Introduction

Excessive bodyweight is an established risk factor for several types of cancer. In particular, epidemiologic data show that obesity defined as body mass index (BMI) ≥30 kg/m^2 increases cancer risk and cancer-specific mortality [1, 2]. Although the precise mechanism is not clear, some obesity-related changes are expected to contribute to an increased risk of cancer. Insulin resistance and hyperinsulinemia stimulate insulin-like growth factor-1 (IGF-1) [3], which promotes cell proliferation, cell survival and angiogenesis [4]. Moreover, reactive oxygen radicals increased by obesity lead to systemic inflammation contributing to cancer development, and adipokines made by excess adiposity secrete cytokines related with carcinogenesis and promote insulin resistance [5]. Recent epidemiologic studies support these mechanisms, suggesting that obesity may affect poor prognosis in some cancers [6, 7].

However, the impact of underweight on prognosis has not been adequately addressed. Although underweight has been reported to be a high-risk factor for recurrence and death in patient with breast cancer [8], its role has not been evaluated in ovarian cancer. Furthermore, even in a recent meta-analysis which showed slightly worse survival in obese patients with ovarian cancer,

the impact of BMI including underweight as well as obesity was unclear because of a large amount of inter-study variation [9, 10].

Therefore, we investigated the impact of underweight on prognosis in patients with advanced-stage ovarian cancer depending on the time of measurement of BMI in relation to the treatment, and thereby evaluated the relationship between underweight and cancer progression with related changes of systemic inflammation and immunity.

Materials and Methods

1. Study population

Clinico-pathologic data for the current study were retrieved from a database of 360 patients registered from two tertiary medical centers (Seoul National University Hospital and Seoul National University Bundang Hospital) between 2000 and 2011. The current study was conducted after the approval by the Institutional Review Board of Seoul National University Hospital. The patients' medical records were reviewed retrospectively, and informed consent was not required because the current study was conducted through a retrospective review of medical records

2. Inclusion or exclusion criteria

We included patients with the following inclusion criteria: those with epithelial ovarian cancer; those with advanced-stage disease, in particular, the International Federation of Gynecology and Obstetrics (FIGO) stage III-IV disease; those who underwent staging operation, and taxane- and platinum-based chemotherapy; those with BMI measured at three time points including 'at diagnosis', 'after surgery', and 'after treatment'. However, we excluded

patients with non-epithelial ovarian cancer, synchronous or metachronous cancer and insufficient data for investigating the impact of BMI on survival.

3. Data collection

BMIs at diagnosis, after surgery, and after treatment were defined as those measured at diagnosis, before the first administration of adjuvant chemotherapy, and after the last administration of adjuvant chemotherapy. Furthermore, all patients were classified into four groups based on the following BMI criteria suggested by the World Health Organization for the Asian population: underweight (BMI <18.5 kg/m^2); normal (18.5 kg/m$^2 \leq$ BMI <23.0 kg/m^2); overweight (23.0 kg/m$^2 \leq$ BMI <27.5 kg/m^2); and obesity (BMI ≥ 27.5 kg/m^2) [11].

For evaluating potentials of cancer progression and related changes of systemic inflammation and immunity, we measured CA-125 level and neutrophil to lymphocyte ratio (NLR). NLR is known as a prognostic factor for recurrence and death in patients with ovarian cancer [12, 13]. Since increased inflammation and decreased immunity by cancer contribute to secondary hematological changes including relative neutrophilia and lymphocytopenia, NLR tends to increase in several types of malignancy [14,

15]. Thus, we measured CA-125 as a tumor marker and NLR as a marker of systemic inflammation and immunity using a radioimmunoassay kit (Fujirebio Diagnostics, Malvern, PA, USA) and SYSMEX XE-2100 (TOA Medical Electronics, Kobe, Japan) at diagnosis, after surgery and after treatment, respectively.

Clinico-pathologic characteristics including age, grade, FIGO stage, histology, neoadjuvant chemotherapy, cycles of adjuvant chemotherapy, optimal debulking surgery, and progression-free survival (PFS) and overall survival (OS) were collected. Patients treated with neoadjuvant chemotherapy received three cycles of taxane- and platinum-based chemotherapy before surgery, and optimal debulking surgery was considered when the size of residual tumor was less than 1 cm in the longest diameter. PFS was defined as the time that elapsed from the date after completion of the primary treatment to the date of clinically proven recurrence. OS was defined as the time that elapsed from the date of diagnosis to the date of cancer-related death or end of the study.

4. Statistical analysis

Kruskal-Wallis, Mann Whitney *U* and Chi-square tests were used to

determine differences in clinico-pathologic characteristics among underweight, normal to overweight, and obesity patients. Furthermore, univariate and multivariate analyses for investigating factors affecting survival were performed using the Kaplan-Meier method with log-rank test and Cox's proportional hazard regression model with hazard ratio (HR) and 95% confidence interval (CI). We conducted these statistical analyses using SPSS software (version 19.0; SPSS Inc, Chicago, IL, USA). A $P < 0.05$ was considered statistically significant.

Results

1. Patients' characteristics

Clinico-pathologic characteristics of all patients are depicted in Table 1. The mean age was 53.9 years (range, 18-80 years), and 5 (1.4%), 23 (6.4%), 256 (71.1%) and 76 patients (21.1%) had stage IIIA, IIIB, IIIC and IV diseases. Furthermore, serous carcinoma was identified in 276 (76.7%) patients while endometrioid, clear cell, mucinous, undifferentiated and mixed carcinomas were observed in 29 (8.1%), 20 (5.6%), 13 (3.6%), 7 (1.9%) and 15 (4.2%). Three cycles of neoadjuvant chemotherapy using taxanes and platinums were administered in 57 patients (15.8%), and the mean value of cycles of adjuvant chemotherapy using the same regimen was 6 (range, 3-12).

Among 360 patients, underweight, normal, overweight and obesity were identified in 12 (3.3%), 162 (45.0%), 150 (41.7%) and 36 (10.0%) at diagnosis; 32 (8.9%), 183 (50.8%), 118 (32.8%) and 27 (7.5%) after surgery; 29 (8.1%), 146 (40.6%), 157 (43.6%) and 28 (7.8%) after treatment. In particular, 7 patients (58.3%) who showed underweight at diagnosis revealed still underweight after treatment (Figure 1).

2. Underweight affecting prognosis

We compared PFS and OS among underweight, normal to overweight and obesity patients according to the treatment time. As a result, only patients with underweight *after treatment* showed poor OS in comparison with those with normal to overweight or obesity (mean value, 44.9 vs. 78.8 or 67.4 months; P=0.05; Figure 2). When we adjusted the result with clinico-pathologic characteristics, underweight after treatment was an unfavorable factor for OS (adjusted HR, 2.29; 95% CI, 1.08-4.85; Table 2).

Next, we compared CA-125 and NLR among underweight, normal to overweight and obesity patients according to the treatment time (Table 3). As a result, CA-125 *at diagnosis* was higher in patients with normal to overweight or obesity than in those with underweight (median value, 865 or 912.5 vs. 185.5 U/ml; P=0.04). Since patients with underweight *at diagnosis* underwent suboptimal debulking surgery less frequently than those with normal weight to overweight or obesity, we made subgroup analyses based on whether optimal debulking surgery was performed. As a result, there were no differences in CA-125 and NLR among underweight, normal to overweight and obesity patients who underwent optimal debulking surgery (median value of CA-125, 161.5 vs. 555 vs. 490 U/ml; P=0.37: median value of NLR, 2.93

vs. 2.51 vs. 2.54; P=0.86), and suboptimal debulking surgery (median value of CA-125, 956 vs. 1,043 vs. 930 U/ml; P=0.68: median value of NLR,3.29 vs. 3.49 vs. 3.43; P=0.55).

Furthermore, the rate of suboptimal debulking surgery was also different between underweight and obesity patients *after surgery* in spite of no differences of CA-125 and NLR. Thus, we also made subgroup analyses according to whether optimal debulking surgery was performed, and CA-125 and NLR were not different among underweight, normal to overweight and obesity patients who underwent optimal debulking surgery (median value of CA-125, 71 vs. 76.5 vs. 65 U/ml; P=0.39: median value of NLR, 2.58 vs. 2.37 vs. 2.68; P=0.43), and suboptimal debulking surgery (median value of CA-125, 216.8 vs. 202 vs. 105 U/ml; P=0.52: median value of NLR, 2.53 vs. 3.05 vs. 3.71; P=0.15).

On the other hand, patients with underweight *after treatment* showed higher NLR than those with obesity in spite of no differences of confounding factors between two groups (median value, 2.15 vs. 1.47; P=0.03).

3. Degree of weight loss affecting prognosis

Next, we compared clinico-pathologic characteristics and prognosis

according to the degree of weight loss in only patients with underweight *after treatment*. All 29 patients with underweight after treatment were divided into two groups according to the following criteria: weight loss ≥10% vs. <10% from the body weight at diagnosis. Clinico-pathologic characteristics based on the degree of weight loss are summarized in Table 4. Although there were no differences in age, FIGO stage, histology, grade, neoadjuvant chemotherapy, cycles of adjuvant chemotherapy, CA-125 and NLR between two groups, the success rate of optimal debulking surgery was higher in underweight patients with weight loss <10% than in those with weight loss ≥10% (83.3% vs. 36.4%; P=0.02).

Furthermore, underweight patients with weight loss ≥10% showed poor PFS and OS in comparison with those with weight loss <10% (PFS, median value, 3.5 vs. 16.8 months; OS, median value, 23.7 vs. 58.1 months; Figure 3). Weight loss ≥10% was also a poor prognostic factor for PFS and OS when adjusted with other clinico-pathologic factors (adjusted HRs, 6.90 and 15.27; 95% CIs, 1.51-31.54 and 1.42-164.5; Table 5).

Discussion

In terms of the association between BMI and cancer risk and prognosis, most of the studies have focused mainly on the impact of obesity, because deleterious mechanisms related to obesity are expected to be unfavorable to cancer patients [16]. However, excessive weight loss can be also associated with poor prognosis because it has similar features to cancer cachexia, a complex metabolic condition characterized by loss of skeletal muscle and body weight, developed in progressive disease [17, 18].

Epidemiologically, the relationship between the risk of mortality and BMI is known to be U-shaped with the increased risk related with either cachexia showing very low BMI or obesity demonstrating very high BMI, whereas emerging data indicate that obesity is associated paradoxically with better prognosis in cancer patients [19]. In the current study, we also found that underweight after treatment was an unfavorable factor for OS in patients with advanced-stage ovarian cancer (adjusted HR, 2.29; 95% CI, 1.08-4.85), whereas obesity was not associated with prognosis regardless of the treatment time. The lack of an association between obesity and prognosis can be explained by the following reasons. The cut-off value for defining obesity is

relatively low in the Asian population than in the Western population (27.5 kg/m^2 vs. 30 kg/m^2), which leads to different effects of obesity based on race. Moreover, obesity can help patients endure the increased resting energy expenditure (REE) which occurs in cancer [17]. This endurance can help patients with advanced-stage ovarian cancer which show 5-year survival rate of approximately 30% [20], to maintain their general condition thereby improving survival. These hypotheses can be supported by a recent epidemiologic data showing no association between obesity and poor prognosis in Asian patients with ovarian cancer [21].

However, underweight can act as a poor prognostic factor in the patients. Theoretically, most of patients with advanced-stage ovarian cancer should recover from underweight after treatment because the Warburg effect, increased glucose uptake by tumors for glycolysis to generate ATP, is expected to reduce with the decrease of tumor burden after treatment [22]. Inversely, failure to regain weight after treatment means that the cancer has potentially progressed, and it is easily identified in patients with cancer cachexia. In detail, systemic inflammation induced and persisted by increased tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), IL-1 and interferon-gamma (IFN-γ), leads to the decrease of protein anabolism and

caloric intake, while promoting the increase of protein catabolism, insulin resistance, lipolysis and REE. Eventually, loss of muscles mass and strength, ineffective host anti-tumor response, loss of whole body fat and impaired immunity occur, leading to physical disability, diminished quality of life and reduced survival [4, 17, 23-26].

To prove this hypothesis clinically, we investigated CA-125 as a tumor marker and NLR as a marker reflecting inflammation and immunity among underweight, normal weight to overweight and obesity patients according to the treatment time. After treatment, although there were no differences of CA-125 among the three groups, underweight patients showed the highest NLR compared with normal weight to overweight and obesity patients, suggesting increased systemic inflammation (neutrophilia) and decreased immunity (lymphocytopenia) in these patients. This means that underweight after treatment is a condition that increases the likelihood of cancer progression, and it can be considered as an early marker for poor prognosis in patients with advanced-stage ovarian cancer. Since 58.3% of patients who were underweight at diagnosis failed to recover from underweight after treatment, we should pay attention to weight change during the treatment period in patients with advanced-stage ovarian cancer.

For understanding the association between cancer cachexia and underweight after treatment, we divided all underweight patients into two groups by the degree of weight loss considering the features of cancer cachexia that impact on patients' function and survival as follows: weight loss >10%; systemic inflammation (C-reactive protein >10mg/l); reduced food intake (<1,500 kcal/day) [17]. As a result, the risk of suboptimal surgery increased in underweight patients with weight loss ≥10% (63.6% vs. 16.7%; P=0.02), and weight loss ≥10% was an independently poor prognostic factor for PFS and OS (adjusted HRs, 6.90 and 15.27; 95% CIs, 1.51-31.54 and 1.42-164.5). It means that severe weight loss (≥10%) after treatment was associated with more unresectable tumors, and an increased risk of cancer progression. However, there was no difference in NLR between two groups in spite of the tendency that it was higher in underweight patients with weight loss ≥10% than in those with weight loss <10% (median value, 2.15 vs. 2.04). We thought that a small number of enrolled patients with underweight led to no statistical difference, and a large-scale cohort should be needed to prove it.

The current study is the first report demonstrating the impact of underweight after treatment on prognosis of gynecologic cancer. However, there are some limitations as follows. First, we could not evaluate the impact of underweight

on prognosis of patients with early-stage ovarian cancer because they showed good prognosis on which we could not understand the impact of underweight. Second, we measured only NLR as an indicator of host inflammation and immunity because other pro-inflammatory markers or cytokines were not included in the clinical setting. Third, all patients in the current study were ethnically homogenous Asians, so that the impact of underweight after treatment on prognosis should be investigated for other ethnic groups.

Conclusively, we found that underweight after treatment may be a poor prognostic factor in patients with advanced-stage ovarian cancer, and this is accompanied by increased tumor-induced inflammation and decreased immunity. Underweight status can act as an early marker to predict poor prognosis. In particular, we should pay attention to weight change during the treatment period because more than half of patients with underweight at diagnosis failed to gain weight, and a weight loss ≥10% after treatment was associated with an increased risk of disease recurrence and mortality.

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Table 1. Clinico-pathologic characteristics of 360 patients

Characteristics	No. of Patients (%)
Age (years)	
<53	170 (47.2)
≥53	190 (52.8)
FIGO stage	
III	284 (78.9)
IV	76 (21.1)
Histology	
Serous	276 (76.7)
Non-serous	84 (23.3)
Grade	
1	10 (2.8)
2	59 (16.4)
3	181 (50.3)
Unknown	110 (30.6)
Residual tumor (cm)	
≥1	173 (48.1)
<1	187 (51.9)
Cycles of adjuvant chemotherapy	
≤6	224 (62.2)
>6	136 (37.8)

FIGO, International Federation of Gynecology and Obstetrics.

Table 2. Clinico-pathologic factors affecting progression-free and overall survivals in all 360 patients with advanced-stage ovarian cancer

Characteristics	Univariate			Multivariate		
	HR	95% CI	P	Adjusted HR	95% CI	P
Progression-free survival						
≥53 years	1.04	0.82-1.32	0.74	–	–	–
Stage IV disease	1.27	0.95-1.69	0.11	–	–	–
Grade 3 disease	1.11	0.80-1.53	0.53	–	–	–
Non-serous histology	1.26	0.95-1.67	0.11	–	–	–
No neoadjuvant chemotherapy	1.62	1.19-2.20	<0.01	1.84	1.18-2.87	<0.01
≤6 cycles of adjuvant chemotherapy	1.03	0.81-1.32	0.81	–	–	–
Suboptimal debulking	1.54	1.21-1.96	<0.01	1.71	1.22-2.39	<0.01
Underweight after treatment	1.25	0.80-1.93	0.33	–	–	–
Overall survival						
≥53 years	1.12	0.79-1.58	0.52	–	–	–
Stage IV disease	1.21	0.80-1.84	0.36	–	–	–
Grade 3 disease	1.21	0.75-1.93	0.44	–	–	–
Non-serous histology	1.58	1.07-2.33	0.02	–	–	–
No neoadjuvant chemotherapy	1.65	1.08-2.54	0.02	1.88	1.28-2.77	<0.01
≤6 cycles of adjuvant chemotherapy	1.16	0.82-1.64	0.42	–	–	–
Suboptimal debulking	1.49	1.05-2.11	0.03	1.67	1.23-2.28	<0.01
Underweight after treatment	2.01	1.13-3.58	0.02	2.29	1.08-4.85	0.03

Table 3. Comparison of CA-125 and neutrophil to lymphocyte ratio (NLR) among underweight, normal to overweight and obesity patients according to the treatment time

BMI	CA-125 (median, U/ml)	NLR (median)	Confounding factors			
			Stage IV disease	Grade 3 disease	Non-serous histology	Suboptimal debulking
At diagnosis						
Underweight	185.5	8[*,†]	1 (8.3)[*,†]	3 (25.0)[*,†]	4 (33.3)[*,†]	2 (16.7)[*]
Normal to overweight	865[*]	9[*,‡]	69 (22.3)[*,‡]	156 (50.3)[*,‡]	69 (22.3)[*,‡]	149 (48.1)[*,†]
Obesity	912.5[*]	6.75[†,‡]	5 (13.9)[†,‡]	21 (58.3)[†,‡]	5 (13.9)[†,‡]	21 (58.3)[†]
P value	0.04	0.47	0.28	0.14	0.33	0.04
After surgery						
Underweight	95.6[*,†]	2.58[*,†]	6 (18.8)[*,†]	15 (46.9)[*,†]	3 (9.4)[*,†]	10 (32.2)[*,†]
Normal to overweight	190.0[*,‡]	2.67[*,‡]	65 (22.3)[*,‡]	146 (50)[*,‡]	67 (22.9)[*,‡]	143 (49)[*,‡]
Obesity	87.5[†,‡]	3.14[†,‡]	5 (18.5)[†,‡]	16 (59.3)[†,‡]	5 (17.9)[†,‡]	16 (59.3)[†,‡]
P value	0.78	0.23	0.83	0.60	0.29	0.08
After treatment						
Underweight	8[*,†]	2.15[*]	5 (17.2)[*,†]	13 (44.8)[*,†]	6 (20.7)[*,†]	10 (34.5)[*,†]
Normal to overweight	9[*,‡]	1.56[*,†]	62 (20.7)[*,‡]	148 (50.7)[*,‡]	67 (22.4)[*,‡]	146 (48.8)[*,‡]
Obesity	6.8[†,‡]	1.47[†]	8 (28.6)[†,‡]	18 (66.7)[†,‡]	4 (14.3)[†,‡]	13 (46.4)[†,‡]
P value	0.21	0.09	0.54	0.27	0.60	0.33

BMI, body mass index; [*,†,‡]no significant difference between two groups with the same symbol.

Table 4. Clinico-pathologic characteristics of 29 underweight patients after treatment according to the degree of weight loss

Characteristics	Weight loss ≥10% (n=11, %)	Weight loss <10% (n=18, %)	P
Age (years)			0.70
<53	7 (63.6)	9 (50)	
≥53	4 (36.4)	9 (50)	
FIGO stage			0.62
III	10 (90.9)	14 (77.8)	
IV	1 (9.1)	4 (22.2)	
Histology			0.17
Serous	7 (63.6)	15 (83.3)	
Non-serous	4 (36.4)	3 (16.7)	
Grade			1.00
1	0 (0)	0 (0)	
2	3 (27.2)	5 (27.8)	
3	4 (36.4)	9 (50)	
Unknown	4 (36.4)	4 (22.2)	
Residual tumor (cm)			0.02
≥1	7 (63.6)	3 (16.7)	
<1	4 (36.4)	15 (83.3)	
Neoadjuvant chemotherapy			1.00
No	9 (81.8)	15 (83.3)	
Yes	2 (18.2)	3 (16.7)	
Cycles of adjuvant chemotherapy			0.36
≤6	10 (90.9)	13 (72.2)	
>6	1 (9.1)	5 (27.8)	
CA-125 (median, range, U/ml)	10.3 (1.8, 104)	8 (3.7, 27.0)	0.52
NLR (median, range)	2.15 (1.05, 41.03)	2.04 (0.56, 7.16)	0.94

NLR, neutrophil to lymphocyte ratio.

Table 5. Clinico-pathologic factors affecting progression-free and overall survivals in 29 patients who showed underweight after treatment

Characteristics	Univariate			Multivariate		
	HR	95% CI	P	Adjusted HR	95% CI	P
Progression-free survival						
≥53 years	1.91	0.79-4.63	0.15	–	–	–
Stage IV disease	1.88	0.68-5.18	0.23	4.89	1.14-20.94	0.03
Grade 3 disease	0.94	0.33-2.70	0.91	–	–	–
Non-serous histology	2.99	1.13-7.90	0.03	–	–	–
No neoadjuvant chemotherapy	1.54	0.51-4.64	0.45	–	–	–
≤6 cycles of adjuvant chemotherapy	1.10	0.40-3.01	0.86	–	–	–
Suboptimal debulking	3.89	1.55-9.74	<0.01	10.04	1.48-68.13	0.02
Weight loss ≥10%	4.07	1.55-10.64	<0.01	6.90	1.51-31.54	0.01
Overall survival						
≥53 years	2.07	0.62-6.92	0.24	–	–	–
Stage IV disease	1.92	0.40-9.25	0.42	11.9	1.00-141.1	0.05
Grade 3 disease	0.77	0.17-3.47	0.73	–	–	–
Non-serous histology	2.58	0.73-9.15	0.14	–	–	–
No neoadjuvant chemotherapy	1.27	0.27-6.02	0.77	–	–	–
≤6 cycles of adjuvant chemotherapy	3.84	0.78-18.91	0.10	–	–	–
Suboptimal debulking	2.64	0.88-7.91	0.08	–	–	–
Weight loss ≥10%	12.81	2.54-64.65	<0.01	15.27	1.42-164.5	0.02

Figure 1. Proportion of underweight patients with advanced-stage ovarian cancer according to the treatment time.

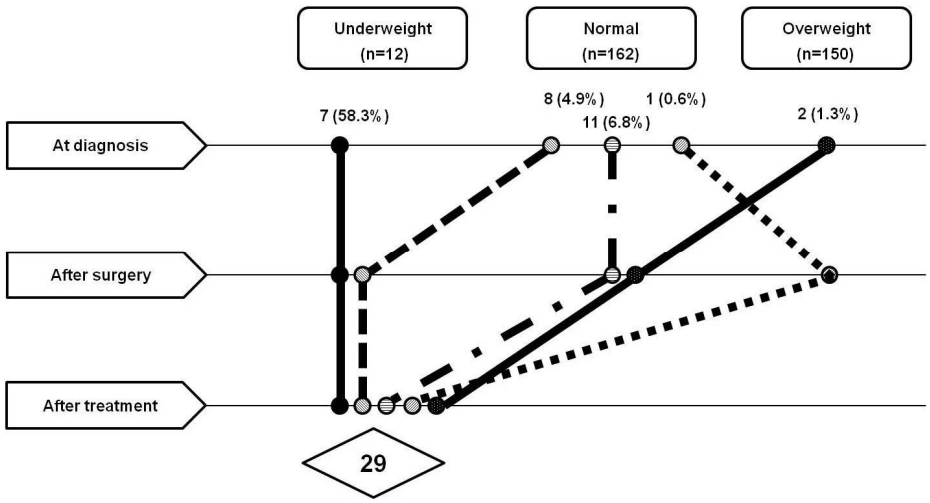


Figure 2. Kaplan-Meier analyses with the log-rank test for comparing progression-free survival and overall survival among underweight, normal to overweight and obesity with advanced-stage ovarian cancer: (A) at diagnosis; (B) after surgery; (C) after treatment.

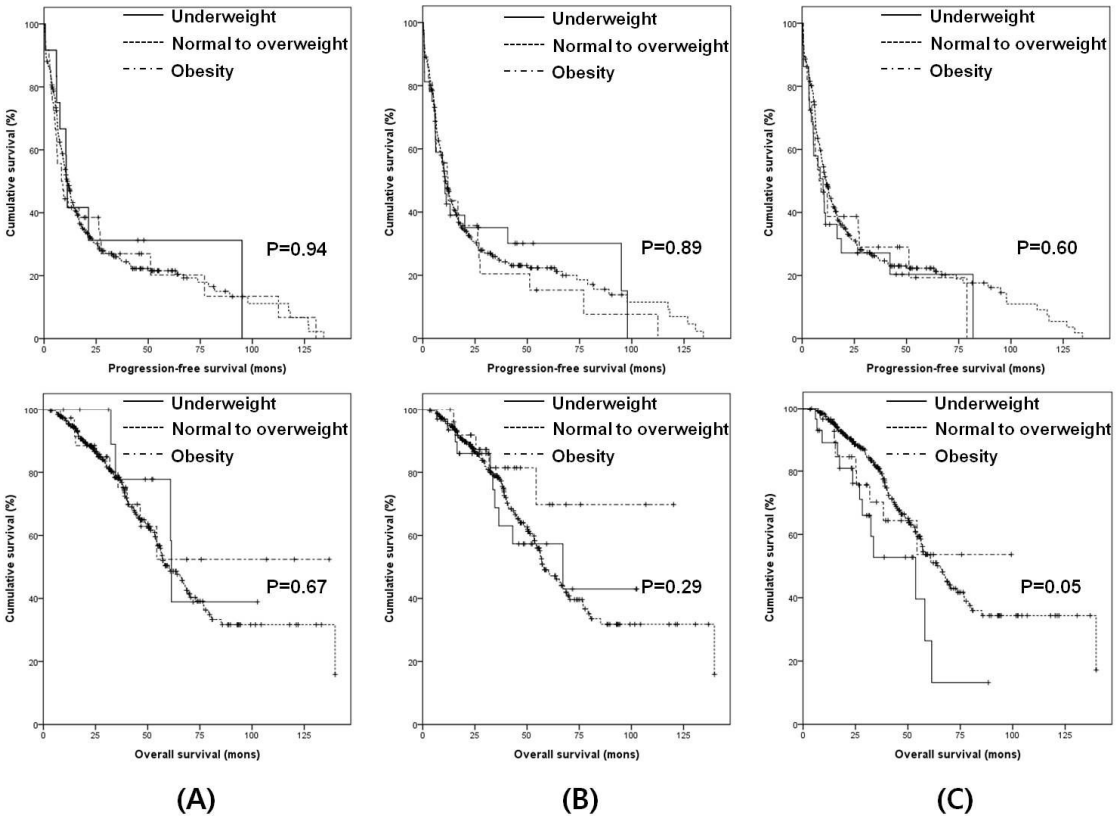
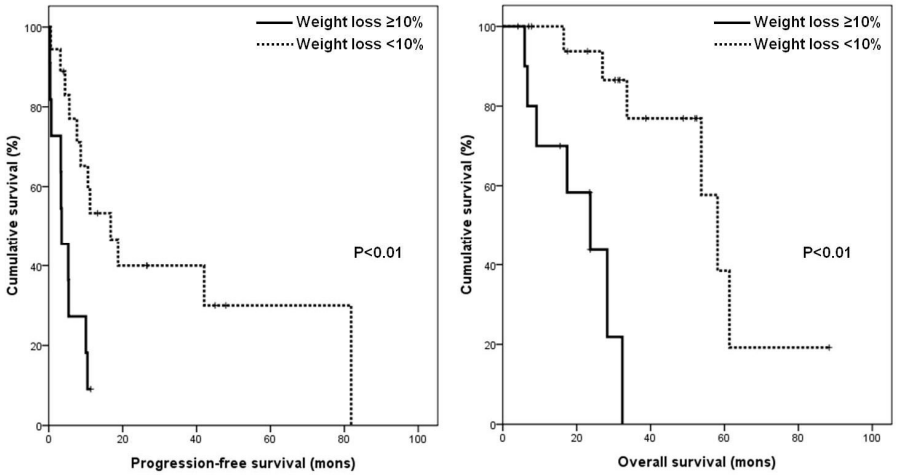


Figure 3. Kaplan-Meier analyses with the log-rank test for comparing progression-free survival and overall survival between weight loss ≥10% and <10% in underweight patients after treatment.



초 록

목적: 저체중 상태가 진행성 난소암의 예후에 미치는 영향을 알아보고자 하였다.

방법: 총 360명의 상피성 난소암 3-4기 환자를 대상으로, 체질량지수 (BMI) 에 따라 세 그룹으로 나누었다: 저체중 (BMI <18.5kg/m^2); 정상-과체중 (18.5kg/m^2≤BMI<27.5kg/m^2); 비만 (BMI≥27.5kg/m^2). 각 그룹의 무진행 생존기간, 전체 생존기간, CA-125, 그리고 개체의 전신 염증 과 면역 상태를 반영하는 지표인 호중구/림프구 비율 (NLR) 을 조사하여, 진단 당시, 수술 후, 치료 후 의 세 치료 시점에 따라 비교 분석하였다.

결과: 치료 후 저체중 그룹이 정상-과체중 또는 비만 그룹에 비해 불량한 전체 생존기간을 보였다 (평균값, 44.9 vs. 78.8 or 67.4 개월; P=0.05). 또한, 다변량분석에서 치료 후 저체중 상태는 전체 생존기간의 불량한 예후인자임을 확인하였다 (보정 비례위험도, 2.29; 95% 신뢰구간, 1.08-4.85). 세 치료 시점에서 그룹간 CA-125 는 차이가 없었으나, 치료 후 저체중 그룹이 비만 그룹에 비해 호중구/림프구 비율이 유의하게 높았다 (중앙값, 2.15 vs. 1.47; P=0.03). 치료 후 저체중 상태인 29명의 환자들만 추가분석 하였을 때, 체중 감소가 10% 미만인 그룹이 10% 이상인 그룹에

비해 높은 최적 종양 감축술 시행율을 보였다 (83.3% vs. 36.4%; P=0.02). 10% 이상의 체중 감소는 무진행 생존기간과 전체 생존기간 모두에서 불량한 영향을 주고 있음을 확인하였다 (보정 비례위험도, 6.90 과 15.27; 95% 신뢰구간, 1.51-31.54 와 1.42-164.5).

결론: 치료 후 저체중 상태는 진행성 난소암의 불량한 예후 인자이며, 이는 개체의 전신 염증 증가와 면역력 감소를 동반한다.

주요어: 치료 후 저체중, 염증, 면역력, 생존, 난소암

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