants further research to better understand its impact. Additionally, the effects of changes in BMI post-diagnosis, along with any related interventions, on long-term survival in endometrial cancer warrant further investigation. These areas could represent important opportunities for future research, particularly in understanding the potential benefits of weight management interventions in improving patient outcomes.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Ondokuz Mayıs University Clinical Researches Ethics Committee (Date: 12.02.2016, Decision No: OMÜ-KAEK-2016/68).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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