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Resistin Level as a Marker of Malnutrition in Diabetic Patients With Chronic Kidney Disease

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ABSTRACT

Background: Malnutrition is a frequent and serious issue in chronic kidney disease (CKD), especially in diabetic patients, and resistin has been suggested as a biomarker linked to insulin resistance, inflammation, and declining nutritional status.

Objective: To evaluate the role of serum resistin as a marker of malnutrition in diabetic cases with chronic kidney disease.

Patients and Methods: This prospective comparative research included 110 diabetic cases with chronic kidney disease recruited from Menoufia University and Shebeen El-Kom Teaching Hospitals. Patients were classified into two groups based on the Subjective Global Assessment (SGA): Group A (well-nourished, n = 55) and Group B (malnourished, n = 55). Resistin concentrations were calculated utilizing enzyme-linked immunosorbent assay (ELISA). Insulin resistance was assessed applying the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). Other laboratory markers included protein-to-creatinine ratio, aspartate aminotransferase (AST), and total iron-binding capacity (TIBC).

Results: Malnourished patients were significantly older (61.4 ± 11.9 vs. 57.2 ± 8.3 years, $p = 0.03$) and had an elevated occurrence of hypertension and autosomal dominant polycystic kidney disease. They demonstrated greater weight loss, decreased dietary intake, and reduced functional capacity (all $p < 0.001$). Laboratory findings showed higher levels of HOMA-IR, protein-to-creatinine ratio, AST, TIBC, and markedly elevated resistin (31.1 ± 8.8 vs. 5.37 ± 1.8 ng/mL, $p < 0.001$).

Conclusion: Serum resistin is markedly higher in malnourished diabetic CKD patients and correlates with worsening nutritional and metabolic status, suggesting its value as an early marker of malnutrition.

Keywords: Resistin, Malnutrition, Diabetes Mellitus, Chronic Kidney Disease

INTRODUCTION

Resistin is a 12.5 kDa cysteine-rich polypeptide that is part of the Resistin-Like Molecules (RELMs) family, a collection of proteins that trigger inflammatory processes. In humans, resistin is predominantly produced by granulocytes, monocytes, macrophages, and cells of the bone marrow. It was additionally identified in the thymus, pituitary gland, hypothalamus, digestive system, skeletal muscle, placenta, and pancreas [(1)].

In humans, resistin is predominantly produced by inflammatory cells, serving as a biomarker for inflammation. While a definitive single receptor for resistin has not been universally identified, several potential receptor interactions—such as those involving CAP1 and Toll-like receptor 4 (TLR4)—have been proposed, reflecting the ongoing complexity of resistin signaling mechanisms. Adenyl cyclase-associated protein-1 (CAP1) and TLR4 are likely functional receptors for resistin. The interaction between resistin and Toll-like receptor 4, along with CAP1, leads to vascular dysfunction and elevated serum inflammatory cytokine levels [(2)].

Toll-like receptor 4, a member of the TLR family, is activated when it recognizes pathogen-associated molecular patterns like lipopolysaccharide (LPS). LPS binding to cell-surface TLR4 triggers TLR4 signaling by promoting interaction with myeloid differentiation factor 2 (MD2) and recruitment of the adaptor protein MyD88, resulting in cytoplasmic-to-nuclear translocation of the transcription factor NF-κB [(3,4)]. Functional studies in HEK cells expressing TLR4 have shown that resistin binds TLR4 and activates its downstream signaling, thereby enhancing NF-κB activation. Resistin appears to bind specifically to TLR4, as it does not bind or activate TLR2 [(5)].

In human monocytes, resistin also engages CAP-1, which initiates a signaling cascade that raises cyclic AMP and activates protein kinase A, culminating in activation of the proinflammatory NF-κB transcription factor [(6)].

Initially, resistin was primarily associated with the progression of insulin resistance. Resistin is currently recognized for its elevated expression in several inflammatory disorders, including septic shock, acute pancreatitis, and osteoarthritis, as well as in autoimmune conditions like lupus erythematosus and rheumatoid arthritis [(7)].

Resistin concentrations are elevated in obese persons. Its level are positively correlated with waist-to-hip ratio and body mass index (BMI). Individuals with metabolic syndrome exhibit elevated levels of resistin in comparison with healthy counterparts. Resistin levels are increased in type 2 diabetes mellitus [(8)].

The etiology of elevated resistin levels remains unclear; however, potential explanations encompass

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diminished renal clearance and subclinical inflammation, which may occur even in the early stages of chronic kidney disease. Furthermore, resistin is believed to induce glomerular inflammation by elevating the synthesis of inflammatory cytokines. Type 2 diabetes mellitus is a progressively prevalent etiology of renal disease and is characterized by the coexistence of multiple metabolic and hormonal diseases that influence resistin concentrations [(9)].

The SGA is a nutrition evaluation instrument that refers to a total assessment of a case's history and physical examination that utilizes structured clinical variables to identify malnutrition. The SGA is recognized as a valid and reliable instrument for predicting death and morbidity related to malnutrition [(10)].

Cases with chronic kidney disease are at significant risk for malnutrition, marked by micronutrient deficiency and protein-energy wasting. The pathogenic mechanisms of malnutrition in chronic kidney disease are complex and include a combination of various pathophysiological changes, such as reduced nutrient intake and appetite, metabolic imbalances, hormonal derangements, heightened catabolism, inflammation, and dialysis-related anomalies. Malnutrition elevates the probability of morbidity, death, and the overall illness burden in these cases [(11,12)].

The goal of this work was to assess the effect of resistin level as a marker of malnutrition in diabetic CKD cases.

PATIENTS AND METHODS

Type of study: This prospective comparative study has been conducted between July 2024 and June 2025, on one hundred and ten patients with diabetic CKD who come to follow at outpatient clinics of Menoufia University Hospitals and Shebeen El-Kom Teaching Hospital.

The study population:

Inclusion criteria: Cases aged ≥ 20 years with diabetes and chronic kidney disease with different stages.

Exclusion criteria: Younger than twenty years, Patients with acute diabetic complications or with insufficient clinical information, Cases with acute kidney injury and cases with acute kidney injury on top of CKD, malignancy, liver disease, and active infectious disease.

Sample Size:

According to a study of previous literature**, Kaynar *et al.*[(13)]** identified a substantial positive association between the presence of protein-energy wasting (PEW) and serum resistin (r: 0.267, p: 0.001).

The sample size was determined utilizing statistical methods and the Sample Size Pro program version 6, with a minimum sample size of 110. The study power was eighty percent, while the confidence level was ninety-five percent.

The sample size (n=110) was determined based on detecting a significant association between serum resistin concentrations and malnutrition among diabetic CKD cases, with a study power of 80% and a confidence level of 95%.

Patients were divided into 2 groups: Group A: 55 well-nourished diabetic CKD patients and Group B: 55 malnourished diabetic CKD patients.

Ethical Consideration:

Written informed consent has been gathered from all participants, and the research was permitted by the Ethical Committee of Menoufia University, Faculty of Medicine (IRP: 8/2023INTM6). Participation in the research was voluntary, and each patient had the right to withdraw at any time. Confidentiality and anonymity of the participants were assured through coding. The Helsinki Declaration was followed throughout the study's conduct.

METHODS

Clinical examination: Each participant underwent complete history taking, a general examination, and a complete physical examination. Vital signs (blood pressure, temperature, respiratory rate, and heart rate) were recorded.

Subjective Global Assessment (SGA), which included five nutritional features—clinical history (unintentional loss of weight, reduced nutrient intake, functional capacity, symptoms affecting oral intake, and metabolic demand) and physical examination (subcutaneous fat loss, muscle wasting, and fluid accumulation) was performed. To ensure inter-rater reliability, the SGA was administered by two trained physicians who were standardized in assessment procedures prior to the study. Waist and mid-arm circumferences and hand grip were also assessed.

Laboratory investigations:

Sampling: after 12-hour fasting, 7 ml of blood was obtained and divided into 3 tubes.

Tube 1: 1 ml was collected in an ethylenediaminetetraacetic acid (EDTA) tube for blood hemoglobin. Tube 2: 1.6 ml whole blood with additional sodium citrate sterile tube for measuring erythrocyte sedimentation rate (ESR).

Tube 3: 4.4 ml was collected in a plain tube and the serum was separated by centrifugation for measurement of biochemical laboratory investigations. For urinary albumin/creatinine ratio (UACR) urine specimen was collected to measure albumin creatinine in urine.

Laboratory investigations: fasting blood glucose (FBG), serum creatinine, serum electrolytes (sodium, potassium, calcium, and phosphorus), liver function tests (AST and ALT and, albumin), total cholesterol, triglycerides, serum iron, ferritin, total iron binding capacity and intact parathyroid hormone (iPTH) were assayed by Beckman Coulter AU680 analyzers **(Beckman Coulter, Indianapolis, USA).**

ESR was done by **Westergren method.** The quantification of C reactive-protein (CRP) determined by the nephelometric technique utilizing Mispa-i2 **(Agape Diagnostics, Switzerland).**

eGFR has been estimated with regard to the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation **[(14)].**

Serum insulin, C-peptide, and resistin were evaluated via ELISA kit with regard to the manufacturer's instructions **(Sunlong Biotech Co., Ltd., Zhejiang, China**).

FBG and fasting insulin concentrations have been utilzed to measure homeostasis model assessment of insulin resistance (HOMA-IR) determined by the following formula: HOMA-IR = fasting insulin (μIU/ml) × fasting glucose (mg/dl)/405 **[(15)]**. Insulin resistance (IR) was defined as HOMA-IR ≥1.7 **[(16)]**.

Primary Outcomes: The primary result was to evaluate the effect of resistin levels as a marker of malnutrition in diabetic CKD cases.

Secondary Outcomes: The 2^{ry} outcome was to compare among the two groups regarding resistin concentration as a marker of malnutrition.

Statistical analysis

Information have been fed to the computer and analyzed utilizing IBM SPSS software package version 22.0. (Armonk, NY: IBM Corp). Qualitative information was described utilizing percent and number. The Kolmogorov-Smirnov test and Shapiro-Wilk test has been applied to verify the normality of distribution. Quantitative information has been described utilizing mean, standard deviation. Significance of the obtained results was judged at the 5% level.

The applied tests were: Chi-square test for categorical variables, to compare between various groups. Independent Sample T test (T): when comparing between two groups (for normal Continuous quantitative data). Mann-Whitney U Test: when comparing among both groups (for abnormal Continuous quantitative data). P-value: level of significance: P-value above 0.05: Non-significant (NS), P-value under 0.05: Significant (S). P-value under 0.001: Highly significant (HS)

RESULTS

The malnourished group was significantly older, had a significantly shorter CKD duration, and showed a significantly higher occurrence of hypertension and autosomal dominant polycystic kidney disease (ADPKD) compared to the well-nourished group. Other demographic, medical, and family history characteristics showed a statistically insignificant variances among both groups.

Table (1): Distribution of patient characteristics among the examined groups.

		Group A (Well Nourished) Number =55	Group B (Malnourished) Number =55	Test	P value
Age (Mean± SD)		57.18±8.3	61.4±11.9	T=2.137	**0.03***
Sex	Female	30 (54.5%)	36 (65.5%)	X^2= 1.364	0.24
	Male	25 (45.5%)	19 (34.5%)		
CKD Duration in years Median (Range)		3(0.5-9)	2(0.5-9)	U= -2.40	**0.016***
Statins	Atorvastatin	2 (3.6%)	0 (0.0%)	X^2= 2.037	0.15
Medical History	Diabetes Mellitus	55 (100%)	55 (100%)	X^2=0.00	1.00
	Hypertension	4 (7.2%)	0 (0.0%)	X^2= 4.151	**0.04***
	Hysterectomy (fibroid)	1 (1.8%)	0 (0.0%)	X^2= 1.009	0.32
	Joint Pain (analgesic abuse)	1 (1.8%)	0 (0.0%)	X^2= 1.009	0.32
	Ischemic Cardiomyopathy	1 (1.8%)	0 (0.0%)	X^2= 1.009	0.32
	FMF	1 (1.8%)	0 (0.0%)	X^2= 1.009	0.31
	Ischemic Heart Disease	5 (9.1%)	9 (16.4%)	X^2=1.31	0.25
	Breast Benign Mass	2 (3.6%)	0 (0.0%)	X^2=2.037	0.15
	Heart Failure	3 (5.5%)	1 (1.8%)	X^2=1.038	0.31
	APCKD	5 (9.1%)	0 (0.0%)	X^2=5.238	**0.02***
	COPD	1 (1.8%)	0 (0.0%)	X^2= 1.009	0.32
	Gout	2 (3.6%)	0 (0.0%)	X^2=2.037	0.15
	Bronchial Asthma	4 (7.2%)	2 (3.6%)	X^2=0.705	0.401
	Renal Stones	5 (9.1%)	3 (5.5%)	X^2=0.539	0.46
	Obstructive Uropathy	3 (5.5%)	0 (0.0%)	X^2=3.084	0.08
	Benign Renal Mass, Hernioplasty	2 (3.6%)	0 (0.0%)	X^2=2.037	0.15
	Stroke	3 (5.5%)	2 (3.6%)	X^2=0.21	0.64
	BPH	2 (3.6%)	2 (3.6%)	X^2=0.00	1.00
	CABG	2 (3.6%)	0 (0.0%)	X^2=2.037	0.15
	SLE	2 (3.6%)	2 (3.6%)	X^2=0.00	1.00
	Ischemic Heart Disease	1 (1.8%)	0 (0.0%)	X^2= 1.009	0.32
	STEMI	1 (1.8%)	0 (0.0%)	X^2= 1.009	0.32
	Atrial Fibrillation	0 (0.0%)	1 (1.8%)	X^2= 1.009	0.32
	Diabetic foot (amputation)	0 (0.0%)	2 (3.6%)	X^2=2.037	0.15
	Nephrotic Syndrome	0 (0.0%)	2 (3.6%)	X^2=2.037	0.15
	Hypothyroid	0 (0.0%)	3 (5.5%)	X^2=3.084	0.08
	Typhoid fever	0 (0.0%)	1 (1.8%)	X^2= 1.009	0.32
	Diabetic foot, Lt Shoulder Fixation	0 (0.0%)	2 (3.6%)	X^2=2.037	0.15
	Double j Stent	0 (0.0%)	3 (5.5%)	X^2=3.084	0.08
	Parkinsonism	0 (0.0%)	2 (3.6%)	X^2=2.037	0.15
	Facial Palsy	0 (0.0%)	1 (1.8%)	X^2= 1.009	0.32
	Recurrent ascites	0 (0.0%)	1 (1.8%)	X^2= 1.009	0.32
Family History	ADPKD	5 (9.1%)	0 (0.0%)	X^2= 5.238	**0.02***
	Diabetes Mellitus	50 (90.9%)	55 (100%)	X^2= 5.238	**0.02***
	Hypertension	37 (67.3%)	41 (74.5%)	X^2= 0.705	0.401
	Ischemic Heart Disease	7 (12.7%)	10 (18.2%)	X^2= 0.626	0.42
	Bronchial Asthma	2 (3.6%)	2 (3.6%)	X^2=0.00	1.00
	SLE	0 (0.0%)	2 (3.6%)	X^2= 2.037	0.153

*: Significant, ADPKD: Autosomal Dominant Polycystic Kidney Disease, APCKD: Adult Polycystic Kidney Disease, BPH: Benign Prostatic Hyperplasia, COPD: Chronic Obstructive Pulmonary Disease, CABG: Coronary Artery Bypass Grafting, FMF: Familial Mediterranean Fever, Lt: Left, SD: standard deviation, SLE: Systemic Lupus Erythematosus, STEMI: ST-Elevation Myocardial Infarction, T: t-test statistic, U: Mann-Whitney U test statistic, X^2: Chi-square test statistic.

Malnourished patients had lower current weight, more weight loss, reduced intake, frequent GI symptoms, and poorer functional capacity compared to the well-nourished group, while usual weight showed no difference.

Table (2): Distribution of Subjective Global Assessment among the examined groups.

	Group A (Well Nourished) Number =55	**Group B (Malnourished) Number =55**	**Test**	**P value**
Usual Weight (kg) Mean± SD	79.38±9.4	82.68±11.3	t =1.6498	0.101
Current Weight (kg) Mean± SD	79.83±9.8	75±10.1	t =2.5221	**0.013***
Weight Loss (%) Mean± SD Range	1.41±1.8 0.0 – 5.5	8.79±5.3 0.0 – 22.2	U=75.63	< **0.001***
Weight Change Past 2 Weeks				
No change	37 (67.3%)	12 (21.8%)	X^2= 66.555	< **0.001***
Increased	18 (32.7%)	2 (3.6%)		
Decreased	0 (0.0%)	41 (74.5%)		
Dietary Intake in Last 2 Months				
Unchanged	55 (100%)	9 (16.4%)	X^2= 79.063	< **0.001***
Less than normal	0 (0.0%)	2 (3.6%)		
Less than usual	0 (0.0%)	25 (45.5%)		
Little solid food	0 (0.0%)	14 (25.5%)		
Only liquids	0 (0.0%)	5 (9.1%)		
Gastrointestinal Symptoms:				
Nausea	5 (9.1%)	7 (12.7%)	X^2=22.273	**0.022***
Vomiting	1 (1.8%)	10 (18.2%)		
Anorexia	0 (0.0%)	7 (12.7%)		
Constipation	1 (1.8%)	2 (3.6%)		
Dry mouth	1 (1.8%)	5 (9.1%)		
Fatigue	2 (3.6%)	2 (3.6%)		
Polyphagia	2 (3.6%)	0 (0.0%)		
Fully quickly	5 (9.1%)	7 (12.7%)		
Epigastric pain	0 (0.0%)	8 (14.5%)		
Refusal of feeding	0 (0.0%)	4 (7.3%)		
Little solid food	0 (0.0%)	2 (3.6%)		
Smells bother me	0 (0.0%)	4 (7.3%)		
Functional Capacity:				
No dysfunction	44 (80%)	10 (18.2%)	X^2= 52.58	< **0.001***
Not my normal life	9 (16.4%)	20 (36.4%)		
Mild dysfunction	2 (3.6%)	0 (0.0%)		
In bed<1/2 of day	0 (0.0%)	5 (9.1%)		
Bed ridden	0 (0.0%)	20 (36.4%)		

SD: standard deviation, *: Significant, U: Mann-Whitney U test statistic

Table 3 shows that there was a statistically insignificant variance in the presence of increased metabolic demand between the well-nourished group and the malnourished group.

Table (3): Distribution of metabolic demand among the examined groups.

	Group A (Well Nourished) Number =55	**Group B (Malnourished) Number =55**	**Test**	**P value**
Increase metabolic demand				
No	53 (96.4%)	49 (89.1%)	X^2= 2.157	0.141
Epicoprid, mg	2 (3.6%)	6 (10.9%)		

The malnourished group had significantly higher HOMA-IR, protein/creatinine ratio, resistin, AST, and TIBC, but lower iron, albumin, cholesterol, and triglycerides compared to the well-nourished group. Other parameters showed no significant variances.

Table (4): Distribution of laboratory data and biochemical profile between the studied groups.

	Group A (Well Nourished) Number =55	**Group B (Malnourished) Number =55**	**Test**	**P value**
Serum Creatinine, mg/dL Mean± SD	4.26±1.6	3.88±1.3	U=1.421	0.231
eGFR, mL/min/1.73m^2 Median (Range)	13.7 (6.6 – 38.8)	15.8 (6.9 – 42.8)	U=-0.774	0.44
Urine Protein /Creatinine Ratio, mg/mg, Median (Range)	0.8 (0.08 – 4.5)	1.9 (0.1 – 8.2)	U=1084	**0.010***
Sodium (Na), mmol/L Mean± SD	133.64±4.9	134.74±3.5	t=1.3548	0.17
Potassium (K), mmol/L Mean± SD	4.28±0.6	4.31±0.6	t=0.2598	0.79
Calcium (Ca), mg/dL, Mean± SD	8.22±0.6	8.12±0.6	t=0.8660	0.38
Phosphate (PO4), mg/dL Median (Range)	5.2 (3.1 – 8.00)	5 (3.00 – 8.00)	U= 0.752	0 .45
Parathyroid Hormone (iPTH), ng/mL, Median (Range)	122.1 (10.8 –581.12)	90 (19.00 – 460.2)	U= 1.5238	0.12
Blood glucose, mg/dL Mean± SD	150.46±35.9	152.55±6.8	U=2.635	0.952
Insulin, µU/mL, Median (Range)	5.75 (2.3 – 25.00)	7.35 (2.4 – 21.9)	U= 0.9831	0.32
HOMA-IR, Median (Range)	1.35 (0.65 – 4.3)	1.9 (0.95 – 4.3)	U=-4.571	< **0.001***
C-Peptide, ng/ml Median (Range)	3.5 (1.1 – 4)	3.4 (1.2 – 9.5)	U=1459	0.748
Hemoglobin (Hb) g/dL Mean± SD	9.48±1.1	9.49±1.4	t=0.0413	0.96
Iron, ug/gl Median (Range)	70.00 (52.00 – 92.00)	49 (15.00 – 83.00)	U=5.1306	< **0.001***
Ferritin, ng/mL Median (Range)	80.00 (60.00 – 574.00)	71.5 (1.8 – 1214)	U=1.3671	0.17
TIBC, ug/gl, Mean± SD	359.01±31.8	414.74±66.2	t=5.5763	< **0.001***
ALT, IU/L, Median (Range)	20 (5.00 - 165)	19.5 (12.00 - 300)	U=-0.1474	0.88
AST, IU/L, Median (Range)	17 (7.00 – 55.00)	21 (10.00 - 550)	U=-2.6728	**0.008***
Albumin (Alb), g/dL Mean± SD	3.19±0.3	3.04±0.2	t=3.0571	**0.003***
CRP, mg/dL, Median (Range)	24 (12.00 – 100)	24 (12.00 – 72.00)	U=0.2079	0 .83
ESR at 1st hour, mm/hr Median (Range)	25 (13.00 – 75.00)	25 (13.00 – 75.00)	U= 0.2670	0.78
ESR at 2nd hour, mm/hr Median (Range)	35 (15.00 - 100)	35 (15.00 - 100)	U= -0.1847	0.85
Total Cholesterol, mg/dL Mean± SD	225.35±30.02	174.44±27.7	t=9.1588	< **0.001***
Triglycerides, mg/dL Mean± SD	142.33±24.1	113.74±20.5	U=883	< **0.001***
Resistin, ng/mL, Mean± SD	5.37±1.8	31.11±4.8	U=15.36	< **0.001***

eGFR: estimated glomerular filtration rate, HOMA-IR: Homeostasis Model Assessment for Insulin Resistance**,** U: Mann-Whitney U test statistic.

According to logistic regression, DBP, albumin (Alb), total cholesterol, triglycerides, and resistin were statistically significant factor influencing malnutrition in diabetic CKD patients.

Table (5): Logistic regression analysis to recognize risk factors related to malnutrition in diabetic CKD patients.

Variables	B	S.E.	Wald	Sig.	Exp(B)	95% C.I. for EXP(B)	
						Lower	Upper
SBP	-0.025	0.014	3.27	0.071	0.975	0.949	1.002
DBP	-0.054	0.024	4.842	0.028	0.947	0.904	0.993
iPTH	0.000	0.001	0.288	0.592	1.000	0.998	1.002
Ca	0.337	0.254	1.761	0.184	1.401	0.851	2.304
HOMA-IR	0.389	0.221	3.095	0.079	1.476	0.957	2.275
C Peptide	-0.002	0.107	0.001	0.982	0.998	0.809	1.231
Iron	0.028	0.149	0.35	0.852	1.028	0.768	1.377
Hemoglobin	0.029	0.011	6.903	0.009	1.029	1.007	1.052
Ferritin	-0.003	0.001	3.075	0.080	0.997	0.995	0.999
TIBC	-0.007	0.003	3.508	0.061	0.993	0.987	0.999
Albumin (Alb)	1.484	0.621	5.704	0.017	4.411	1.306	14.897
Total Cholesterol	0.056	0.010	29.681	<0.001	1.058	1.037	1.079
Triglycerides	0.041	0.009	19.893	<0.001	1.042	1.024	1.060
GP - GSA	-28.403	1212.97	0.001	0.981	0	0	0
Resistin	**0.371**	**0.141**	**6.945**	**0.008**	1.449	1.099	1.910

SBP: systolic blood pressure, DBP: diastolic blood pressure.

DISCUSSION

Comparison between the studied groups illustrated that the malnourished group was significantly older (p=0.03), had a significantly shorter CKD duration (p=0.016), and showed a significantly elevated occurrence of hypertension (p=0.04) and autosomal dominant polycystic kidney disease (p=0.02) compared to the well-nourished group. Other demographic, medical, and family history characteristics showed a statistically insignificant variances amid the two groups (p > 0.05).

These outcomes are in line with the results of **Shengrui *et al.* [17],** who performed a recent systematic review and identified older age, longer disease duration, and the presence of comorbidities as significant risk factors for malnutrition in CKD patients.

In contrast, **Elsayed and ElKazaz [18]** reported differing observations among end-stage renal disease (ESRD) cases having hemodialysis. Their findings indicated that malnourished patients had a significantly longer duration on dialysis (p = 0.004); however, no significant associations were found between malnutrition and variables like age, sex, or comorbidities in their cohort. The discrepancies may be due to differences in disease stage as their study investigated patients exclusively with ESRD on maintenance hemodialysis, while this study focused on diabetic CKD patients across varying stages.

In the current research, there was a statistically insignificant variance in the occurrence of increased metabolic demand between malnourished and well-nourished diabetic CKD patients. While a slightly higher proportion of malnourished individuals exhibited signs of increased metabolic demand, this variance didn't reach statistical significance (p-value equal 0.141). These outcomes suggest that elevated metabolic demand may not be a primary distinguishing factor of malnutrition in this patient population and highlight the need to consider a broader range of nutritional and clinical indicators during assessment. However, larger, multicenter research is warranted to validate these preliminary findings and to explore potential contributing factors with greater statistical power.

These observations are supported by previous studies. **Elsayed and ElKazaz [18]** reported a significant negative

correlation between malnutrition and mid-arm circumference ($p = 0.003$), reinforcing the association between nutritional status and muscle mass. Similarly, **Koor *et al.*** [19] in a study of 66 dialysis patients, demonstrated that mid-arm circumference is a reliable indicator of malnutrition. Also, in concordance with the present research, **Sheikh *et al.*** [20] found that mid-arm muscle circumference (MAMC) was significantly lower in malnourished CKD patients compared to well-nourished individuals ($p < 0.001$). Additionally, triceps skinfold thickness (TSFT), an indicator of subcutaneous fat, was significantly reduced in malnourished cases versus well-nourished patients (p-value under 0.001). Notably, TSFT was ≤10 mm in 91.1% of malnourished cases, compared to only 16.3% of well-nourished individuals, underscoring the elevated sensitivity of TSFT in detecting malnutrition.

The significant elevation in HOMA-IR level observed in the malnourished group likely reflects underlying insulin resistance and altered pancreatic function, as common metabolic complications in advanced CKD. Elevated resistin levels among malnourished diabetic CKD patients further support its role as a pro-inflammatory adipokine closely linked to insulin resistance and the malnutrition-inflammation-atherosclerosis (MIA) syndrome. These outcomes agree with those of **Marques *et al.*** [21] who stated a significant association between elevated resistin and HOMA-IR in malnourished diabetic CKD patients.

According to logistic regression, DBP, albumin (Alb), total cholesterol, triglycerides, and resistin were statistically significant factors influencing malnutrition in diabetic CKD patients ($P < 0.05$). Elevated diastolic blood pressure may contribute to hemodynamic stress and vascular alterations that exacerbate malnutrition. Hypoalbuminemia is a well-established marker of poor nutritional status and chronic inflammation. Dyslipidemia in CKD can reflect both metabolic disturbances and inflammatory states. Also, resistin has role as a metabolic and inflammatory mediator links it to both nutritional status and cardiovascular complications. [22]

One of the key findings of our analysis was the significant association between diastolic blood pressures and malnutrition. This outcome is consistent with the work of **Marques *et al.*** [21] who identified malnutrition as being associated with pulse pressure, a marker of cardiovascular burden. Hemodynamic stress, which results from prolonged elevated blood pressure, along with fluid overload and subclinical inflammation, likely exacerbates the malnutrition observed in CKD patients.

CONCLUSION

In diabetic CKD patients, malnutrition was associated with older age, poorer dietary intake, reduced functional capacity, and significant biochemical changes. Malnourished patients showed lower albumin, iron, cholesterol, and triglycerides, alongside higher HOMA-IR, TIBC, and notably **elevated resistin** levels. Logistic regression identified DBP, albumin, total cholesterol, triglycerides, and resistin as independent predictors of malnutrition. Among them, resistin appeared to be a strong marker, reflecting its link to inflammation and metabolic dysregulation in CKD. These findings highlight the importance of early nutritional assessment and suggest that resistin may serve as a useful biomarker for detecting malnutrition in diabetic CKD patients.

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