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Original Research

Metabolic Syndrome and Ovarian Cancer Staging: A Retrospective Analysis of Chinese Patients

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Abstract

Background: Metabolic syndrome (MetS) has been associated with various cancers, including breast and endometrial cancer. However, its role in the severity of ovarian cancer remains unclear. This study aimed to investigate the association between MetS and the severity of ovarian cancer. **Methods**: A retrospective analysis was performed on 309 ovarian cancer patients hospitalized at Beijing Friendship Hospital from January 2013 to January 2024. The Chinese Diabetes Society criteria were used to define MetS. The association between ovarian cancer severity and MetS was evaluated using multivariable logistic regression models. **Results**: The overall prevalence of MetS in ovarian cancer was 15.69% (48/306), with 15% (17/113) in Stage I, 10% (3/31) in Stage II, 17% (18/103) in Stage III, and 17% (10/59) in Stage IV. After adjusting for confounding variables (fully adjusted Model 3), logistic regression analysis showed that ovarian cancers in Stage II (odds ratio (OR): 0.814, 95% confidence interval (CI): 0.192–3.440), Stage III (OR: 1.121, 95% CI: 0.478–2.629), and Stage IV (OR: 1.315, 95% CI: 0.479–3.609) was not significantly associated with MetS compared to Stage I. **Conclusion**: MetS was not found to be associated with ovarian cancer severity. Further prospective studies are needed to determine the causal relationship between MetS and ovarian cancer severity.

Keywords: metabolic syndrome; ovarian cancer; obesity; hypertension; hyperlipidemia; abnormal lipid metabolism; hyperglycemia/type 2 diabetes; insulin resistance

1. Introduction

Modern lifestyle and dietary habits have changed greatly in the past 30 to 40 years, from a relatively small incidence of cancer to the scales and types of malignant tumors currently diagnosed. Aggregate exposure, characterized by obesity, hypertension, and hyperlipidemia are risk factors for cancer and metabolic syndrome (MetS) is a general term for metabolic disorders related to obesity, abnormal lipid metabolism, and hypertension hyperglycemia/type 2 diabetes. Insulin resistance (IR) leads to hyperinsulinemia, which in turn produces a series of metabolic disorders that can lead to atherosclerosis. Currently, the average global incidence of MetS is 13.61%, while the incidence in China is 14%–18%. The study has confirmed that the various malignant tumor ocurrences are related to MetS [1].

MetS increases the risk of various gynecological cancers in women. Dong *et al*. [2] demonstrated that MetS and its related components significantly impact the initiation, progression, treatment response and prognosis forbreast cancer. Through a systematic review and meta-analysis of 17,772 endometrial cancer cases, Wang *et al*. [3] found that MetS was significantly associated with endometrial cancer risk (odds ratio (OR): 1.62; 95% confidence interval (95% CI) = 1.26–2.07). In a large-scale study of East Asian women, Park *et al*. [4] discovered that women with obesity combined with MetS had a significantly increased risk of endometrial cancer (hazard ratio (HR) = 2.18). Regarding the relationship between MetS and ovarian cancer, a recent systematic review and meta-analysis by Chen *et al*. [5] that included five studies, showed no significant correlation between MetS and ovarian cancer risk (OR = 1.29, 95% CI: 0.90–1.84), although significant associations were found in certain subgroup analyses. Most existing research on MetS and ovarian cancer has primarily focused on the overall risk of developing the disease rather than its severity or stage. Additionally, many studies do not stratify analyses by tumor grade or stage, which limits the understanding of how individual MetS components may differentially impact ovarian cancer progression [1,5]. For instance, while studies like Chen *et al*. [5] have explored the association between MetS and ovarian cancer risk, they do often not investigate how specific MetS components such as obesity, hyperglycemia, or hypertension influence the severity or stage of the disease. This gap underscores the need for more nuanced research that examines the differential impacts of MetS components on ovarian cancer severity, particularly within diverse populations such as the Chinese cohort examined in this study. Given the increased prevalence of MetS in the Chinese population and the gaps identified in existing research—particularly the limited focus on disease severity and the lack of stratified analyses by tumor stage—the current cross-sectional study aimed to investigate the relationship between MetS and ovarian cancer severity in

a Chinese adult population. By addressing the foregoing shortcomings, it sought to provide a more comprehensive understanding of the metabolic factors influencing ovarian cancer progression and outcomes in this demographic.

2. Materials and Methods

2.1 Study Population

Data were collected between January 2013 and January 2024 from the Obstetrics and Gynecology Center of Beijing Friendship Hospital, which is affiliated with the Capital Medical University. Inclusion criteria: Histologically confirmed ovarian cancer, complete clinical and pathological data, age $\geq$18 years. Exclusion criteria: Previous history of other malignancies, incomplete metabolic parameter data, patients who received neoadjuvant chemotherapy.

A total of 309 patients with ovarian cancer were pathologically diagnosed through surgery, three patients without lipid data were excluded and 306 patients were enrolled. These patients were classified into Stage I (113 cases), Stage II (31 cases), Stage III (103 cases), and Stage IV (59 cases) according to the Chinese Gynecological Oncology Clinical Practice Guidelines (2023 edition) for operation-pathological staging of ovarian cancer, fallopian tube cancer and peritoneal cancer (International Federation of Gynecology and Obstetrics (FIGO), 2013). The study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee of Beijing Friendship Hospital (Approval No. 2025-P2-073-01). Informed consent was obtained from all participants prior to inclusion in the study.

2.2 Definition of MetS

MetS is defined by the Chinese Diabetes Society [6] as having three or more of the following abnormalities: (1) Body mass index (BMI) $\geq$25.0 kg/m^2; (2) Patients with fasting plasma glucose (FPG) $\geq$6.1 mmol/L or 2-hour plasma glucose $\geq$7.8 mmol/L and/or diagnosed diabetes mellitus and treatment; (3) Systolic blood pressure $\geq$140 mmHg or diastolic blood pressure $\geq$90 mmHg and/or diagnosed and treated for hypertension; (4) Triglycerides (TG) $\geq$1.7 mmol/L and/or high-density lipoprotein cholesterol (HDL-C) $<$1.0 mmol/L

2.3 Diagnostic of Ovarian Cancer

Diagnosis of ovarian cancer was based on FIGO's staging classification for cancer of the ovary, fallopian tube and peritoneum.

2.4 Other Covariates

Demographic data included age, marital status, nationality, medical insurance status, work status, menopause history, fertility history, genetic history and chronic history. The standard method was used to measure height and body mass. Lineage accounts for genetic and sociocultural factors that may affect disease prevalence and progression. Medical insurance status served as a proxy for access to healthcare services, impacting the management and outcomes of both MetS and ovarian cancer. Full-time employment is related to socioeconomic status, lifestyle factors, and stress levels. The average value of three measurements was taken as the final value, with blood pressure measured in a supine or sitting position after 15 minutes of quiet rest. After fasting for 8–12 h, blood samples were collected intravenously for detection of hemoglobin, alanine aminotransferase, aspartate aminotransferase, fasting blood glucose, uric acid, total cholesterol, triglyceride, low density lipoprotein and high density lipoprotein levels. The BMI was calculated using the formula: weight in kilograms divided by the square of height in meters (kg/m^2).

2.5 Statistical Analysis

The data were analyzed using SPSS version 26.0 software (IBM Corp., Armonk, NY, USA). Categorical and continuous variables are presented as percentages and mean $\pm$ standard deviation (SD), respectively. Data normality was assessed using the Shapiro-Wilk test. Chi-square tests were used for categorical variables when all expected frequencies were $\geq$5; Fisher's exact test was used when expected frequencies were $<$5. Student's *t*-test was used for normally distributed continuous variables and the Mann-Whitney U test for non-normally distributed continuous variables. Multivariate logistic regression analysis was used to examine the cross-sectional association between MetS and ovarian cancer severity. Based on potential confounders, different models were listed to assess their impact on the association between MetS and ovarian cancer severity. Model 1 included basic demographic variables (age, marital status, race, medical insurance and full-time employment). Model 2 added clinical variables (postmenopausal, reproductive history and family history of ovarian cancer) to Model 1. Model 3 further included health-related variables (Chronic disease, allergic diseases, hemoglobin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and uric acid) to Model 2. OR with associated 95% CI are shown to estimate the association. All significance tests were two-tailed and statistical significance was assumed at $p < 0.05$.

3. Results

3.1 Baseline Characteristics of Participants

Table 1 shows the baseline characteristics of the total sample (MetS and non-MetS). A total of 306 hospitalized patients were included in the study. The mean age of this sample population was 55 $\pm$ 12 years, 274 (90%) were married, 290 (95%) had medical insurance, 238 (78%) had stable work, 214 (70%) were postmenopausal, 269 (88%) had a reproductive history, 6 (2.0%) had a family history of ovarian cancer, 154 (50%) had chronic disease and 40 (13%) had allergic diseases. Among all the participants, the

Table 1. Baseline characteristics of participants.

Variable	Overall (n = 306)	No MetS (n = 258)	MetS (n = 48)	Test	*p*-value
Age (Mean ± SD) (years)	55.07 ± 12.01	54.21 ± 12.18	59.71 ± 9.88	$t = -2.950$	0.003*
Marital status				$\chi^2 = 0.274$	0.600
Unmarried, n (%)	32 (10)	28 (11)	4 (8)		
Married, n (%)	274 (90)	230 (89)	44 (92)		
Race				Fisher's	>0.999
Han, n (%)	303 (99)	255 (99)	48 (100)		
Non-Han, n (%)	3 (1)	3 (1)	0 (0)		
Medical insurance				Fisher's	0.086
Yes, n (%)	290 (95)	242 (94)	48 (100)		
No, n (%)	16 (5)	16 (6)	0 (0)		
Full-time employment				$\chi^2 = 0.254$	0.614
Yes, n (%)	238 (78)	202 (78)	36 (75)		
No, n (%)	68 (22)	56 (22)	12 (25)		
Postmenopausal, n (%)	214 (70)	173 (67)	41 (85)	$\chi^2 = 6.490$	0.011*
Reproductive history, n (%)	269 (88)	225 (87)	44 (92)	$\chi^2 = 0.756$	0.384
Family history of ovarian cancer	6 (2)	6 (2)	0 (0)	Fisher's	0.595
Chronic disease, n (%)	154 (50)	110 (43)	44 (92)	$\chi^2 = 38.919$	<0.001***
Allergic diseases, n (%)	40 (13)	37 (14)	3 (6)	$\chi^2 = 2.332$	0.127
Hemoglobin (Mean ± SD)	125.37 ± 15.56	124.86 ± 15.61	128.15 ± 15.15	$t = -1.346$	0.179
ALT (Mean ± SD)	16.10 ± 10.39	15.87 ± 10.14	17.35 ± 11.67	$t = -0.907$	0.365
AST (Mean ± SD)	21.81 ± 17.29	21.80 ± 18.57	21.84 ± 7.36	$t = -0.015$	0.988
Uric acid (Mean ± SD)	281.36 ± 81.87	278.63 ± 82.25	295.84 ± 79.07	$t = -1.338$	0.182
Staging of ovarian cancer				$\chi^2 = 1.202$	0.753
Stage I, n (%)	113 (37)	96 (37)	17 (35)		
Stage II, n (%)	31 (10)	28 (11)	3 (6)		
Stage III, n (%)	103 (34)	85 (33)	18 (38)		
Stage IV, n (%)	59 (19)	49 (19)	10 (21)		

Notes: Continuous variables are presented as mean ± standard deviation. Categorical variables are presented as number of cases (percentage). ***$p < 0.001$. *$p < 0.05$.

ALT, alanine aminotransferase; AST, aspartate aminotransferase; SD, standard deviation; MetS, metabolic syndrome.

prevalence of MetS was 15.69%. Overall, MetS inpatients were significantly older than non-MetS inpatients (59.71 ± 9.88 years *vs.* 54.21 ± 12.18 years, respectively; $p < 0.05$), with a higher percentage of patients who have given birth. Postmenopausal inpatients had a significantly higher percentage of MetS than those postmenopausal inpatients (85% *vs.* 67%, $p < 0.05$). Additionally, inpatients patients with chronic diseases were significantly more likely to have MetS ($p < 0.05$).

3.2 Association between MetS and Ovarian Cancer Severity

Table 2 shows the results of the multivariate logistic regression analysis on the association between MetS and ovarian cancer severity. Model 1, after adjusting for age, marital status, race, medical insurance and full-time employment, showed that MetS was not significantly associated with ovarian cancer severity. Model 2, after adjusting for age, marital status, race, medical insurance, full-time employment, postmenopausal, reproductive history and family history of ovarian cancer, showed that MetS was not significantly associated with ovarian cancer severity. In the fully adjusted Model 3, the results remained unchanged and MetS was not significantly associated with ovarian cancer severity.

4. Discussion

In this study, the prevalence of MetS in ovarian cancer for Chinese people was found to be 15.69%, but MetS was not significantly associated with ovarian cancer severity. This was unaffected following adjustment for potentially confounding variables.

4.1 Comparison with Other Studies

Ovarian cancer continues to be the deadliest gynecologic malignancy. Patients with both diabetes mellitus and obesity have poorer outcomes, yet research correlating metabolic abnormalities such as MetS to ovarian cancer risk, severity and outcomes is lacking [7]. Esposito *et al.* [8] demonstrated a borderline correlation between MetS and epithelial ovarian cancer (EOC) in a study that included 654 cases of ovarian cancer from two European cohort studies. Bjørge *et al.* [9] found an increased risk of ovarian

Table 2. Associations between metabolic syndrome and risk of ovarian cancer.

Characteristic	Model 1		Model 2		Model 3	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
Ovarian cancer (stage)						
I	1.000	Ref	1.000	Ref	1.000	Ref
II	0.532 (0.138–2.051)	0.360	0.515 (0.133–1.991)	0.336	0.814 (0.192–3.440)	0.779
III	0.931 (0.438–1.978)	0.852	0.951 (0.447–2.026)	0.897	1.121 (0.478–2.629)	0.793
IV	0.903 (0.371–2.195)	0.822	0.918 (0.374–2.255)	0.853	1.315 (0.479–3.609)	0.595

Notes: OR, odds ratio; 95% CI, 95% confidence interval; Ref, reference.

Model 1: Age, marital status, race, medical insurance full-time employment.

Model 2: Model 1 + Postmenopausal, reproductive history family history of ovarian cancer.

Model 3: Model 2 + Chronic disease, allergic diseases, hemoglobin, ALT, AST, uric acid.

cancer mortality in women with MetS who were under 50 years old. However, most studies of MetS components and ovarian cancer do not present analysis stratified by tumor grade.

Here, components such as blood glucose levels and blood pressure did not show significant associations with cancer stages. This may explain the overall lack of association between MetS and ovarian cancer severity observed in this study. Wu *et al*. [10] conducted a comparative study between China and the global population, highlighting that while hyperglycemia contributes to the disease burden of ovarian cancer, its direct association with cancer severity may vary across populations. This suggests that the influence of specific MetS components on cancer progression may differ based on regional and genetic factors.

The relationship between the components of MetS and the incidence of ovarian cancer has been studied with controversial results, while the severity of ovarian cancer has been little studied. Nevertheless, obesity, type II diabetes mellitus and MetS have been associated with poor outcomes in epithelial ovarian cancer [7]. Studies have demonstrated increased EOC risk in patients with obesity [11,12]. Women with a BMI >25 kg/m^2 have a higher incidence of epithelial ovarian cancer when compared to their normal weight counterparts. There was increased risk of EOC for incremental increases in BMI [11]. The time that obesity develops during a woman's life may impact her ovarian cancer risk. Multiple studies have demonstrated that an elevated BMI in adolescence/early adulthood increases the subsequent risk for epithelial ovarian cancer [13,14]. Among postmenopausal women, Liu *et al*. [14] found no relationship between overweight/obesity and ovarian cancer. Michels *et al*. [15] found women over the age of 65 with MetS had reduced ovarian cancer risk when compared to women not meeting the same diagnostic criteria, whereas, analysis from a pathological perspective has demonstrated obesity is associated with endometrioid ovarian tumors [12].

The relationship between high triglycerides and ovarian cancer risk is inconsistent across large prospective studies. Effect magnitudes from the Me-Can study for high triglycerides and risk of serous tumors were not statistically significant, while baseline hypertension enhanced the likelihood of endometrioid cancers [16]. Whereas, Zhang *et al*. [17] have demonstrated a link between high blood cholesterol and increased risk of ovarian cancer. Huang *et al*. [18] found diabetes increased risk for ovarian cancer in their meta-analysis. In those with early stage disease, Shah *et al*. [13] found diabetic patients have a poorer prognosis. However, this study also demonstrated that diabetics were less likely to have either stage I or IV disease than their non-diabetic counterparts. In the Me-Can study [16], reduced ovarian cancer risk with high blood glucose was suggested. However, in the Women's Health Initiative, ovarian cancer was not associated with glucose and insulin levels.

Comparing the findings reported here with those from other regions, several key differences and similarities emerge. Discrepancies may be attributed to genetic, lifestyle and environmental differences between populations. Asian populations, including the Chinese, often have different body composition profiles and metabolic responses when compared to Western populations, which could influence the impact of MetS components on cancer progression [19].

Moreover, regional variations in healthcare access, diagnostic practices and treatment protocols may also contribute to differing outcomes. In China, the prevalence of certain MetS components, such as elevated BMI, might manifest differently in relation to ovarian cancer compared to other regions. Additionally, cultural factors influencing diet, physical activity and healthcare-seeking behaviors could play a role in modulating the relationship between MetS and ovarian cancer severity [8].

The findings reported here are consistent with the Asian study that also reported limited associations between MetS and ovarian cancer severity, suggesting that the impact of MetS may vary across different ethnic and regional contexts [20]. However, the limited number of studies in Asian populations underscores the need for more extensive research, both to validate the results presented here and explore the underlying mechanisms driving these regional differences.

4.2 Potential Mechanisms

Ovarian cancer, like other malignant tumors, is a disease of abnormal cell proliferation. These abnormal uncontrolled cells grow, infiltrate and spread to other tissues, requiring a lot of energy and nutrients to support their rapid growth and mobility. Therefore, patients with malignant tumors often have metabolic abnormalities. Simultaneously, some metabolic diseases such as obesity, hyperlipidemia, diabetes, also increase the risk of malignant tumors by affecting the processes of cell growth, proliferation and apoptosis. MetS increases the risk of several cancers [21]. The mechanisms linking MetS and ovarian cancer incidence and progression are incompletely understood. A variety of pathologic factors may contribute to cancer risk in patients with metabolic derangements, including altered adipokine and cytokine expression, altered immune responses to tumor cells and changes in pro-tumorigenic signaling pathways [7]. Chronic low-grade inflammation, a symptom of MetS, further exacerbates these processes by promoting an environment conducive to cancer progression [22]. Insulin resistance and hyperinsulinemia, common in MetS, activate insulin-like growth factor signaling pathways, which have been implicated in enhancing cancer cell proliferation and inhibiting apoptosis [22]. Moreover, estrogen and endocrine disorders associated with MetS influence ovarian cancer development and progression by altering hormonal balances that regulate cell growth and differentiation [23].

Adipocytokines, such as leptin and adiponectin, serve as key molecular mediators linking MetS and ovarian cancer. Leptin, elevated in obese individuals, promotes ovarian cancer cell proliferation and invasion by activating signaling pathways such as JAK/STAT and PI3K/Akt [23]. Conversely, adiponectin, typically reduced in obesity, has antiproliferative and pro-apoptotic effects on ovarian cancer cells, highlighting the complex interplay between different adipokines and cancer biology [23]. These molecular interactions underscore the importance of metabolic health in modulating cancer progression and outcomes.

Furthermore, hyperglycemia and insulin resistance, components of MetS, contribute to a tumor-promoting environment by increasing the availability of glucose for rapidly dividing cancer cells and by activating insulin growth factor pathways signaling pathways that facilitate cancer cell survival and growth [22,24]. Dyslipidemia, another MetS component, influences cancer progression through lipid metabolism alterations that affect cell membrane synthesis and signaling pathways critical for cancer cell proliferation and metastasis [22].

4.3 Clinical Implications

More research is needed to examine the effects of MetS on ovarian cancer risk and mortality, as well as the underlying pathophysiologies in patients with obesity, diabetes mellitus, and MetS that may be targeted for therapeutic intervention [3]. MetS is not a strong ovarian cancer risk factor, though specific components may play a role in its development. The high prevalence of MetS calls attention to the importance of early improving and managing metabolic health among women and fortunately, many risk factors for obesity, diabetes and MetS are modifiable by diet and exercise.

Meanwhile, understanding the signaling pathways and molecular mechanisms between metabolic abnormalities and ovarian cancer will help develop new therapeutic strategies to regulate metabolism to control tumor growth and metastasis. Therefore, maintaining a healthy lifestyle, reasonable diet, moderate exercise, weight control, amongst other things helps to prevent metabolic diseases and malignant tumors. Simultaneously, patients who already have metabolic diseases, should be treated and controlled in time to reduce malignant tumor risk.

4.4 Strengths and Limitations

To the author's knowledge, this study is the first analysis of any association between metabolic syndrome and ovarian cancer severity. Furthermore, the same hospital laboratory results were used as the data source which provides greater accuracy, objectivity and reliability. Covariates were evaluated at baseline and models adjusted to improve the accuracy of test results. However, the study had several limitations. First, as a retrospective study, some individual data were missing, requiring data correction. It should be possible to collect more complete data from prospective trial results, such as the change in tumor markers before and after surgery. In that way, research becomes more meaningful for guiding clinical diagnosis and treatment. Second, this study used data from Beijing Friendship Hospital in the past ten years. Therefore, the results of this study may not be representative of current ovarian cancer patients.

5. Conclusions

In conclusion, this study suggests that MetS is not significantly associated with ovarian cancer severity. However, due to its retrospective nature and potentially confounding factors, further prospective research is required to more fully elucidate the role of MetS in the pathogenesis and progression of ovarian cancer.

Availability of Data and Materials

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Author Contributions

MJ and YJ were involved in the conception and design, or analysis and interpretation of the data; MJ and KZ were responsible for drafting the paper and revising it critically for important intellectual content; KZ contributed

to the acquisition, analysis, and interpretation of data for the work. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki. The study was approved by the Ethics Committee of Beijing Friendship Hospital, Capital Medical University (Approval No. 2025-P2-073-01). All patients signed an informed consent form.

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Conflict of Interest

The authors declare no conflict of interest.

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