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RESEARCH PAPER

Association of Adiponectin With biochemical parameters in patients with chronic kidney diseases on Hemodialysis process in Erbil city

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A B S T R A C T:

Adiponectin (Adipo.) is a hormone produced by adipocytes that circulate in the blood. Serum Adipo. concentrations are elevated in several chronic kidney disease (CKD) conditions and are a predictor of end-stage renal disease. The association between Adipo. levels and the progression of the disease are not well established. This study aimed to determine if there was a relationship between serum Adipo. levels and inflammatory, metabolic, and CKD progression in Erbil. In a case-control study of 200 participants, 100 with CKDs were on hemodialysis (HD) in Erbil teaching hospital. To collect the necessary information from participants, a study questionnaire was used. Adipo. hormone levels and other metabolic parameters were measured. Total Cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), very-low-density lipoprotein (VLDL), fasting blood sugar, C-reactive protein (CRP), glycosylated hemoglobin (HbA1c), creatinine, albumin, and urea were all measured in the blood. 90.0% of the cases were at stage 5 of CKD, the Serum Adipo. levels were higher when compared with a healthy control group (13.26 ±0.96 µg/ml vs 2.17±0.75 µg/ml). In conclusion, our study found A high level of Adipo. in CKD patients can predict inflammation and malnutrition and is considered a biomarker for renal dysfunction.

KEY WORDS: Adiponectin, chronic kidney disease, diabetes mellitus, Glomerular Filtration Rate, C-reactive protein, and Haemodialysis..
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1. INTRODUCTION:

Chronic kidney disease has been regarded as a critical public health problem. with an increasing global prevalence. It is critical to identify factors that can predict and prevent its progression(Belloet al. 2005). Adipo.is a collagen-like plasma protein (247 amino acids) that is only found in adipose tissue (Hirako, 2016). It makes up approximately 0.05-0.1% of total serum proteins (Tsao et al., 2002). It is circulating at high levels (5-30mg/L) in the blood. It has three basic shapes: trimer (Low MW), hexamer (Middle MW), and multimer (High MW) (Achari and Jain, 2017).

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Patients with CKD who are on maintenance dialysis, both HD and peritoneal dialysis, have higher serum Adipo. levels. According to (Zoccali et al., 2003), Adipo. levels are higher in patients with renal dysfunction, especially at the end stage of the disease it might be an increased risk of death in patients with stage 3 or 4 of CKD. A 5-year follow-up research from Taiwan found high serum Adipo. increased risk of end-stage renal dysfunction in cases of non-diabetic CKD, as well as proteinuria(Kuo et al., 2019), is strongly linked to circulating Adipo. levels. Low Adipo. levels in serum have been linked to weight gain, insulin resistance(Balsan et al., 2015), Diabetes type 2, cardiovascular disease, metabolic syndrome (Chow et al., 2007), and atherosclerosis(Shimada et al., 2004). Adipo. is largely eliminated by the hepatic cells and is thought to play only a minor impact in

kidney clearance physiology (Halberg et al. 2009). Only Both monomers and dimers can pass through the glomerular filtration barrier and be detected in urine. Adipo. is found primarily in arterial endothelium and smooth muscle cells in the kidneys, capillary endothelium, and, epithelial cells in the brush border of the proximal and distal tubules. Adipo. may also be produced by proximal tubular cells, especially in inflammatory circumstances (Perri et al. 2013; Christou and Kiortsis 2014). Adipo. receptors are located on endothelial cells, podocytes, mesangial cells, and Bowman's capsule epithelium inside glomeruli. They're also found in the renal tubular cells (Kim and Park 2019). The present study aims to investigate the relationship between serum Adipo. levels and a variety of demographic and clinical parameters in CKD patients. We also examined the relationship between serum Adipo. levels and the stages of CKD.

Materials and methods

A case-control study was used to achieve the study objectives, it was performed at Dialysis Unit at Erbil Teaching Hospital in Erbil city. The current study included data collection, patient examination, and laboratory investigations (biochemical and hormonal analysis) from September 25th 2021 to the 1st of March 2022. The subjects of our study were grouped into two categories the first group of 100 patients with CKD, on dialyzer machines, and the second group of 100 healthy subjects who served as control, all were healthy volunteers and had no evidence of any renal disease. Patients with CKD of different causes are aged from 18 to 76 years. The study questionnaire and consent statement were translated into the local language to assure it was properly understood by the study subjects. After informing each subject about the study and obtaining their permission, the structured questionnaire was completed via direct interviews covering different variables. including knowledge about family history of kidney diseases. Measurements: this included Creatinine, albumin, urea, HbA1c, CRP, and Lipid Profile. After overnight fasting and before breakfast all subjects had their blood drawn. The blood samples were subjected to biochemical and hormonal assays. The other parts of the blood samples were drawn into tubes with anticoagulants. Samples were properly chilled directly, later serum was centrifuged, separated, and kept at -20°C until laboratory analysis. lipid profile, creatinine, albumin, urea, CRP, and HbA1c were measured by the COBAS INTEGRA 400 plus. A human commercial ELISA kit was used to measure total serum Adipo. concentrations. Every specimen was examined, and all findings were reported as mean values. eGFR was calculated using the CKD Epidemiology Collaboration formula based on biomarkers of filtration such as creatinine and age.

Statistical analysis

The data were analyzed with SPSS version 26 and the results have been expressed as mean and standard deviation. The data are normally distributed, and the chi-square test was used to compare multiple groups. the independent t-test was used to compare the studied parameters between control and case-study groups. A linear regression model with the Pearson correlation coefficient was used to assess the relationship among variables. In all tests, a p-value of less than 0.05 was considered statistically significant.

Results

We enrolled a total of 200 participants in our study and divided them into two equal groups, 100 cases, and 100 controls.

Table (1) shows that there was a significant statistical association between study groups and Adipo. levels, with the vast majority (98 %) of cases having high levels while none of the control study group had high levels, and the vast majority (96%) of the control group had low levels while only 1% of cases had a low level. The p-value for the Chi-square test was =0.001.

Table 1: Association between study groups and Adipo. level.

Variable	Categories	Study groups		p-value, Chi-square test
		Cases	Control	
Adipo. level	Low	1	96	0.001
		1%	96%	
	Normal	1	4	
		1%	4%	

	High	98	0	
		98%	0%	
Total		100	100	
		100%	100%	

Table (2) reveals that there was a significant statistical association between study groups and DM, there was no DM of any type in control groups and most (63%) of cases did not have DM while (20%) of cases had type 1 DM. The Chi-square test was done and the p-value was (0.001).

Table 2: Association between study groups and DM.

Variable	Categories	Study groups		p-value, Chi-square test
		Cases	Control	
DM	No	63	100	0.001
		63%	100%	
	Type I DM	20	0	
		20%	0%	
	Type II DM	17	0	
		17%	0%	
Total		100	100	
		100%	100%	

DM: diabetes mellitus

Results in the table (3) show that there was a significant difference and negative weak correlation between study groups and means of Adipo., serum creatinine, blood urea, albumin, TC, triglyceride, HDL, VLDL, CRP, eGFR, HbA1c, and BS. On average, the cases had a much higher Adipo. level of (13.26 µg/ml) compared to the lower level for the control group (only 2.17 µg/ml), the mean serum creatinine was (8.80 mg/dL) for cases in reverse to control group (0.59 mg/dL), as well as the mean blood urea was in a high level (178.82 mg/dL) compared to (16.13 mg/dL) for control study, albumin was (3.64 g/dL) for control and (2.07 g/dL) for cases, control group had (121.79 mg/dL) cholesterol while (158.20 mg/dL) TC in cases group bodies, cases group bodies released more (154.18 mg/dL) than control group (110.57 mg/dL) triglyceride, the high level (76.37mg/dL) of control group in HDL compared to (32.23 mg/dL) of cases group, cases were (78.84 mg/dL) less LDL than control group (82.04 mg/dL), cases group in VLDL and CRP takes a large place (30.90 mg/dL) and (1.30 mg/dL) compared to control groups (24.19 mg/dL) and (0.29 mg/dL). The mean HbA1c was 6.84% of cases compared to 4.91% of the control group, the mean BS, cases group released (161.65 mg/dL) while control groups (108.32) mg/dL.

Table 3: Differences between study groups regarding the numerical variables

variables	groups	Mean± Std. deviation	p-value
Adipo. (µg/ml)	Cases	13.26±6.95	0.001
	Control	2.17±0.75	
eGFR	Cases	6.63±2.86	0.001
	Control	113.72±16.75	
S. creatinine	Cases	8.80±2.56	0.001
	Control	0.59±0.15	
Albumin g/dL	Cases	2.07±0.49	0.001

	Control	3.64±0.68	
B. urea mg/ dL	Cases	178.82±44.18	0.001
	Control	16.13±3.43	
Total cholesterol mg/dL	Cases	158.20±30	0.001
	Control	121.79±34.18	
Triglyceride mg/dL	Cases	154.18±96.68	0.001
	Control	110.57±24.09	
HDL mg/dL	Cases	32.23±19.73	0.001
	Control	76.37±12.04	
LDL mg/dL	Cases	78.84±25.05	0.280
	Control	82.04±15.49	
VLDL mg/dL	Cases	30.90±19.32	0.001
	Control	24.19±5.74	
CRP mg/dL	Cases	1.30±1.95	0.001
	Control	0.29±0.25	
FBS (mg/dL)	Cases	161.65±68.91	0.001
	Control	108.32±14.41	
HbA1c%	Cases	6.84±2.21	0.001
	Control	4.91±0.49	

TC: Total Cholesterol, TG: triglycerides, HDL-C: high-density lipoproteins cholesterol, LDL-C: low-density lipoproteins cholesterol, VLDL: very-low-density lipoprotein cholesterol, HbA1: glycated hemoglobin, BS: Blood Sugar, eGFR: estimated glomerular filtration rate.

There was a negative very weak correlation between Adipo. with serum creatinine and albumin for CKD cases, for all cases the correlation coefficient (r) was less than 0.3 and these correlations were statistically non-significant with p-values more than 0.05, as shown in table (4).

Table 4: Correlation between Adiponectin and serum creatinine and albumin in CKD cases.

Measurements		Adipo. (µg/ml)	Interpretation
S. creatinine	Pearson Correlation	-0.141	Negative, weak correlation
	Sig. (2-tailed)	0.161	Non-significant
	N	100	
Albumin g/dL	Pearson Correlation	-0.044	Negative, weak correlation
	Sig. (2-tailed)	0.664	Non-significant
	N	100	

Discussion

The current study shows that circulating serum Adipo. levels, are significantly higher in CKD patients, as shown in table (1). Its levels elevated above normal in patients on maintenance HD had significantly greater in the cases of (13.26 µg/ml) compared to the lower level for the control group only (2.17 µg/ml) (P=0.01) as shown in (Tables2 and 3). Similar results have been reported by (Markaki et al., 2016), with Adipo. levels (20.5 g/ml), and (Menon et al., 2006) mean ± SD was (12.8 ± 8.0 µg/ml). Our result is incompatible with previous studies that have found elevated serum Adipo. levels in CKD patients(Kamal, 2014; Rhee et al., 2015; Kim et al., 2016).

The underlying mechanisms that link high serum Adipo. levels and kidney disease progression are indefinite. (Kim and Park 2017). Adipo. a

vasoactive adipokine accounts for approximately 0.01 % of plasma protein in humans. (Arita et al., 1999). In kidneys is found in the arterial endothelium, smooth muscle cells, and capillary endothelium(Christou and Kiortsis 2014). when the kidneys are damaged proximal and distal tubule epithelial cells secrete more Adipo.(Perri et al., 2013).

The outcome of this study noticed a highly significant difference (P=0.01) between the cases and control groups table as shown in tables (2 and 3). Diabetic renal failure is negatively correlated with insulin resistance, fasting serum insulin, and fasting blood glucose levels (Orchard et al., 2002; Saraheimo et al., 2003).

In this study, higher Adipo. was significantly associated with both worsening renal function and increased proteinuria after measuring renal function and eGFR (Table 3). High circulating Adipo. levels have been observed in patients with CKD due to impaired urinary excretion, set the clearance of serum Adipo. through glomerular filtration, indicating that the kidneys contribute to the elimination of this protein.

The National Kidney Foundation (NKF) published clinical practice guidelines on CKD in 2002, recommending estimating eGFR using a prediction equation that includes serum creatinine measurements(Levey et al., 2000; Johnson et al., 2004; Shahbaz and Gupta, 2021).
The eGFR is a measure of how well the kidneys filter blood. It is calculated using patients' serum creatinine and age. The current study found a lesser eGFR in comparison with the control group as shown in table (3). A similar study by(Menon et al., 2006) found mean ± SD (GFR) in cases was (33 ± 12 ml/min per 1.73 m²). Moreover, serum Adipo. levels were significantly higher in CKD patients than in the control group (P=0.01), and plasma Adipo. was inversely related to the (eGFR) in CKD patients (r=-0.570, P<0.001)(Sedighi and Abediankenari, 2013).

Creatinine levels significantly increased as a result of kidney damage(Moses and Johnkennedy, 2013). In this study, the mean serum creatinine was (8.80 mg/dL) for cases in reverse to the control group (0.59 mg/dL) (P=0.01), there was a negatively weak correlation between them as in table (3 and 4), similar results have been reported by (Fink et al., 1999) with the mean creatinine levels among patients on HD were (9.2 ± 0.1 mg/dL). Because creatinine and urea concentrations change inversely with GFR, they can be used to assess the degree of kidney damage.

Lesser albumin in our study with (2.07 g/dL) for cases, and (3.64 g/dL) for control(P=0.01), in the table (3 and 4), Hypoalbuminemia is one of the most important risk factors for death in HD patients. These findings were supported by many researchers (Yeun and Kaysen 1997; Bologa et al. 1998; Kaysen et al. 2004)who have indicated that decreased serum albumin levels have been linked to nutritional inflammation markers in HD patients.

We found an increase in blood urea, which was at a higher level compared to the control study, and significantly (P=0.01), in table (3) are inconsistent with other studies (Shavit et al., 2012; Azra, 2014). Urea excretion is influenced by hydration, water re-absorption, and GFR; any changes in blood urea levels indicate kidney damage; when the function drops below 25-50% of normal it becomes elevated (Sharma et al. 2011). This increased mean value indicated that there was a slight obstruction in excreting urea in kidney disease patients, as well as an impairment of renal function, either due to a decrease in GFR or an obstruction that interfered with urinary excretion (Kamal, 2014).

The relationship between Adipo. and plasma lipid levels have been reported in several studies in which the concentration of Adipo. is inversely correlated with body fat mass (Cnop et al., 2003; Baratta et al., 2004; Swarbrick and Havel, 2008; de Carvalho et al., 2009; Rhee et al., 2015).
In our study, the comparison between groups showed that serum TC levels varied significantly among groups (case: 158.20±30mg/dL, control:121.79±34.18 mg/dL, P = 0.01)as shown in table (4), this finding is in agreement with the previous studies with (194.80±30.75 mg/dL) in CKD patients (Singh et al., 2019).

The mean value of serum TG in CKD patients was higher than the control group, The comparison between groups showed that serum Triglyceride levels varied significantly among groups (case: 154.18 ±96.68 mg/dl, control: 110.57 ±24.09 mg/dl, P= 0.01) shown in table (3). Similarly, the results (223.91±65.69) for CKD patients with HD,

the study by(Singh et al., 2019). Patients with HD often suffer from moderate hypertriglyceridemia, and the heparin used in HD inhibits lipoprotein lipase (LPL), which is responsible for the destruction and hydrolysis of TG(Näsström et al., 2003).

HDL levels are generally lower in CKD patients, particularly those on dialysis(Riwanto et al., 2015), this is a result of impaired maturation of HDL, which is associated with lower levels of apolipoproteins and cholesterol acyltransferase, abnormal post-translational modifications, and decrease in the metabolism of triglyceride-rich lipoproteins(Tward et al. 2002; Vaziri 2010).

The current study found difference in HDL in CKD patients compared to healthy patients and it was also statistically significant (case: 32.23±19.73 mg/dl, control: 76.37±12.04 mg/dl, (p =0.01)), shown in table (4). This result is in agreement with(Schaeffner et al. 2003; Vaziri 2010).

Lipid abnormalities (dyslipidemia) are very common in CKD patients. Patterns of dyslipidemia are inconsistent across different stages and categories of CKD, as different lipoprotein spectra are observed in CKD patients who are not undergoing HD and who are undergoing peritoneal dialysis.
In this study the level of serum LDL in patients with CKD was non-significant (P=0.280) it was lesser in comparison with the control group (case: 78.84±25.05 mg/dl, control: 82.04±15.49mg/dl) as shown in table (4). Similar research reported a negative relationship between CKD and serum LDL cholesterol levels that is due to malnutrition and inflammation in dialysis patients(Lowrie and Lew, 1990; Kilpatrick et al., 2007; Lacquaniti et al., 2010).
Several mechanisms may contribute to the impaired LDL metabolism, and dialysis may result in additional lipid homeostasis defects such as increased catabolic rate of (Apolipoprotein AI) protein roles that have a specific role in lipid transport and metabolism that support the clinical expression of these mechanisms(Attman et al. 1999; Näsström et al. 2005; Vaziri 2006).
We observed in the present study the proportion of TG in VLDL in dialysis patients was slightly higher but significantly increased compared to controls (cases: 30.90 ± 19.32 mg/dl, controls: 24.19 ± 5.74 mg/dl), P =0.001), as in table (3). Similar result with (102.63±21.05 mg/dL) for patients with HD(Baria et al., 2013). The increased VLDL cholesterol level is due to VLDL catabolism that is delayed. Uremia has a low HDL cholesterol content as well as a low (Apolipoprotein CII) activator for lipoprotein lipase concentration. Normally, Apolipoprotein C-II is transferred from HDL to VLDL in the plasma. Reduced Apolipoprotein C-II levels result in decreased triacylglycerol catabolism and VLDL metabolism. As a result, the VLDL concentration rises, this result is in agreement with previous results (Drueke and Lacour, 1995; Baria et al., 2013).

CRP is a protein produced by the liver. It is released into the bloodstream as a result of inflammation it is increased by 30 to 50% in patients on dialysis, The effects of Adipo. include the decreased phagocytic activity of macrophages, inhibition of macrophage production of inflammatory cytokines, and decreased adipose tissue.(Kaysen et al., 2004; Welters et al., 2014). In our study, the CRP was significantly correlated with Adipo. (case: 1.30±1.95 mg/dl, control: 0.29±0.25 mg/dl, P = 0.001) shown in table (4), This increase is a result of many factors, including cardiovascular damage, poor oral, and periodontal health, graft or fistula infections, incompatible dialysis membranes, dialysates, endotoxin exposure, back filtration, chronic infections, and malnutrition(Rao et al., 2006; Nadeem et al., 2009; Pejcic et al., 2011; Brito et al., 2012; Yoo et al., 2012).

Adipo. improves hepatic and systemic insulin resistance in the liver via activating the adenosine monophosphate-activated protein kinase and peroxisome proliferator-activated receptors(AMPK-PPAR-pathways), which increases fatty acid oxidation as well as reduces gluconeogenic availability, and inhibits both glycogenolysis and gluconeogenesis (Combs et al., 2001) this is by inhibiting rate-limiting enzymes for hepatic glucose production(Matsuda and Shimomura, 2014). Adipo. can inhibit glycogen synthesis by limiting the availability of gluconeogenic substrates, in addition to suppressing glucose 6-phosphatase and Phosphoenolpyruvate carboxykinase(Miller et al., 2011). In our study, the comparison between groups showed that fasting blood sugar levels

were higher significantly among groups (case: 161.65±68.91 mg/dL, control: 108.32±14.41 mg/dL, P = 0.01) shown in table (4). Similar results in CKD patients on dialysis with (187 ±82 mg/dL)were found by (Lee et al., 2013).

For measuring glycemia only fasting glucose levels for people who have mild diabetes are not given a complete measure, and postprandial glucose levels may better explain variations in mean glycated hemoglobin concentration (HbA1c) (Hernandez et al., 2013). Our study shows a significant association as in table (3) has been found between HbA1c and CKD (case: 6.84 ±2.21 mg/dL, control: 4.91±0.49 mg/dL, P =0.01). A similar result was recorded by (Bloomgarden and Handelsman, 2017).

CONCLUSIONS

1- We demonstrated in the current study higher serum Adipo. The level was a significant association according to the results we get, which might be associated with CKD.
2- This study demonstrates a statically significant difference in the mean concentration of fasting serum T. Ch, TG, LDL-C, and HDL-C between normal healthy persons and patients with CKD.
3- The negative association relationship between adiponectin, creatinine, albumin, and urea levels In CKD patients.
4- The results showed a significantly higher serum adiponectin concentration in patients with diabetes.

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Conflict of Interest

The authors declared that they have no conflicts of interest.

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