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Assessment of Serum Omentin-1 in Prostate Cancer: Correlation with Anthropometric and Biochemical Factors

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Abstract. **General Background:** Omentin-1, a novel adipokine predominantly secreted by visceral adipose tissue, modulates glucose metabolism, inflammation, and carcinogenesis, yet its clinical significance in prostate cancer remains underexplored. **Knowledge Gap:** While adipokines are implicated in obesity-related malignancies, the specific relationship between serum omentin-1 levels and prostate cancer, particularly regarding body mass index and metabolic parameters, is not fully understood. **Aims:** This study evaluated serum omentin-1 concentrations in 88 newly diagnosed prostate cancer patients compared to 88 age-matched healthy controls, examining correlations with anthropometric measurements and biochemical markers. **Results:** Serum omentin-1 levels were significantly elevated in prostate cancer patients, particularly in younger age groups and normal-weight to overweight categories, with negative correlation to BMI ($r=-0.3$, $p<0.01$) and positive correlation to PSA ($r=0.5$, $p<0.01$); multivariate regression identified omentin-1 as an independent protective factor (OR=0.79, $p<0.0001$). **Novelty:** This research establishes omentin-1 as an independent predictor of prostate cancer risk alongside traditional biomarkers. **Implications:** These findings suggest omentin-1 may serve as a complementary biomarker reflecting metabolic-oncological interactions in prostate cancer risk stratification.

Keywords: Omentin-1, Prostate Cancer, Body Mass Index, Prostate-Specific Antigen, Adipokines

Highlights:

1. Omentin-1 levels negatively correlate with BMI but positively correlate with PSA levels.
2. Elevated omentin-1 particularly evident in younger age groups and normal-weight individuals.
3. Omentin-1 identified as independent protective factor against prostate cancer in regression analysis.

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Introduction

Omentin-1 is a recently identified adipokine predominantly expressed in visceral adipose tissue, where it plays a significant role in modulating insulin sensitivity, glucose metabolism, and inflammatory responses [1][2]. Recent research suggests that adipokines, including omentin-1, may be involved in tumor initiation, angiogenesis, and progression, particularly in obesity-related cancers such as prostate cancer[2] [3][4]. Prostate cancer remains one of the most common malignancies among men worldwide, with metabolic alterations, obesity, and chronic inflammation recognized as important contributing factors to its development[2][3] [5]. Although the role of adipokines in cancer has been increasingly studied, the specific function of omentin-1 in prostate cancer pathogenesis is not yet fully understood[2] [6]. Therefore, this study aims to evaluate the relationship between serum omentin-1 levels and clinical variables including BMI, age, type of therapy, and comorbidities such as diabetes and hypertension in patients with prostate cancer compared to healthy controls.

Materials and Methods

Study Design

This case–control study was conducted at Al-Najaf National Cancer Hospital from December 2024 to July 2025. The study included 176 participants: 88 patients with histopathologically confirmed prostate cancer and 88 age-matched healthy controls. Ethical approval was obtained from the College of Medicine, University of Basrah, and all participants provided written informed consent.Patients were eligible if they had confirmed prostate cancer and a PSA level above 4 ng/mL.

Exclusion Criteria

Controls were healthy males without history or clinical evidence of :

1. Pcancer or BPH
2. Individuals with a history of other malignancies previous prostate surgeries
3. Acute infectious diseases
4. Chronic liver and kidney diseases were excluded

Physical and biochemical measurements

Clinical information including age, body mass index (BMI), Blood pressure was measured with a mercury sphygmomanometer and was taken twice after at least 10 minutes between measurements and the average value was selected for data analysis and comorbidities such as hypertension and diabetes was recorded for all participants.

Biochemical Analysis

Serum omentin-1 concentrations were measured using a commercial ELISA kit (Cloud-Clone Corp., USA) following the manufacturer's protocol. PSA levels were analyzed using the VIDAS automated system (Biomerieux, France), while fasting blood sugar ,lipid profile, Blood Urea and serum creatinine parameters were determined using the DRI-CHEM NX600 analyzer (Fujifilm, Japan).

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Statistical Analysis

Statistical analysis was carried out using SPSS version 23.0. Independent t-test and Mann–Whitney U test were applied for distribution of Serum omentin among the studied groups according to age and BMI. Pearson and Spearman correlation analyses were performed to assess associations between serum omentin levels and study variables and biochemical parameters in the study population.Correlation and regression analyses were performed to evaluate associations between serum omentin-1 levels and BMI, age, and comorbidities. A p-value < 0.05 was considered statistically significant.

Results

Distribution of Serum omentin by age groups in patients and controls: The distribution of serum omentin levels among patients with prostate cancer and healthy controls across different age categories is shown in **Table 1**. In the <50 years group, patients exhibited markedly elevated omentin concentrations compared with controls (38.7 ± 28.9 ng/ml vs. 25.8 ± 6.2 ng/ml, p<0.01). Similarly, in the 60–69 years group, patients showed a highly significant increase in omentin levels relative to controls (47.1 ± 19.1 ng/ml vs. 25.4 ± 7.7 ng/ml, p=0.0009). In the 51–59 and ≥70 years groups, although mean serum omentin concentrations were higher in patients than in controls, the differences did not reach statistical significance (p=0.4 and p=0.08, respectively). Overall, these findings indicate that elevated omentin levels in patients compared with controls are particularly evident in younger (<50 years) and intermediate (60–69 years) age categories.

Table 1. Distribution of Serum omentin by age groups in patients and controls.

Age Group (years)	Group	N (176)	Serum Omentin (ng/ml) Mean ± SD	P-Value
<50	**Control**	28	25.8±6.2	**<0.01**
	Patients	5	38.7±28.9	
51–59	**Control**	17	28.9±6.1	0.4
	Patients	21	42.2±23.9	
60-69	**Control**	15	25.4±7.7	**<0.01**
	Patients	29	47.1±19.1	
≥70	**Control**	28	25.1±6.5	0.08
	Patients	33	41.7±24.2	

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*Significant at $p < 0.05$

*Non-significant at $p > 0.05$

* All analyses for these variables were performed using the Mann–Whitney U test.

Distribution of Serum omentin among the studied groups according to BMI in study population:

Distribution of serum omentin levels across BMI categories (<18.5, 18.5–24.9 [normal], 25–29.9 [overweight], ≥30 [obese]) in patients and controls shown in **table 2**. Among the more populated groups, overweight individuals (BMI 25–29.9) exhibited the highest mean omentin concentrations, with controls at 25.9 ± 6.3 ng/ml (n = 47) and patients at 41.1 ± 21.6 ng/ml (n = 49). Interestingly, the normal-weight subgroup (BMI 18.5–24.9), despite its smaller sample size, showed the highest mean omentin levels in both controls (30.4 ± 4.5 ng/ml, n = 23) and patients (47.7 ± 22.7 ng/ml, n = 22). The Under weight <18.5 was showed the highest mean omentin levels patients76.3±10.9.

Table 2. Distribution of Serum omentin among the studied groups according to BMI.

BMI Category	**Group**	**N (176)**	**Serum Omentin (ng/ml) Mean ± SD**	**P-Value**
<18.5 (Under weight)	**Control**	-	-	-
	Patients	3	76.3±10.9	
18.5–24.9 (Normal)	**Control**	23	30.4±4.5	**0.03**
	Patients	22	47.7±22.7	
25–29.9 (Overweight)	**Control**	47	25.9±6.3	**0.008**
	Patients	49	41.1±21.6	
≥30 (Obese)	**Control**	18	20.9±5.9	0.1
	Patients	14	37.4±22.8	

*Significant at $p < 0.05$

*Non-significant at $p > 0.05$.

* All analyses for these variables were performed using the Mann–Whitney U test.

Correlation of serum omentin level with other Variables of the Study:

The correlations between serum omentin levels and different study variables is shown in **Table 3** . A significant negative correlation was observed with BMI (r = –0.3, p<0.01), indicating that higher adiposity is associated with lower serum omentin levels.

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No statistically significant associations were found between serum omentin and age, residency, smoking status, comorbidities, cancer stage, metastasis, lymph node status, or Gleason score (p>0.05).

Table 3. Spearman Correlation of serum omentin level with other Variables of the Study.

Variables		**r-Value /P-Value**
BMI (kg/m²)	**Correlation Coefficient**	-0.3
	P-Value	**<0.01**
Age	**Correlation Coefficient**	**0.02**
	P-Value	**0.6**
Residency	**Correlation Coefficient**	-0.1
	P-Value	0.1
Smoking state	**Correlation Coefficient**	-0.01
	P-Value	0.9
Comorbidities	**Correlation Coefficient**	0.07
	P-Value	0.3
Cancer Stage	**Correlation Coefficient**	-0.04
	P-Value	0.6
Metastasis	**Correlation Coefficient**	0.04
	P-Value	0.7
Node status	**Correlation Coefficient**	-0.09
	P-Value	0.4
Gleason Score	**Correlation Coefficient**	-0.1
	P-Value	0.6

*Significant at $p < 0.05$

*Non-significant at $p > 0.05$.

*Pearson and Spearman correlation analyses were performed to assess associations between serum omentin levels and study variables.

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Spearman Correlation of Serum omentin levels with the biochemical parameters in the study population:

The relationship between serum omentin levels and biochemical parameters in the study population is illustrated in **table 4** . Significant positive correlations was observed with PSA levels ($r = 0.5$, $p<0.01$), indicating that higher PSA concentrations were associated with increased serum omentin. Additionally, a significant negative correlation was found with fasting blood sugar (FBS) ($r = –0.1$, $p=0.008$) and HDL cholesterol ($r = –0.2$, $p=0.02$), suggesting that altered glucose metabolism and lipid profile may influence circulating omentin levels.

A positive correlation was also observed with BUN ($r = 0.16$, $p=0.02$), whereas no significant associations were detected with cholesterol, triglycerides, LDL, VLDL, or serum creatinine ($p>0.05$). Overall, these results indicate that serum omentin is closely linked to PSA and certain metabolic parameters, highlighting its potential role as a biomarker reflecting both oncological and metabolic status.

Table 4. Correlation of Serum omentin levels with the biochemical parameters in the study population.

Variables		**r-Value /P-Value**
PSA (ng/ml)	**Correlation Coefficient**	0.5
	P-Value	**<0.01**
FBS (mg/dl)	**Correlation Coefficient**	-0.1
	P-Value	**0.008**
Cholesterol (mg/dl)	**Correlation Coefficient**	0.05
	P-Value	0.4
Triglycerides (mg/dl)	**Correlation Coefficient**	-0.04
	P-Value	0.5
HDL (mg/dl)	**Correlation Coefficient**	-0.2
	P-Value	**0.02**

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LDL (mg/dl)	**Correlation Coefficient**	0.1
	P-Value	0.1
VLDL (mg/dl)	**Correlation Coefficient**	-0.04
	P-Value	0.5
S-creatinine (mg/dl)	**Correlation Coefficient**	-0.03
	P-Value	0.6
BUN (mg/dl)	**Correlation Coefficient**	0.16
	P-Value	**0.02**

*Significant at $p < 0.05$

*Non-significant at $p > 0.05$.

*Pearson and Spearman correlation analyses were performed to assess associations between serum omentin levels and biochemical parameters.

Multivariate logistic regression analysis of predictors of prostate cancer:

The multivariate logistic regression analysis of predictors of prostate cancer is shown in **table 5** . Serum omentin-1 was significantly associated with reduced risk of prostate cancer (OR=0.79, p<0.0001). Total PSA was a strong positive predictor (OR=1.97, p<0.0001). BMI also showed a significant inverse association (OR=0.81, p=0.008). In contrast, age, smoking status, and most comorbidities did not show significant associations, except for the absence of comorbidities which was positively associated with prostate cancer risk (OR=9.38, p=0.01).

Table 5. The multivariate logistic regression analysis of predictors of prostate cancer.

Predictor	**SE**	**OR (95% CI)**	**P-value**
Serum Omentin	0.056	0.79 (0.7- 0.00008)	**0.00002**
Total PSA	0.17	1.97(1.52- 0.000002)	**0.00005**

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BMI	0.08	0.81(0.68- 0.0000009)	**0.008**
Age	0.03	1.01 (0.95 -0.00001)	0.8
Comorbidities (HTN)	1.4	1.27 (0.05- 0.00001)	0.9
Comorbidities (None)	0.9	9.38 (1.82 -0.0000006)	**0.01**
Comorbidities (HTN+DM)	1.1	2.55 (0.28 -0.0002)	0.4
Not Smoking	0.9	1.7 (0.3 -0.000001)	0.6
Smoking	0.99	1.36 (0.2 -0.0000001)	0.8

* Significant at $p < 0.05$

* Non-significant at $p > 0.05$

*Multivariate logistic regression analysis was performed to identify independent predictors of prostate cancer.

Discussion

The present study highlights that serum omentin-1 levels are significantly elevated in prostate cancer (PCa) patients compared to controls, especially in younger and intermediate age groups and in normal/overweight BMI categories. This pattern is consistent with the growing recognition of adipokines as mediators between metabolic status and cancer risk. Elevated omentin-1 in PCa patients, particularly among younger and intermediate-aged groups, may reflect the complex interplay between adipose tissue signaling and tumorigenesis. While some studies have shown omentin-1 to be positively associated with age and sex hormone-binding globulin, its relationship with metabolic syndrome in PCa remains unclear[6]. In other cancers, such as head and neck cancer, omentin-1 levels also vary with BMI, supporting the idea that adipokine profiles are influenced by both cancer status and body composition[7]. The negative correlation between omentin-1 and BMI observed here is in line with findings from cardiovascular and metabolic research, where higher adiposity is linked to lower omentin-1 concentrations[8][9]. The positive association between omentin-1 and PSA in PCa patients suggests a potential role for omentin-1 as a biomarker reflecting tumor presence or activity. This is supported by evidence that omentin-1 is elevated in several malignancies,

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including prostate and colorectal cancers, and may be involved in cancer development and progression [10] [2][11]. However, the lack of significant associations with cancer stage, Gleason score, or comorbidities in this study suggests that omentin-1 may be more indicative of metabolic and inflammatory status than of tumor aggressiveness[6][2]. Multivariate analysis in this study identified omentin-1, PSA, and BMI as independent predictors of PCa risk. This aligns with research showing that genetic and metabolic markers in adipokine pathways can enhance risk prediction for prostate cancer beyond traditional clinical factors[12]. Furthermore, the role of omentin-1 in other obesity-related cancers and metabolic diseases underscores its potential as a biomarker and therapeutic target[2][13][11] [14][15].

Conclusion

In summary, the findings of this study indicate that serum omentin-1 levels are significantly elevated in patients with prostate cancer compared with healthy controls and are closely associated with body mass index and selected metabolic and biochemical parameters. The observed negative correlation between omentin-1 and BMI, alongside its positive association with prostate-specific antigen, underscores the complex interplay between adipose tissue–derived factors, metabolic status, and prostate carcinogenesis. Importantly, multivariate analysis identified serum omentin-1 as an independent predictor of prostate cancer, suggesting its potential value as a complementary biomarker alongside established clinical indicators. These results imply that omentin-1 may reflect both oncological and metabolic alterations rather than tumor aggressiveness alone, which has relevant implications for risk stratification and a more integrated understanding of prostate cancer biology. Nevertheless, the cross-sectional nature of the study limits causal interpretation. Future research should therefore focus on large-scale longitudinal and mechanistic studies to clarify the causal role of omentin-1 in prostate cancer development and progression, explore its interaction with metabolic pathways, and evaluate its utility in early diagnosis, prognosis, and potential therapeutic targeting.

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