Determination of lept in and adiponectin level in patients with End-stage renal

Sarah F.Hussein ,Alaa Hussein J. Al-qaisi,TS Morad

Department of Chemistry, College of Science, Al-Nahrain University, Baghdad,Iraq.

Abstract:

Chronic Renal Failure (CRF) is a term used to describe an irreversible decline in renal function that often occurs over a number of years. In many situations, an initial insult to the kidney function leads to additional nephron loss and the requirement for dialysis or kidney transplantation End stage renal disease(ESRD) is the term used to describe this ailment.
A number of metabolic, vascular, and inflammatory illnesses are caused by adipokines. In order to determine whether certain associated biomarkers can aid in the diagnosis of CRF patients, this study compares the levels of inflammatory adipocytokines (leptin and adiponectine), as well as their ratios, in ESRD patients and healthy controls.
Methods: In all, 70 ESRD patients30 men and 40 femaleswith ages ranging from (30 to 55) years old and 30 controls 13 males and 17 females with ages ranging from (25 to 60) years old were included in the study. All participants' biochemical characteristics, demographic information, and serum leptin and adiponectin concentrations were examined.
Results: According to our research, people with chronic renal failure had significantly greater serum levels of leptin and adiponectin than did healthy controls.leptin and adiponectin had positive correlation with urea and creatinine.
Conclusion: We found differences in adipokine levels that might be used to detect chronic renal failure

Aims: Analyze of serum levels of Leptin and Adiponectin inend-stage renal disease (ESRD)and Healthy individuals.

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Due to impaired renal function, there is a buildup of toxins and extra water in CRF. The preferred method for treating ESRD and removing built-up toxins from the body is dialysis. Patients receiving dialysis have a cardiovascular risk that is 10–20 times greater than that of healthy individuals. Dialysis and inflammatory kidneys both impact endothelial function,

Introduction: CRF, or chronic renal failure, is the most common public health issue affecting the aged population globally. Having a damaged kidney is the main cause of CRF. According to the glomerular filtration rate (GFR), there are five stages of CRF, and stage 5 (GFR 15 ml/min/1.73m2) is sometimes referred to as an end-stage renal disease (ESRD)[1].

from adipose tissue, circulating in the plasma with trimer form. The most readily available adipose tissue protein, adiponectin, is also thought to have anti-inflammatory and anti-atherosclerotic properties. Nuclear factor kappa B (NF-B) signaling pathway is used to inhibit proinflammatory molecules and has an anti-inflammatory effect on vascular walls. . that kidney function decline associated with elevated adiponectin is due to decreased renal clearance and compensatory responses to adiponectin resistance. It can also inhibit the subsequent proinflammatory effects by suppressing tumor necrosis factor (TNF)- and interleukin (IL)-6 and promoting anti-inflammatory cytokines such as IL-10. Adiponectin receptors, which come in two isoforms, mediate the effects of adiponectin (AdipoR1 and AdipoR2) [6].

MATERIALS AND METHODS

The current study was conducted in AL-Yarmouk**Study participants:** teaching hospital, during the period from October 2021 to March 2022. The present study included 70 (30 males and 40 females) ESRD patients from 30 to 60 years old and 30 (13 males and 17 females) healthy control group ranging from 25-60 years old. This study was approved by the Department of Chemistry, College of Science, Al-Nahrain University.

Exclusion criteria: History of hypertension, diabetes mellitus, hypothyroidism, and hepatic or renal disorders, smoking, and drug used were all eliminated from the current study.

Sample collection: Patients and healthy individuals provided seven milliliters of venous blood placed into gel tubes for 15 minutes to coagulate. Serum was isolated from blood samples by centrifugation at 1840 x g for 15 minutes at room temperature. The serum was separated into aliquots and kept at -70°C until testing**.**

increasing the risk of hypertension and heart issues. Therefore, the risks associated with receiving dialysis should be understood by both doctors and patients. Patients with CRF need to be informed as soon as possible about the disease, treatments, dietary requirements, and other factors necessary for managing the condition and leading a normal life[2].

Adipose tissue not only serves as a passive energy storage reservoir, but it also serves as an endocrine gland, producing and secreting a wide range of bioactive peptides known as adipokines[3]. A variety of metabolic, vascular, and inflammatory illnesses have different etiologies, which are all influenced by adipokines[4]. Adipokines include the hormones adiponectin, resistin, leptin, visfatin, plasminogen activator inhibitor type 1 (PAI-1), tumor necrosis factor alpha (TNF-), interleukin (IL)-6, and IL-8[5].

One of the future proteins in inflammatory biomarkers might be leptin.single-chain Obese (ob) gene encoded 16 kDa protein is mostly released by adipocytes, while it can also be made by cardiomyocytes and vascular smooth muscle cells. Controlling the stimuli that cause appetite, regulating food intake, and regulating energy expenditure are its three main functions[7]. Leptin is predominantly eliminated via a mix of glomerular filtration and tubular breakdown and is also a proinflammatory adipocytokine. Its levels rise in tandem with those of glucocorticoids, insulin, other cytokines, and fat. It has been demonstrated that weight reduction is followed by a drop in leptin serum levels in both healthy individuals and patients with diabetes mellitus. These findings imply that metabolic syndrome includes elevated leptin concentrations and that these conditions may contribute to an increased risk of cardiovascular disease[8]

Another pro-inflammatory adipokine that has the potential to help with CRF diagnosis is Adiponectin, 30-kDa protein

Table (1) shows the demographic data of the two studied groups end-stage renal disease (ESRD) and control. The results obtained from the preliminary analysis shown in table (1) indicated that there was no significant difference in BMI (P = 0.0993) between ESRD and control groups. There were no significant differences between ESRD group and the control group regarding age (P=0.5563).

Table (2) shows laboratory data from the blood analysis among the two groups. Our findings indicate there were significant differences between the ESRD or control groups regarding serum levels of urea and creatinine and there were significantly higher in patient group (P< 0.0001) compared to controls.

Table (3) shows the different adipokine concentrations among the two groups. Statistical analysis revealed that serum levels of leptin were significantly higher (p< 0.0001) among two groups, as shown in figure (1). Furthermore, serum levels of adiponecctin were significantly (P< 0.0066) in ESRD group compared to controls, as shown in figure (2).

Measurement of Body Mass Index:BMI was measured by dividing weight (in Kilograms, Kg) by height squared (in meter, m) for each participant**.**

Biochemical analysis: serum levels of leptin, and adiponectin were measured using enzyme-linked immune-sorbent assay (ELISA) provided by (SunLong ,china). The photometric method was used to evaluate the serumurea and creatinine provided by (Linear, Spain).

Statistical Analysis of Parameters: Demographic and biochemical data in the present study were performed using GraphPad Prism software version 8.0.2 (San Diego, California, USA). T-test unpaired was performed to assess mean ± standard deviation (STD) and significant differences (P-value) among means of the two studied groups. Correlations between parameters in the present study were estimated with Pearson's correlation coefficient. P ≤ 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic datafor the ESRD and control groups

Variables	Control	**Patients**	P -Value	Significant
Age(year)	53.39±8.2	54.53±6.9	0.5563	NS
Gender (Male\Female)	13/17	30/40		
BMI	29.6 ± 2.4	30.2 ± 3.4	0.0993	NS

Table 2: Clinical and laboratory parameters between the ESRD and control groups (Mean±SD)

Variables	Control	**Patients**	P -Value	Significant
Urea(mg/dl)	27.21 ±4.100	136.3 ± 37.39	<0.0001	HS
Creatinine(mg/dl)	0.871 ± 0.135	6.400 ± 1.787	<0.0001	HS

Table 3: Comparison of adipokine concentrations among the ESRD and control groups

Variables	Control	**Patients**	P -Value	Significant
Leptin(pg/ml)	128.0 ± 18.11	257.5 ± 37.43	<0.0001	HS
Adiponectin(ng/ml)	± 0.28401.139	2.837 ± 1.853	<0.0066	S

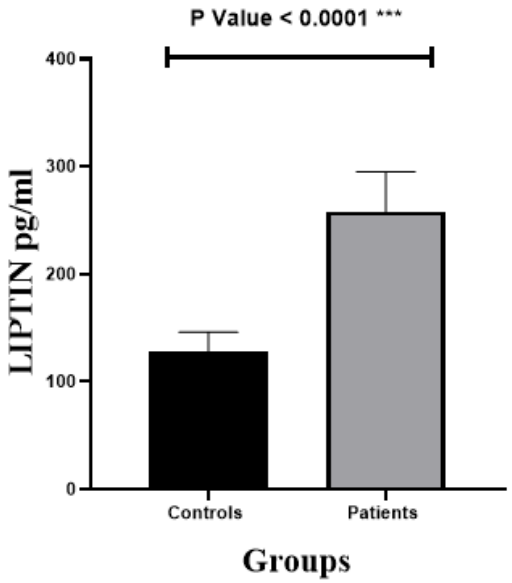

Figure (1): Leptin Levels in Patients and Control Group

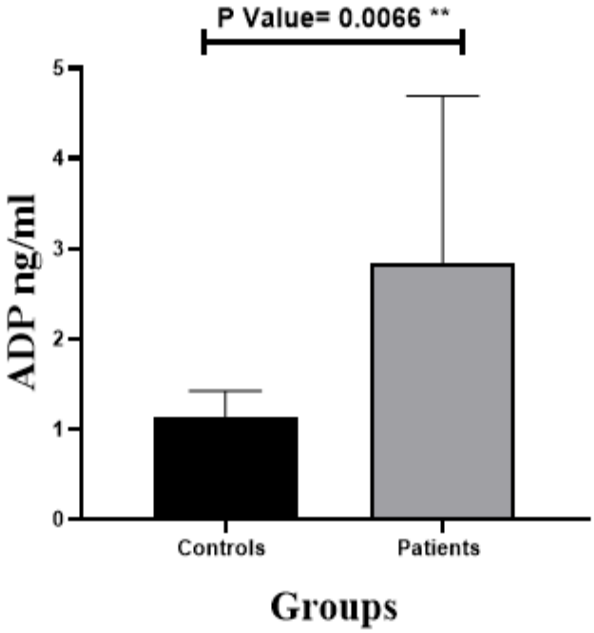

Figure (2): Adiponectin Levels in Patients and Control Group

DISCUSSION

our research, we looked at the serum levels of leptin and adiponectine in

End-stage renal disease (ESRD) has been linked in several studies to adipokines.In

concentration of urea, mainly due to decreased secretion of the kidneys, which is similar to the findings of the previous studies(E. R. Sarhat, and A. A. Salih) [17].

Our study found that increased adiponectin was linked to a higher risk of ESRD, regardless of other ESRD-related factors like BMI and elements of the metabolic syndrome[17]. Numerous phases of CKD and maintenance dialysis have been linked to increased levels of circulating adiponectin. Despite concurrently increasing circulating adiponectin levels, the expression of adiponectin mRNA and AdipoR1 is upregulated in visceral and subcutaneous fat tissue in ESRD patients[18]. Additionally, it has been discovered that adiponectin receptor signaling is postreceptor blocked[19].All of these findings suggest that as kidney function declines, the paradoxical rise in adiponectin may not only be a reflection of decreased renal clearance but also be related to the resistance effect of adiponectin. Third, the elevation in adiponectin might be a defense mechanism against atherosclerosis and chronic inflammation, two factors that define CKD state. Additionally, the presence of insulin resistance, especially in the earliest stages of chronic kidney disease, may cause an adaptive response to the changed metabolic profiles in the form of increased adiponectin[20].

Adiponectin correlated significantly with urea (r = 0.3395, P<0.0495) and tended to correlate with creatinine (r = 0.4045, P = 0.0177) in kidney allograft recipients.[16]

CONCLUSION

Our findings reveal that leptin and adiponectin levels are significantly higher. Therefore, these alterations in adipokine levels may play a key role in the development of end-stage renal disease (ESRD).

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patients with CRF to see what role adipokines have in the disease's etiology.

serum leptin level is highly significant (p< 0.0001) increase in ESRD patients as compared to control group, which is similar to the findings of the previous studies(N. P. Singh)[9], (M. Nearmeen)[10] but different with(M. Schambelan)[11].

The disparity may result from variations in the study's patient population. The observed hyperleptinemia may be caused by HD patients' reduced renal clearance, which leads to leptin retention[10]

Leptin is an adipokine that adipocytes release and regulates inflammation and lipid metabolism. In this cross-sectional investigation, patients receiving hemodialysis (HD) were tested for serum leptin levels[12].

Because renal function declines, the kidneys play a crucial role in eliminating circulating leptin. Serum leptin levels rise with the advancement of CKD, even after adjusting for age and body fat mass[13]. Leptin is frequently high in patients with renal failure.

Patients with renal failure have higher serum leptin levels than healthy people. There are few studies on the correlations between serum leptin and renal function and inflammation in patients with impaired glomerular filtration rate (GFR)[14].

The kidneys' excretory, endocrine, homeostatic, and metabolic functions are all compromised in acute renal failure (ARF). Leptin is a polypeptide hormone generated from adipose tissue that is primarily biodegraded by the kidneys. As a result, chronic renal failure (CRF) has an elevated plasma leptin concentration[15].

Leptin was related positively to serum urea (r = 0.302, P<0.05),and correlated significantly with creatinine (r = 0.4647, P = 0.0056).[16]

Serum level Adiponectin is highly significant (p< 0.01) increase in CRF patients as compared to control group.

Adipokines are significantly increased in the plasma of patients with high

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