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RESEARCH ARTICLE

Serum Omentin-1 Level as a Biomarker in Prostate Cancer

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Abstract

Objective: To evaluate serum omentin-1 levels in prostate cancer patients relative to healthy controls and to assess its variation between tumor stages and Gleason score categories. **Methodology:** This case-control research involved 176 participants (88 prostate cancer patients and 88 controls) from the National Teaching Oncology Hospital in Najaf, Iraq. Serum omentin-1 concentrations were verified by ELISA tests, and relationships with PSA, BMI were measured. ROC analysis was used to assess diagnostic efficacy. **Results:** Omentin-1 levels were markedly elevated in prostate cancer patients (43.4 ± 22.6 ng/mL) compared to controls (26.1 ± 6.6 ng/mL, $p < 0.01$). ROC analysis showed reasonable diagnostic accuracy (AUC = 0.73). **Conclusion:** Serum omentin-1 levels are elevated in prostate cancer, and no significant differences were observed across tumor stages or Gleason score categories, indicating that its elevation does not appear to be stage-dependent in this cohort. Further studies are needed to elucidate the mechanistic role of omentin-1 in prostate carcinogenesis, as well as its diagnostic and prognostic potential.

Keywords: Diagnostic biomarkers- Omentin-1- Prostate cancer

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Introduction

Prostate cancer (PCa) is the second most often diagnosed malignancy globally and the fifth leading cause of cancer-related mortality in males, with over 1,460,000 new cases and more than 396,000 deaths recorded in 2022 [1]. From a clinical perspective, PCa exhibits substantial variability; some individuals present with indolent tumors, whilst others progress to aggressive disease characterized by a pronounced propensity for progression and metastasis [2]. In the early stages, symptoms usually appear as lower urinary tract dysfunction, which includes more frequent urination, nocturia, and hesitation. In contrast, severe illness may manifest with urinary retention and bone metastases, mostly impacting the axial skeleton [3]. A full array of risk factors has been definitively established, including advancing age, African ancestry, specific germline mutations, elevated insulin-like growth factor (IGF) levels, and a family history of prostate cancer (PCa). Lifestyle factors, such as diets rich in saturated fats and red meat, tobacco use, alcohol intake, obesity, reduced physical activity, and environmental exposures to chemicals or radiation, may increase the risk of disease [4]. Despite extensive investigation, the etiology of prostate cancer (PCa) remains intricate, including genetic, hormonal, environmental, and inflammatory variables that provoke molecular changes leading to the malignant transformation of prostate epithelial cells [5]. In this context, chronic inflammation, triggered by infections, dietary factors, or hormonal imbalances, plays a substantial role in the etiology of prostate cancer (PCa). Inflammatory mediators induce the generation of reactive oxygen species (ROS), leading to DNA damage and epigenetic alterations. Proliferative inflammatory atrophy (PIA) lesions are considered potential precursors of high-grade prostatic intraepithelial neoplasia (PIN) and cancer [6].

Omentin-1, also termed intelectin-1, is a 34 kDa adipokine encoded by the ITLN1 gene on chromosome 1q21.3. It is the predominant isoform found in human plasma and is secreted mainly from visceral adipose tissue, with markedly higher expression compared to subcutaneous fat [7-11]. Beyond adipose tissue, it is also expressed in endothelial, mesothelial, vascular, and

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intestinal Paneth cells, exerting autocrine, paracrine, and endocrine functions [12]. Functionally, Omentin-1 modulates insulin and IGF-1 receptor signaling, contributes to glucose and energy metabolism, supports cardiovascular health, and exhibits anti-inflammatory and antioxidant effects [9, 13]. Its circulating levels are regulated by obesity, insulin sensitivity, inflammation, genetic factors, and hormones such as adiponectin, insulin, and fibroblast growth factor-21 [12, 14-16]. In oncology, Omentin-1 displays a context-dependent role. Elevated levels have been reported in mesothelioma, hepatocellular, prostate, colorectal, gastric, and pancreatic cancers, whereas decreased levels occur in renal cell carcinoma [17]. Mechanistically, it can influence tumor progression through apoptosis regulation, Akt pathway modulation, and angiogenic factor secretion, impacting both endothelial and stem cell activity Omentin-1 is regarded as a possible biomarker for cancer progression and prognosis, however its specific role in carcinogenesis is not well-defined. Despite increasing evidence indicating the significance of Omentin-1 in other malignancies, there is a paucity of data concerning its serum levels in prostate cancer patients and their potential correlation with clinical features [18]. The purpose of this research is to investigate the possibility of Omentin-1 as a biomarker for the development and prognosis of prostate cancer by measuring blood levels of the protein and characterizing their association with clinical variables.

Materials and Methods

Study Design

This case-control research was conducted at the National Teaching Oncology Hospital in Najaf, Iraq, from December 2024 to July 2025. This study included two groups: newly diagnosed prostate cancer patients and age-matched healthy controls. The Scientific Committee of the College of Medicine at the University of Basrah granted ethical clearance. Additionally, the hospital ethics board approved this study. All participants provided written informed consent after obtaining detailed information about the study's aims and methodologies.

There were eighty-eight participants, ranging in age from forty to eighty-five. Recently discovered prostate cancer that was confirmed by histopathology was detected in all patients. Inclusion was determined by having a PSA level higher than 4 ng/mL and not having had any kind of treatment for prostate cancer before, such as hormonal therapy, radiation, or chemotherapy. Meanwhile, 88 age-matched volunteers were included in the control group. Neither their medical history nor their clinical examinations have shown any signs of prostate cancer or benign prostatic hyperplasia (BPH).

Exclusion Criteria

Participants were excluded if they had any of the following:

1- Receiving curative medication for prostate cancer.

2- Patients with a history of other malignancies or previous prostate surgeries.

3- Acute infectious diseases.

4- Chronic liver and kidney diseases.

Physical and biochemical measurements

Age, smoking status, family history, and clinical data including comorbidities such as hypertension, diabetes mellitus, cardiovascular disease, and renal disease were all recorded from all participants. A person's body mass index (BMI) was determined by recording their anthropometric data, which included their weight and height. Prostate cancer patients had their Gleason scores and TNM stages documented.

Biochemical Analysis

Measurement of Serum Omentin-1

Following the manufacturer's instructions, serum omentin-1 concentrations were measured using a commercial ELISA kit (Cloud-Clone Corp, USA).

Other Biochemical Parameters

A fully automated VIDAS system developed by Biomerieux in France, which is based on the ELFA (Enzyme Linked Fluorescent Assay) concept, was used to measure PSA concentrations. The fully automated DRI-CHEM NX600 device (Fujifilm, Japan) was used to measure fasting blood glucose, serum urea, serum creatinine, total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL-C).

Statistical Analysis

All experiments were performed in triplicate, and the results were expressed as mean ± standard deviation. Data normality was assessed using the Shapiro–Wilk test, and homogeneity of variance was evaluated using Levene's test. Based on these assumptions, normally distributed variables such as age and BMI were compared using the independent t-test, while non-normally distributed biochemical parameters including Blood Pressure, serum omentin-1, PSA, fasting blood sugar, cholesterol, LDL, VLDL, triglycerides, HDL, creatinine, and BUN, as well as weight, height and Blood Pressure were analyzed using the Mann–Whitney U test. Categorical variables such as residency, smoking status, and comorbidities were assessed using the Chi-square test. Comparisons across patient subgroups for tumor stage and Gleason score were conducted using the Kruskal–Wallis test. Receiver-operating characteristic (ROC) analysis was employed to evaluate the diagnostic performance of serum omentin-1 and PSA in distinguishing patients from controls. A p-value < 0.05 was considered statistically significant. All analyses were conducted using IBM SPSS Statistics 23.0.

Results

Socio-demographic characteristics among the study groups

The socio-demographic features of prostate cancer Patients (88 participants) and a control group (88 healthy adults) are shown in Table 1. The research revealed significant differences in age (P=0.006) and body mass index (BMI) (P=0.009) between prostate cancer patients and healthy controls. In contrast, there were no statistically significant differences between the groups regarding residency (urban vs. rural, P=0.7), smoking status (P=0.8), or comorbidities (P=0.2). Similarly, systolic and diastolic blood pressures did not differ significantly between the two groups (P=0.3 and P=0.8, respectively).

Biochemical parameters of study participants

Table 2, serum omentin-1 levels were significantly higher in prostate cancer patients compared with healthy controls (43.4 ± 22.6 vs. 26.1 ± 6.6 ng/mL; P < 0.01). Likewise, PSA levels showed a marked increase in the patient group (32.1 ± 31.1 vs. 1.68 ± 0.9 ng/mL; P < 0.01). For the remaining biochemical parameters, no statistically significant differences were observed between patients and controls, including fasting blood sugar (P = 0.7), cholesterol (P = 0.6), triglycerides (P = 0.2), HDL (P = 0.2), LDL (P = 0.4), VLDL (P = 0.2), serum creatinine (P = 0.5), and BUN (P = 0.1).

Serum omentin levels in prostate cancer patients, stratified by Gleason score and tumor stage

In Table 3, Serum omentin-1 levels showed no significant variation across tumor stages (T1–T4) or Gleason score categories. Patients in different stages exhibited closely similar mean values (Kruskal–Wallis p = 0.753), and the same pattern was observed across Gleason grades, where well-, moderately-, and poorly differentiated tumors showed nearly identical concentrations (p = 0.746).

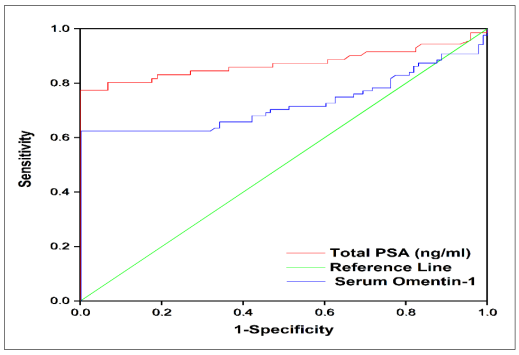

Figure 1. ROC Curve Analysis of Serum Omentin (Blue line), and PSA (Red line) in the Study Population

ROC Curve Analysis

The diagnostic performance of serum Omentin-1 and PSA in distinguishing prostate cancer patients from healthy controls was evaluated using ROC analysis.

Serum Omentin-1: AUC = 0.73, optimal cut-off = 34.6 ng/ml, sensitivity = 63%, specificity = 88%.

PSA: AUC = 0.87, optimal cut-off = 2.04 ng/ml, sensitivity = 85%, specificity = 70%.

These results are summarized in Table 4 and illustrated in Figure 1.

Discussion

Prostate cancer (PCa) continues to represent a major public health burden, accounting for approximately 19% of all new cancer cases and 9% of cancer-related deaths in men globally [19]. We investigated the clinical significance of serum omentin-1 in PCa patients vs healthy controls in an effort to determine its possible role as a biomarker. Serum levels of omentin-1 were markedly elevated in prostate cancer patients (43.4 ± 22.6 ng/mL) when compared with healthy controls (26.1 ± 6.6 ng/mL, P < 0.01). The very high serum values likely point to its potential involvement in the pathophysiology of prostate

Table 1. Sociodemographic Characteristics of Study Participants

Parameters	Subgroup	Healthy Control N=88	Patients N=88	P-Value
Age	-	59.7±13.4	65.2±9.2	0.006
BMI	-	27.02±3.4	25.6±3.7	0.009
Blood Pressure	Systolic	124±11.5	127±14.9	0.3
	Diastolic	80.3±8.05	80.4±8.69	0.8
Residency	Urban	48 (54.5%)	50 (56.8%)	0.7
	Rural	40 (45.5%)	38 (43.2%)	
	Non-smoker	50 (56.8%)	52 (59.1%)	0.8
Smoking status	Ex-smoker	8 (9.1%)	9 (10.2%)	
	Smoker	30 (34.1%)	27 (30.7%)	
	DM	13 (14.7%)	11(12.5%)	0.2
Comorbidities	HTN	8 (9.1%)	5 (5.7%)	
	HTN+DM	4 (4.5%)	8 (9.1%)	
	None	63 (71.5%)	64 (72.7%)	

Significant value: p < 0.05, p < 0.01, p < 0.001; Non-significant value: p ≥ 0.05; Independent t-test / Mann– Whitney U / Chi-square test

Table 2. Biochemical Parameters of Participants in Different Subgroups

Parameters	Healthy Control N=88	Patients N=88	P-Value
Serum Omentin-1 (ng/ml)	26.1±6.6	43.4±22.6	<0.01
Total PSA (ng/ml)	1.68±0.9	32.1±31.1	<0.01
FBS (mg/dl)	105.5±18.6	106.6±21.5	0.7
Cholesterol (mg/dl)	196.9± 23.6	198.1± 33.4	0.6
Triglycerides (mg/dl)	138.1± 40.7	134.6± 51.5	0.2
HDL (mg/dl)	49.7± 9.8	48.04± 12.3	0.2
LDL (mg/dl)	119.6± 25.8	123.14± 35.5	0.4
VLDL (mg/dl)	27.6± 8.1	26.9± 10.3	0.2
S-creatinine (mg/dl)	0.86± 0.16	0.87± 0.24	0.5
BUN (mg/dl)	35.2± 10.3	33.46± 12.46	0.1

*Significant value: $p < 0.05$, $p < 0.01$, $p < 0.001$; * Non-significant value: $p \geq 0.05$. * All analyses for these variables were performed using the Mann–Whitney U test.

Table 3. Serum Omentin Levels in Prostate Cancer Patients, Stratified by Gleason Score and Tumor Stage

Group	N (patients) (88)	Category	Serum Omentin (ng/ml)	
			Mean ± SD	P-Value
Tumor Stage	10	T1	49.5±18.3	0.753
	25	T2	41.5±20.1	
	30	T3	43.6±24	
	23	T4	42.5±24.6	
Gleason Score	7	Well-differentiated (score <7)	44.8± 21.9	0.746
	38	Moderately differentiated (score =7)	45.3±23	
	43	Poorly differentiated (score >7)	41.8±23	

*Kruskal–Wallis test were used for comparisons among tumor stages and Gleason score groups. * Statistically significant at $p < 0.05$; * Statistically non-significant at $p > 0.05$

cancer disease and its utility as a biomarker. Findings are in line with those of Zhou et al. [7], Uyeturk et al., and Fryczkowski et al., that demonstrated pronounced omentin-1 levels in PCa that could be established as a non-invasive diagnostic marker [20,21]. When serum omentin-1 levels were compared across prostate cancer stages (T1–T4), no statistically significant differences were observed among the groups. Although patients in stage T1 showed numerically higher mean concentrations compared with more advanced stages, these variations did not reach statistical significance, indicating that the observed pattern may simply reflect natural biological variability rather than a stage-dependent effect. Accordingly, the current data do not support any definitive interpretation regarding omentin-1 behavior across tumor stages. Similarly, when stratified by Gleason score, omentin-1 levels did not differ significantly between well-differentiated, moderately differentiated, and poorly differentiated tumors. These findings align with those reported by Uyeturk et al. [20], who also found no association between circulating omentin-1 and histopathological grade, suggesting that omentin-1 is unlikely to reflect tumor aggressiveness. Evidence from other malignancies further supports the context-dependent and variable behavior of omentin-1. Shen et al. [22], in renal cancer and Fazeli et al. [23], in colorectal cancer both reported inconsistent relationships between omentin-1 and tumor stage, reinforcing that this adipokine may not follow a uniform pattern across different cancers. Experimental data offer several mechanistic hypotheses but do not establish causality in humans. Mogal et al. [24], reported that omentin suppresses prostate cell proliferation by downregulating the tumor suppressor gene Nkx3.1, while Cheng et al. [25], demonstrated that mesenchymal stem cells in the tumor microenvironment drive androgen-dependent PCa cells toward androgen-independent, more aggressive forms. This supports the concept that decreased omentin-1 levels in advanced stages may reflect the loss of protective metabolic regulation.

Apart from prostate carcinoma, a number of researchers have investigated the role of omentin in other cancers. Omentin inhibited hepatocellular carcinoma formation by activation of the JNK pathway and by upregulating p53 [26], and Ji et al. [27], observed enhanced apoptosis in colon cancer stem cells. Yet contrary evidence does exist: Dec et al. [18], demonstrated that omentin promoted colon cancer growth, and Zhang et al. [28], shows that colorectal carcinoma cells secrete omentin locally, which may accelerate tumorigenesis through glucose uptake [29] and PI3K/Akt signaling [30], from the well-established pathway that promotes prostate cancer [31]. Omentin-1 is primarily synthesized by visceral adipose tissue, where the expression of PI3K/Akt contributes to mesenchymal

Table 4. Receiver-operating Characteristic (ROC) Curve Analysis for Serum Omentin and PSA in the Study Population

Markers	AUC	p-value	Cut-off point	Sensitivity	Specificity
Serum omentin	0.73	<0.0001	34.6	0.63	0.88
PSA	0.87	<0.0001	2.04	0.85	0.7

stem cell survival [18]. Moreover, other components of the tumor microenvironment (adipocytes, macrophages and endothelial cells) release proangiogenic mediators (VEGF and fibroblast growth factors) that promote tumor invasion and progression [32, 33].

We obtained findings based on the ROC analysis which indicate that serum omentin-1 had a moderate discriminative performance, with an AUC of 0.73, sensitivity of 63%, and specificity of 88%, corresponding to an optimal cut-off value of 34.6 ng/mL. In contrast, PSA showed superior diagnostic accuracy, achieving an AUC of 0.87 with a cut-off value of 2.04 ng/mL (sensitivity 85%, specificity 70%). These results indicate that although PSA remains the more robust marker, omentin-1 may still provide additional diagnostic value as a complementary biomarker, This observation agrees with Fryczkowski et al.'s findings [21] on omentin's diagnostic potential as an adjunct biomarker to PSA.

Molecularly, prior studies imply that omentin can modulate several pathways. Zhang et al. [26], showed that omentin suppresses hepatocellular carcinoma growth by the upregulation of p21 and p53 proteins, induces apoptosis by promoting Bax/Bcl-2 transcription, and inhibits caspase-3 activation. Omentin also was found to activate the Akt pathway and modulate PI3K/Akt-eNOS-Ras signaling that could be implicated, and promote or inhibit cancer depending on cellular context [18, 27-34]. While these pathways enable theoretically a rationale for omentin-1's potential role in cancer biology, the current study reveals no direct mechanistic relationships. Our data show serum omentin-1 is high in PCa and different between diseases; however, these observations indicate associations and not causality. Such mechanistic studies along with longitudinal designs are needed to better elucidate whether omentin-1 exerts a causal role in PCa progression or represents the only metabolic perturbations of the disease. The present case–control study is limited in some aspects as follows:

First: The design enables comparisons between prostate cancer patient's vs healthy controls, yet does not allow for causal inference in terms of relation of serum omentin-1 levels to the development/progression of prostate cancer. Second: The trial was performed in only one city, and the sample had a small size, so the results may not be applicable over larger populations. Third: Dietary habits, frequency of physical activity and unmeasured metabolic or hormonal parameters were not fully controlled and could have affected omentin-1 levels.

Finally, the study had no longitudinal follow-up to assess temporal alteration of omentin-1 at different stages of disease onset and after different interventions. Nevertheless, multicenter prospective studies are required to confirm these observations and to better clarify the potential clinical relevance of omentin-1 in PCa.

In conclusion, serum omentin-1 was significantly elevated in prostate cancer patients compared to healthy controls, suggesting a possible association between elevated omentin-1 levels and the presence of prostate malignancy. No significant differences were observed with respect to prostate cancer stage and Gleason score levels in this cohort, however, indicating that omentin-1 does not reflect disease stage or histological aggressiveness. Overall, the findings imply omentin-1 could be used as an adjunct biomarker in prostate cancer detection. However, additional work is necessary to elucidate its biological function and to evaluate a reasonable diagnostic or prognostic potential in clinical practice.

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Author contributions

Conceptualization: AMM and AIA; Methodology: AMM and AIA; Software: AF; Validation: HJS; Formal Analysis: AMM and AIA; Investigation: AMM; Supervision, Project Administration, and Data Curation: AIA and HJS; Writing – Original Draft Preparation: AMM, AIA. Writing – Review & Editing: AMM and AIA. All authors have read and approved the final version of the manuscript

Conflicts of interest

The authors deny there are any financial conflicts of interest.

Data availability upon reasonable request.

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