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ORIGINAL PAPER

Serum leptin and glucagon-like peptide-1 levels in diabetic patients with end-stage renal disease on hemodialysis – a cross-sectional study in Iraqi patients

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ABSTRACT

Introduction and aim. Diabetic kidney disease (DKD), a common complication of type 2 diabetes mellitus, is the leading cause of end-stage renal disease (ESRD). This study aimed to evaluate differences in serum leptin and glucagon-like peptide-1 (GLP-1) levels between diabetic ESRD patients undergoing hemodialysis and healthy controls.

Material and methods. A cross-sectional observational study was conducted involving 65 participants: 31 type 2 diabetic patients with ESRD on hemodialysis and 34 healthy controls. Serum leptin and GLP-1 concentrations were measured using enzyme-linked immunosorbent assays.

Results. Patients with ESRD exhibited significantly higher serum leptin levels (1.7±1.0 ng/mL) compared to controls (1.4±0.7 ng/mL; p=0.001), and significantly lower GLP-1 levels (19.6±11.2 pmol/L vs. 37.0±25.7 pmol/L; p=0.001).

Conclusion. Elevated leptin levels and reduced GLP-1 concentrations in diabetic ESRD patients suggest a potential role of these biomarkers in renal injury and metabolic regulation. The findings highlight the therapeutic promise of GLP-1 receptor agonists in this population.

Keywords. Chronic kidney disease, diabetic kidney disease, end-stage renal failure, glucagon-like peptide 1, hemodialysis, leptin

Introduction

The global prevalence of diabetes mellitus (DM) has dramatically increased, affecting approximately 11% of the world's population by 2021. A significant complication of diabetes, particularly type 2 diabetes mellitus (T2DM), is diabetic kidney disease (DKD), also known as diabetic nephropathy (DN), which develops in 20–50% of T2DM patients.[1] DKD is a leading cause of chronic kidney disease (CKD) and end-stage renal disease (ESRD) worldwide, significantly contributing to morbidity and mortality. It progressively impairs kidney function, beginning with glomerular hyperfiltration and advancing through stages marked by microalbuminuria and declining glomerular filtration rate (GFR),[2] ultimately leading to ESRD. CKD, defined as kidney damage or reduced function for over three months with health consequences, is cause, GFR (stages G1–G5), and albuminuria (A1–A3) levels (CGA classification).[3]

Leptin is an endocrine hormone secreted primarily by adipose tissue. It is involved in multiple metabolic processes, including regulation of body weight, food intake, and energy balance, as well as inflammatory and hemostatic pathways that contribute to cardiovascular and hypertensive disorders.[4] The relationship between leptin and renal disorders has been the subject of numerous investigations. Elevated leptin levels have been

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associated with hyperglycemia and increased blood pressure, the latter likely mediated by enhanced sympathetic nervous system activity, as well as renal impairment. Patients with CKD, particularly those with DKD, often exhibit higher serum leptin levels due to decreased renal clearance and increased adipose tissue synthesis.[5] Leptin promotes renal inflammation and fibrosis through pathways including transforming growth factor-beta (TGF-β) and other signaling methods, leading to glomerulosclerosis and proteinuria, hallmark features of DKD. Consequently, leptin is considered a potential marker for renal injury.[6] Elevated leptin levels have also been associated with endothelial dysfunction, which is characterized by increased collagen synthesis and mesangial cell hypertrophy, both of which can exacerbate kidney injury.[6,7]

The glucagon-like peptide-1 (GLP-1) is an essential hormone for controlling hunger and glucose metabolism. Because of its various physiological effects, GLP-1 has drawn interest for its medicinal potential in treating obesity, T2DM, and other disorders. The primary function of GLP-1 is the incretin effect, which helps maintain glucose homeostasis by increasing insulin release in response to meals.[8] Additionally, it decreases food intake, delays stomach emptying, and affects gut motility, all of which help with weight management and diabetes treatment. Beyond its metabolic roles, GLP-1 exhibits neuroprotective and cardioprotective effects, reduces inflammation, and influences cognitive functions such as memory and learning.[9] There is growing recognition of the link between GLP-1 hormone levels and CKD, especially concerning the protective role of GLP-1 receptor agonists (GLP-1 RAs) in CKD patients. It has been demonstrated that GLP-1 RAs reduce the incidence of major adverse kidney events in patients with CKD and type 2 diabetes by 18%.[10] To our knowledge, this study is among the first to assess the correlation between serum leptin and GLP-1 levels in diabetic patients with ESRD undergoing hemodialysis within the Iraqi population. Although prior research has examined these biomarkers across diverse populations, there is a shortage of data specifically from Iraq and analogous underrepresented areas. Our study offers region-specific insights into the hormonal profiles of this patient cohort, which may vary due to genetic, environmental, and lifestyle factors distinctive to this population. These results enhance the comprehension of the metabolic and inflammatory pathways associated with DKD in Iraqi patients and underscore the potential for formulating more individualized treatment approaches, including the application of GLP-1 receptor agonists, specifically designed to meet the requirements of this population. Ultimately, this research addresses a deficiency in the literature and advocates for the incorporation of diverse populations in forthcoming biomarker and therapeutic studies.

Aim

This study examines the potential differences in leptin and GLP-1 hormone concentration and their sensitivity and specificity for DKD patients on hemodialysis in Iraqi patients. This study offers a novel addition among the few undertaken in Al-Basrah, Iraq, offering population-specific insights into biomarker behavior in an underrepresented community.

Material and methods

This study was designed as an observational, cross-sectional analysis aimed at comparing leptin and GLP-1 levels in diabetic ESRD patients and healthy controls. This study was conducted in Basrah Teaching Hospital from January 2024 to April 2024. Demographic and laboratory data were collected. A total of 65 patients were included, of whom (n=31) end-stage renal disease patients with diabetes mellitus on hemodialysis were classified and defined according to the National Kidney Foundation KDOQI definition for chronic kidney disease.[11] Patients with an estimated glomerular filtration rate (eGFR) <15 mL/min/1.73 m² and were receiving maintenance hemodialysis. eGFR was computed by using the Modification of Diet in Renal Disease (MDRD) equation:[12]

$$\text{eGFR} = 186 \times [\text{serum Cr}] - 1.154 \times \times [\text{age}] - 0.203 \times [0.742 \text{ if women}]$$

Which is known to be more accurate. Patients undergoing chronic hemodialysis were eligible to participate in the study if they agreed to do so, and control participants were healthy volunteers without diabetes or kidney disease (n=34), with median ages of 57.5±7.6 and 41±10.6 years, respectively. The control group was younger on average (41±10.6 years) compared to the ESRD group (57.5±7.6 years). This age difference was statistically significant ($p<0.0001$). The lack of age matching represents a potential confounding factor and is acknowledged as a limitation of this study. Written consent was obtained from all participants. Serum samples from the participants were collected and stored at –80°C until analysis. Each group has made the following biomarkers: blood pressure, random blood sugar (RBS), glycated hemoglobin A1c (HbA1c), urea, creatinine, and eGFR. Leptin and GLP-1 levels were detected by using enzyme-linked immunosorbent assay kits (BT LAB, China), which have a sensitivity of 0.021 ng/mL and an inter-assay coefficient of variation of <10%, and a sensitivity of 1.27 pmol/L with an inter-assay coefficient of variation of <10%, respectively. The study protocols received approval from the ethics committees of the Basrah Teaching Hospital and the Basrah Dialysis Center.

Ethics approval

The study received approval from the Ethics Committee of the College of Pharmacy at the University of Basrah. (Protocol No. 248, dated December 15, 2023) and the Basrah Health Directorate (Protocol No. 74, dated January 27, 2024). All procedures were performed in compliance with the Declaration of Helsinki and any institutional rules. Informed written consent was acquired from all subjects before enrollment. Participants were notified of the study's objective and their entitlement to withdraw at any moment without repercussions, and the confidentiality of their personal data.

Inclusion criteria

Patients aged 30 to 70 of both genders suffering from renal failure, diabetic mellitus, and hypertension, were involved in the study.

Exclusion criteria

Pregnant women, breastfeeding, smokers, thyroid dysfunction, patients with a history of chronic liver disease, hepatitis type B and C, malignancy, rheumatoid arthritis, patients on dipeptidyl peptidase 4 (DPP-4) inhibitors agents, GLP-1 agonists agents, immunosuppressant agent, disease-modifying antirheumatic drug, kidney transplantation, and stroke were excluded from participating in this study.

Statistical analysis

The data were defined as means and medians. A t-test analysis (unpaired test) was conducted to compare continuous variables. Normality of data distribution was calculated using the Shapiro–Wilk test. Group comparisons were performed using a two-tailed unpaired t-test for normally distributed variables. A p-value <0.05 was considered statistically significant. Regression analysis was not performed due to sample size limitations, which is a limitation of this study. Also, no formal power calculation was performed due to the exploratory nature of this study.

Results

The ESRD group was significantly older than the control group (57.5±7.6 years vs. 41.0±10.6 years; $p < 0.001$). This age difference was not controlled for in the analysis and is acknowledged as a potential confounding factor. urea in the disease group shows significantly higher levels as compared to the control group (123.9±73.6 mg/dL vs 20.6±6.7 mg/dL, p<0.0001). Creatinine levels are significantly higher in the disease group compared to the control group (7±2.6 mg/dL vs 0.8±0.2 mg/dL, p<0.0001). The eGFR value in the disease group is significantly lower than in the control group (8.9±4 mL/min/1.73 m^2 vs 106.1±22.7 mL/min/1.73 m^2, p<0.0001). the HbA1c (%), the disease group shows significantly higher HbA1c% levels as compared to the control group (8.2±1 vs 5.3±0.6%, p<0.0001). The systolic blood pressure (SBP) levels in the disease group show significantly higher systolic values as compared to the control group (147.7±15.8 mmHg vs 121.1±3 mmHg, p<0.0001). The diastolic blood pressure (DBP) levels in the disease group are significantly higher than those in the control group (86.9±10.7 mmHg vs 80.7±1.9 mmHg, p<0.031). Leptin levels in the disease group show significantly higher leptin values as compared to the control group. (1.7±1ng/mL vs 1.4±0.7 ng/mL, p<0.001). Concerning GLP-1 levels, the disease group 1 shows significantly lower systolic value as compared to the control group (19.6±11.2 pmol/L vs 37±25.7 pmol/L, p<0.001, Table 1).

Table 1. Laboratory data (expressed as mean±SD)

Parameters	Disease group (n=31)	Control group (n=34)	p-value	Mean difference (95% CI)	Cohen's d
Urea (mg/dL)	123.9±73.6	20.6±6.7	<0.0001	+103.3 (77.3 to 129.3)	2.03
Creatinine (mg/dL)	7±2.6	0.8±0.2	<0.0001	+6.2 (5.28 to 7.12)	3.44
eGFR (mL/min/1.73 m^2)	8.9±4	106.1±22.7	<0.0001	-97.2 (-105.0 to -89.4)	-5.84
HbA1c (%)	8.2±1	5.3±0.6	<0.0001	+2.9 (2.49 to 3.31)	3.56
RBS (mg/dL)	205.5±91	106.7±11.5	<0.0001	+98.8 (66.5 to 131.1)	1.56
Systolic (mmHg)	147.7±15.8	121.1±3	<0.0001	+26.6 (20.95 to 32.25)	2.39
Diastolic (mmHg)	86.9±10.7	80.7±1.9	0.031	+6.2 (2.38 to 10.02)	0.83
Leptin (ng/mL)	1.7±1	1.4±0.7	0.001	+0.3 (-0.12 to 0.72)	0.35
GLP-1 (pmol/L)	19.6±11.2	37±25.7	0.001	-17.4 (-26.9 to -7.9)	-0.86

Leptin was significantly correlated with serum creatinine and p=0.005, eGFR with p=0.015, GLP-1 with p=0.001, SBP with p=0.002, and DBP with p=0.025. The other data, age, HbA1c, RBS, and urea, were not significantly correlated with leptin, as shown in Table 2.

Table 2. Correlation table between serum leptin and metabolic, renal, and hemodynamic parameters in patients

Parameters	Leptin	
	r	p
Age	0.156	0.078
HbA1c%	0.057	0.519
RBS (mg/dL)	-0.050	0.578
Urea (mg/dL)	0.093	0.293
Creatinine (mg/dL)	0.246	0.005
eGFR (mL/min/1.73 m^2)	-0.213	0.015
GLP-1 (pmol/L)	0.295	0.001
Systolic blood pressure (mmHg)	0.275	0.002
Diastolic blood pressure (mmHg)	0.197	0.025

Discussion

The principal functions of the kidneys are the elimination of waste products and the regulation of water balance, and the management of blood pressure. ESRD is a severe condition necessitating prompt intervention, including dialysis or kidney transplantation.[13]

A decline in various filtration markers, such as eGFR, is consistently associated with increased ESRD

risk. As in the current study, an elevation in biomarkers related to renal functions, including serum urea, serum creatinine, and a decrease in eGFR, as seen in Table 1, in the disease group, leads to ESRD. These findings are consistent with Rebholz et al.[14] Declines in eGFR were consistently associated with progression to ESRD.

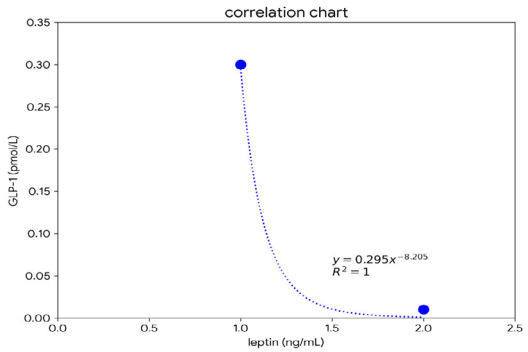

Fig. 1. Correlation chart between leptin and GLP-1

Hypertension is a significant risk factor for ESRD, with various studies highlighting its impact on kidney health. In this current study, elevation in blood pressure in the disease group contributes to renal deterioration, which agrees with the current findings. That contrasts with Kim et al., who found that higher hypertension exposure (systolic BP≥140 mmHg) was linked to increased ESRD risk.[15] Studies conducted at several sites reveal a clear relationship between hypertension and elevated leptin levels.[16,17]

Factors including hyperglycemia, inflammation, and fibrosis affect the declining renal function in DKD and the progression to ESRD.[18]

In this study, patients in the disease group with high levels of random blood sugar and HbA1c have ESRD and are on hemodialysis. This agrees with the results of the investigations of Oluboyo and Azeez et al.[19,20] However, these studies included a larger number of patients and included other renal markers, such as uric acid and cystatin C. Chronic hyperglycemia leads the kidneys to undergo structural and functional alterations. In contrast, CKD has the potential to impair glucose metabolism, which may lead to the development of diabetes and insulin resistance.

Higher leptin levels in the disease group are associated with an increased cardiovascular risk profile, consistent with previous findings in ESRD populations. Hence, the kidneys can undergo pathogenic alterations. These alterations can cause more protein to seep into the filtration. Leptin's direct profibrotic actions on glomeruli suggest a possible interaction between leptin and glomerular endothelial/mesangial cells; furthermore, the kidney's augment expression of type IV collagen results from increased leptin levels.[5]

On the other hand, low GLP-1 levels in the disease group are noteworthy because they correspond with the progression of renal impairment. This finding is consistent with the study by Diwesh Chawla et al. 2023.[21] Which also reports a low level of GLP-1. GLP-1, which is predominantly produced in the gastrointestinal tract, plays a protective role in kidney function, primarily through its receptor (GLP-1R).

A low level of GLP-1 is owing to its fast breakdown by DPP-4. The study, Lebherz et al.[22] disagreed with our research, which states that GLP-1 levels are significantly elevated in critically ill patients and those with end-stage renal disease compared to healthy participants; this increase can be attributed to both enhanced GLP-1 secretion and reduced clearance, particularly in inflammatory conditions and chronic kidney disease. In our study, the level of GLP-1 in the control group was higher than in the disease group, but in the study of Cao et al.[23] the comparison of (GLP-1) hormone levels between healthy controls and disease patients revealed substantial differences. Healthy people often have greater GLP-1 levels, which are essential for glucose metabolism and kidney protection. Meal intake boosts their strong GLP-1 response, improving insulin secretion and glucose homeostasis. In contrast, the disease group frequently has low GLP-1 levels, contributing to metabolic and renal problems. This is consistent with the results of this current study. Healthy people often have greater GLP-1 levels, which is compatible with this study's results.

Leptin has significant correlations with GLP-1 (r=0.295, p=0.001), as in Table 2, moderately and significantly supports a metabolic-hormonal interplay between the adipose and incretin systems. Leptin and GLP-1 both play roles in energy balance, appetite, and insulin dynamics.

Research suggests that leptin stimulates GLP-1 secretion and regulates its impact on insulin secretion and appetite control. A study by Tomasik et al. 2020 indicates that leptin may serve as an advantageous biomarker for predicting GLP-1 activity.[24]

It should be noted that the disease group was older on average than the control group. Age is known to influence several metabolic and inflammatory biomarkers, including leptin and GLP-1, and may partially explain the observed differences. While our statistical analysis aimed to account for these differences, residual confounding cannot be excluded.

Study limitations

This study has several limitations. The sample size was quite limited, which may have restricted the generalizability of the findings to larger groups. Second, the control and disease groups were not age-matched, potentially introducing confounding effects. Third, the cross-sectional design prevents any inference of caus-

al relationships between the measured biomarkers and disease status. Finally, the absence of multivariate analyses restricts our ability to adjust for potential covariates, such as comorbidities or medication use, that may have influenced the results.

Conclusion

This cross-sectional study demonstrated that patients with diabetic ESRD undergoing hemodialysis had significantly higher serum leptin levels and lower GLP-1 concentrations compared to healthy controls. These findings support previous evidence suggesting that leptin may contribute to kidney injury and comorbidities, while GLP-1 may offer protective renal effects. Despite the limited sample size and absence of age-matching between groups, the results highlight the potential relevance of these biomarkers in the pathophysiology of DKD. Further longitudinal and interventional studies are needed to evaluate the prognostic and therapeutic implications of leptin and GLP-1, particularly in the context of GLP-1 receptor agonist therapy in CKD patients.

Declarations

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Author contributions

Conceptualization, H.H.F., R.D.S. and J.K.H.; Methodology, J.K.H.; Software, R.D.S.; Validation, H.H.F., R.D.S. and J.K.H.; Formal Analysis, R.D.S.; Investigation, J.K.H.; Resources, R.D.S.; Data Curation, H.H.F.; Writing – Original Draft Preparation, H.H.F.; Writing – Review & Editing, J.K.H.; Visualization, H.H.F.; Supervision, R.D.S.; Project Administration, J.K.H.; Funding Acquisition, H.H.F.

Conflicts of interest

All authors declare that there are no conflicts of interest in this article.

Data availability

All data are available from the corresponding author and will be made available upon request.

Ethics approval

The study received approval from the Ethics Committee of the College of Pharmacy at the University of Basrah (Protocol No. 248, dated December 15, 2023) and the Basrah Health Directorate (Protocol No. 74, dated January 27, 2024).

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