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Serum Resistin in Iraqi Men with Prostate Cancer: Associations with Renal Function and Serum Electrolytes

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ABSTRACT:

Background: Resistin is an inflammatory adipokine produced mainly by monocytes and macrophages in humans. Evidence linking circulating resistin to prostate cancer is limited and inconsistent.

Objective: To compare serum resistin between men with prostate cancer and controls and to examine its association with creatinine, urea, sodium, and potassium.

Methods: A case-control study was conducted in men with prostate cancer and apparently healthy controls. Serum resistin and the selected biochemical variables were measured, group values were compared, and correlations with resistin were calculated.

Results: The reported resistin value was 34.01 ng/mL in the prostate cancer group, compared with a control interval of 7-22 ng/mL ($p <= 0.01$). Creatinine and urea were higher in patients, whereas sodium and potassium were lower. Resistin correlated positively with creatinine ($r = 0.401$), urea ($r = 0.322$), potassium ($r = 0.207$), and sodium ($r = 0.158$).

Conclusion: Higher resistin accompanied renal and electrolyte abnormalities in the patient group. Renal impairment may had been influence circulating resistin; therefore, these current findings should be interpreted as an associations rather than evidence of diagnostic or prognostic utility. Confirmation requires a larger, clinically characterized cohort and multivariable analysis.

1. INTRODUCTION

Prostate cancer is a disease with high clinical heterogeneity, ranging from indolent localized tumors to overt metastatic disease. Although an elevated serum concentration of prostate-specific antigen continues to be cornerstone detection and follow-up, it is not specific for malignancy. Benign prostatic hyperplasia and prostatitis also elevate PSA, motivating exploration of markers that reflect alternative features of tumor biology [1, 2].

Adipokines represent good candidates, as they link metabolism to immune signaling and the tumor microenvironment. While resistin was first discovered by your connection with insulin resistance, in humans the majority of circulating protein is produced fibroblasts, monocytes and macrophages. It can enhance inflammatory signaling, and dysregulated adipokine expression has been associated with tumor growth, angiogenesis and invasion [3–8].

The literature on prostate cancer does not tell a single consistent story. Chung et al. reported higher expression of resistin in malignant prostate tissue compared to benign, with expression associated with tumor grade and stage [1,3]. By contrast, Housa

et al. reported no difference in circulating resistin between men with and without benign prostatic hyperplasia and non-metastatic prostate cancer [2,4]. Later experimental work indicated that resistin stimulates migration of PC3 prostate cancer cells in vitro and invasion of breast cancer cells [9]; however, the in vitro results cannot define clinical utility as a serum marker [10].

Population-specific clinical data remain scarce. This study therefore compared serum resistin in Iraqi men with prostate cancer, and controls, and examined the association of resistin with creatinine, urea sodium and potassium.

2. MATERIALS AND METHODS

2.1 Study design and setting

This case-control study was carried out at AL-Amal Hospital Baghdad city/Iraq between February to Decembe2024. The study included 100 man with a documented diagnosis of prostate cancer and 100 healthy man as control.

2.2 Eligibility criteria

Men would be eligible for the patient group if prostate adenocarcinoma had been confirmed histopathologically by examine for prostate biopsy specimens. Control participants had no known history of prostate cancer and no acute inflammatory illness at recruitment. The exclusion criteria for both groups were acute infection, systemic inflammatory disease, chronic kidney disease unrelated to prostate cancer, severe liver disease, recent surgery or trauma, use of medicines expected to substantially alter inflammatory biomarkers, and incomplete laboratory data.

2.3 Blood sampling and laboratory measurements

Venous blood was obtained from participants. Serum resistin was quantified by an enzyme-linked immunosorbent assay (ELISA) using kit supplied by Biological company from United States. The normal value of resistin for males: 3-13 ng /ml.

Creatinine, urea, sodium, and potassium were measured using The standard methods to measure crenatinie and urea in blood serum rely on colorimetric, enzymatic, and mass spectrometry techniques to assess kidney function, sodium and potassium measure in blood serum by Ion-selective electrode (ISE) analysis. Resistin is reported in ng/mL, creatinine in mg/dL, urea in mg/dL, and sodium and potassium in mEq/L.

Statistical Analysis

Statistical analysis: SPSS 26. Data were represented as mean ± SD. Differences in study groups were assessed with a Student's independent t-test on the data sets. Pearson correlation coefficients were also calculated to measure relationships between prolactin concentrations and thyroid hormone parameters. Statistical significance was defined at $p < 0.05$.

3. RESULTS AND DISCUSSION

3.1 Biochemical findings

The values supplied for analysis are summarized in Table 1. Compared with the control values, the patient group had higher creatinine (3.56 vs 0.987 mg/dL) and urea (35.31 mg/dL vs a reported control range of 8-24 mg/dL). Sodium and potassium were lower in the patient group (120 vs 140 mEq/L and 1.12 vs 4.5 mEq/L, respectively). The reported resistin value was 34.01 ng/mL in patients; the control value was provided as a range of 7-22 ng/mL. Each comparison was recorded as $p <= 0.01$ in the source table.

Table 1. Reported biochemical values in the control and prostate cancer groups

Parameter	Control value	Patient value	Reported p-value
Creatinine (mg/dL)	0.987	3.56	<= 0.01
Urea (mg/dL)	8-24*	35.31	<= 0.01

Parameter	Control value	Patient value	Reported p-value
Sodium (mEq/L)	140	120	<= 0.01
Potassium (mEq/L)	4.5	1.12	<= 0.01
Resistin (ng/mL)	7-22*	34.01	<= 0.01

**Presented as a range in the source file. Before submission, specify the group sample sizes and whether every value represents a mean, median, reference interval, or another summary measure; add SD or IQR where appropriate.*

3.2 Correlation with serum resistin

All four coefficients were positive. The largest association was with creatinine (r = 0.401), followed by urea (r = 0.322), potassium (r = 0.207), and sodium (r = 0.158). On conventional descriptive thresholds, the creatinine association was moderate, those for urea and potassium were weak, and that for sodium was very weak. Statistical significance and confidence intervals were not provided for these coefficients.

Table 2. Correlations between serum resistin and biochemical variables

Variable	**Correlation (r)**	**Descriptive strength**
Creatinine (mg/dL)	0.401	Moderate positive
Urea (mg/dL)	0.322	Weak positive
Sodium (mEq/L)	0.158	Very weak positive
Potassium (mEq/L)	0.207	Weak positive

4. DISCUSSION

Two observations stand out in these data: serum resistin was higher in the prostate cancer group, and the strongest recorded correlation was between resistin and creatinine. The second observation is an important way when interpreting the first. When a patient creatinine value of 3.56 mg/dL, the renal dysfunction is plausible source of a confounding, and the available results cannot establish whether the resistin concentration difference is related to malignancy, an impaired renal function, a systemic inflammation, or a combination of these factors.

Prior the findings support biological interest in resistin but also argue for caution. Increased resistin staining has been documented in prostate cancer tissue, with greater expression in more advanced or poorly differentiated tumors [1]. Laboratory experiments further suggest that resistin can increase the migratory and invasive behavior of PC3 cells [10]. Neither observation demonstrates that serum resistin can distinguish prostate cancer from non-malignant disease. Indeed, Housa et al. reported similar circulating concentrations in benign prostatic hyperplasia and non-metastatic prostate cancer [2]. Contradictory observations between studies may illustrate the tumor burden, inflammatory state, adiposity, renal function, treatment exposure and assay characteristics.

The renal outcomes merit independent consideration. Patients had higher levels of creatinine and urea, both of which correlated positively with resistin. Detecting temporal direction is not possible as the design was observational and measurements were reported at one time point. Future analyses should use resistin adjusted for estimated glomerular filtration rate and potential relevant comorbidities, rather than relying on the unadjusted association to favour a possible cancer-specific mechanism.

There was also a significant difference in electrolyte values. Serum sodium of 120 mEq/L along with potation of 1.12 mEq/L are extreme laboratory derangements in practice [38]. Thus, before publication, the original records, measurement units and sample handling should be verified for transcription errors. If the values are confirmed then data on symptoms, treatment and history of renal or endocrine disease and timing of sampling will be required to support an interpretation. The poor correlations with resistin do not, by themselves, establish a causal role for resistin in either electrolyte abnormality.Taken together, the data justify further study but not yet the designation of resistin as a diagnostic or prognostic biomarker. A clinically useful biomarker would need to add information beyond PSA, tumor stage, Gleason score, renal function, inflammatory status, and body composition. Contemporary work on adipokines and periprostatic adipose tissue supports this broader, multivariable approach [6,7,9,11].

5. LIMITATIONS

Interpretation is constrained by missing sample sizes, participant characteristics, recruitment details, measures of dispersion, exact p-values, and confidence intervals. PSA, Gleason score, tumor stage, treatment status, body mass index, inflammatory markers, and estimated glomerular filtration rate were not supplied. It is also unclear whether the control entries shown as ranges are observed group results or laboratory reference intervals. Finally, the correlation method and the statistical significance of the correlation coefficients were not reported. These omissions preclude assessment of confounding, diagnostic accuracy, and independent association.

6. CONCLUSION

In the same dataset men with prostate cancer, we found a significantly higher serum resistin value in these patients in comparison to controls, as well as impaired kidney function (higher creatinine and urea), and less sodium and potassium. Creatinine was the metabolite that correlated more closely with resistin. Conclusions: The results suggest that resistin may be associated with the pattern of renal-metabolic disturbance, but it is not specific to prostate cancer and does not have potential diagnostic or prognostic value. The source data and analysis require validation in a larger, characterized cohort.

7. PRIORITIES FOR FURTHER STUDY

Recruit a sufficiently powered sample and report all demographic & clinical characteristics for both groups.

To study the association of resistin with PSA, Gleason score, tumor stage, metastasis, treatment status, body mass index and inflammatory markers.

Patients are stratified according to renal function using estimated glomerular filtration rate and multivariable models adjusted for renal insufficiency.

Use ROC analysis only after you have full characterization of dataset and reference group; report effect sizes, 95% confidence intervales, exact p-values.

Independent validation of any diagnostic threshold identified with relevant non-malignant prostatic disease.

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AUTHOR CONTRIBUTIONS

ammal esmaeel ibrahim: Conceptualization, Investigation.Methodology, Laboratory Analysis.

Data Curation,

Saba hameed majeed: Formal Analysis.Supervision, Writing—Review & Editing.

CONFLICT OF INTEREST

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

DATA AVAILABILITY STATEMENT

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

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