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Evaluation of adipocytokines and oxidative stress biomarkers in sera of hemodialysis patients

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ABSTRACT

Chronic renal failure is a condition characterized by the inability of the kidney to effectively eliminate toxins, solutes, and excess water, necessitating the use of hemodialysis. The current study aimed to evaluate adipocytokines and oxidative stress biomarkers in sera of hemodialysis patients. This study involved 60 hemodialysis patients, of whom 64% were female and 36% were male. Additionally, 40 healthy control volunteers participated in the study. Adipocytokines and oxidative stress markers were measured using the ELISA technique, whereas the spectrophotometric method measured lipid profile and renal function. The t-test, correlation, receiver operating characteristic, and logistic regression were used in the statistical analysis. Patients undergoing hemodialysis exhibited significant differences compared to healthy controls in serum concentrations of several parameters. Significantly higher levels of adiponectin (23.46 ng/mL), resistin (5.50 ng/mL), visfatin (47.0 ng/mL), and chemerin (169.0 ng/mL) were measured in patients undergoing hemodialysis as compared to healthy controls. Similarly, significantly higher levels of oxidative stress markers such as superoxide dismutase (3.78 U/mL), glutathione peroxidase (129.79 pg/mL), and malondialdehyde (4.66 mmol/L) were measured in patients undergoing hemodialysis as compared to healthy controls. In conclusion, adipocytokine parameters and oxidative stress markers have been found to be abnormal in hemodialysis patients and have an effect on atherosclerosis and heart failure progression.

INTRODUCTION

Most kidney disorders lead to a reduced glomerular filtration rate and elevated urea levels typically progress to chronic renal failure (CRF) [1]. CRF is a condition considered by the inability of the kidney to effectively eliminate toxins, solutes, and excess water, necessitating the use of hemodialysis. A key characteristic of CRF is inflammation, a major contributor to mortality in dialysis patients. As a result of frequent dialysis treatments, patients with CRF tend to produce increased levels of cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which are associated with increased chronic inflammation and decreased leukocyte activation [2] as well as affects the central nervous system and causes a loss of appetite due to the elevated release of pro-inflammatory cytokines, which disrupts hormone production associated to central regulation of appetite [3].

The inflamed dysfunctional kidneys result from the deficiency in several enzymes, including high-density lipoproteins (HDL) and Apo A1. The inflammation affects methionine synthase, impairing the clearance of homocysteine and leading to hyperhomocysteinemia and anaemia due to vitamin B12 deficiency [4, 5]. Oxidative stress is linked directly or indirectly with renal diseases [6]. Oxidative stress can arise through various mechanisms, including an excess of reactive oxygen species (ROS), which is a primary cause of liver disorders [6]. Moreover, in diabetes, both type I and

type II patients are prone to elevated levels of oxidative stress, which significantly contributes to disease pathogenesis due to a deficiency in antioxidant enzymes [7]. Additionally, in other inflammatory autoimmune conditions, certain elements like copper (Cu) can exert pro-inflammatory effects, leading to increased oxidative stress [8].

Adipokines currently play a significant function in the biology of many different human organs. End-stage CRF is known to have a directly related and strong association with several variables like adipokines and adipose tissue contributing more than other biological components [9].

Although dialysis is unable to fully recover the lost functions of the kidneys, it still partially maintains the kidney [10]. However, patients with CRF are often experiencing either continuous ambulatory peritoneal dialysis (CAPD) or hemodialysis (HD) [11]. In HD-CRF, the risks of bleeding complications, infections, electrolyte imbalances, and cardiovascular dysfunction increase [12]. These data show the importance of evaluating parameters like lipoproteins, adipocytokines, and oxidative stress, especially in CRF patients undergoing HD. Thus, the current study aims to evaluate adipocytokines and oxidative stress biomarkers in sera of hemodialysis patients.

MATERIALS AND METHODS

Subjects

The study involved 100 participants, who were divided only into two groups. Sixty participants were in the first group who suffered from CRF and were undergoing HD. At the same time, the control group included 40 healthy individuals of the same age and sex as the patients. Blood samples were collected in gel tubes and sodium citrate tubes, centrifuged, and then stored at –20°C. The study was approved by the University of Technology (UOT) Biological Research Ethical Committee, within the guidelines of the World Medical Association Declaration of Helsinki Ethical Principles (2013) requirements and given the number AS-AC 45038. All patients gave their informed consent to participate in the trial.

The sample selection was based on the clinical diagnosis of CRF and patients having undergone HD for two years. The study was conducted at Al Karama Teaching Hospital between February 2023 and November 2023, and all patients were over the age of 18.

Exclusion criteria included HD patients with durations of treatment either shorter or longer than two years, as well as individuals with a history of heart diseases, diabetes mellitus, chronic inflammatory disorders such as rheumatological diseases, and liver failure. Pregnant and lactating women were also excluded. Additionally, all patients participating in the trial were not taking any medication that could potentially interfere with the study results.

Analysis of lipid profile and renal function

The equipment utilized in this investigation was Roche Cobas e411 (Cobas Lipid Panel, Roche Diagnostics, Mannheim, Germany) for performing some laboratory tests, including total cholesterol (TC), triglycerides (TG), low-density lipoproteins (LDL), HDL, and very low-density lipoproteins (VLDL) as a lipid profile. In addition, renal function was determined by measuring urea and creatinine (Spinreact. Sant Esteve d'en Bas, Spain).

Analysis of adipocytokines and oxidative stress biomarkers

The adipocytokine profile and oxidative stress markers were measured by the sandwich ELISA method, adiponectin (Cat. No.: CAN-APN-5000; Diagnostics Biochem Canada Inc., Canada), Resistin (Cat. No.: ARG80885; Eagle Bioscience Inc., Canada), Visfatin (Cat. No.: EH482RB; Thermo Fisher Scientific Inc., USA), Chemerin (Cat. No.: KT-9860; Kamiya Biomedical Company, USA), Superoxide Dismutase (SOD) activity (Cat. No.: CS0009; Merck, Germany), Glutathione Peroxidase (GPx) (Cat. No.: MAK437; Merck, Germany), malondialdehyde (MDA) (Cat. No.: E-BC-K028-M; Elabscience, USA), which uses a purified antibody to capture antigen, increasing sensitivity and specificity.

Statistical analysis

The analysis of data was conducted using the Statistical Package for Social Sciences version 26.0 (SPSS v26). Mean± standard deviation (SD) or percentage used to display the results. Independent sample *t*-tests were utilized to compare the data. To determine the accuracy of adipocytokines between HD patients and the control group, we used the Receiver Operating Characteristic (ROC) analysis. Additionally, the Pearson rank correlation and logistic regression correlation tests have been applied. A statistically significant value was set at $P < 0.05$.

RESULTS

Demographic characteristics

Table 1 illustrates the demographic characteristics of both patients with HD and healthy controls. There was a significant higher in BMI, TC, TG, LDL, urea, and creatinine in HD patients as compared to healthy controls (Table 1). However, there was a significant decrease in HDL in HD patients as compared to healthy controls (Table 1).

Table 1. Demographic characteristics of patients with HD and controls.

Characteristic		Patients (n=60)	Healthy control (n=40)	*P-value*
Age (years)		37.17 ± 8.02	41.85 ± 11.87	0.020*
Sex	Male	24 (40.0%)	12 (30.0%)	0.307
	Female	36 (60.0%)	28 (70.0%)	
BMI kg/m^2		28.90 ± 3.52	26.13 ± 3.03	< 0.001*
Lipid Profile				
TC mg/dL		191.43 ± 22.2	140.65 ± 27.59	< 0.001*
TG mg/dL		186.13 ± 16.44	117.95 ± 26.13	< 0.001*
HDL mg/dL		38.78 ± 3.19	51.0 ± 8.47	< 0.001*
LDL mg/dL		144.38 ± 18.19	99.57 ± 22.62	< 0.001*
VLDL mg/dL		30.75 ± 5.37	22.55 ± 6.28	< 0.001*
Renal Function				
Urea mg/dL		30.98 ± 3.87	22.45 ± 6.60	< 0.001*
Creatinine mg/dL		2.33 ± 0.55	0.72 ± 0.28	< 0.001*

* Significant value $p < 0.05$

Levels of oxidative stress markers in HD patients

The level of oxidative stress markers between HD patients and controls is shown in Figure 1. SOD concentrations in HD patients were significantly decreased. Interestingly, GPx and MDA concentrations were significantly elevated in HD patients as compared to healthy controls (Figure 1).

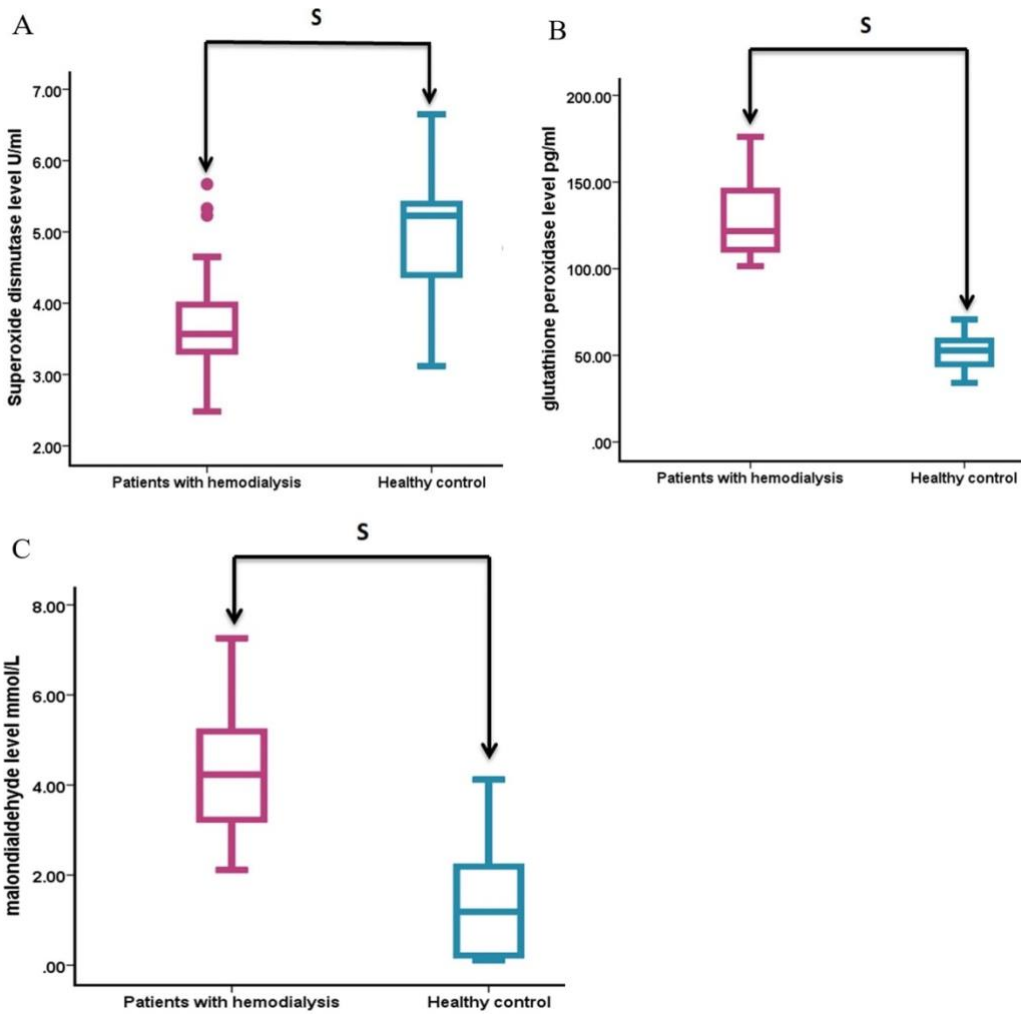

Figure 1. Serum oxidative stress parameters concentrations such as (A) levels of superoxide dismutase (SOD) in HD patients and controls, (B) levels of glutathione peroxidase (GPx) in HD patients and controls, and (C) levels of malondialdehyde (MDA) in HD patients and controls. S indicates $P < 0.05$.

Levels of adipocytokines in HD patients

The results of the adipocytokine profile (adiponectin, resistin, visfatin, and chemerin) between HD patients and controls are shown in Figure 2. The concentrations of adiponectin, resistin, visfatin, and chemerin were significantly increased in HD patients as compared to healthy controls (Figure 2).

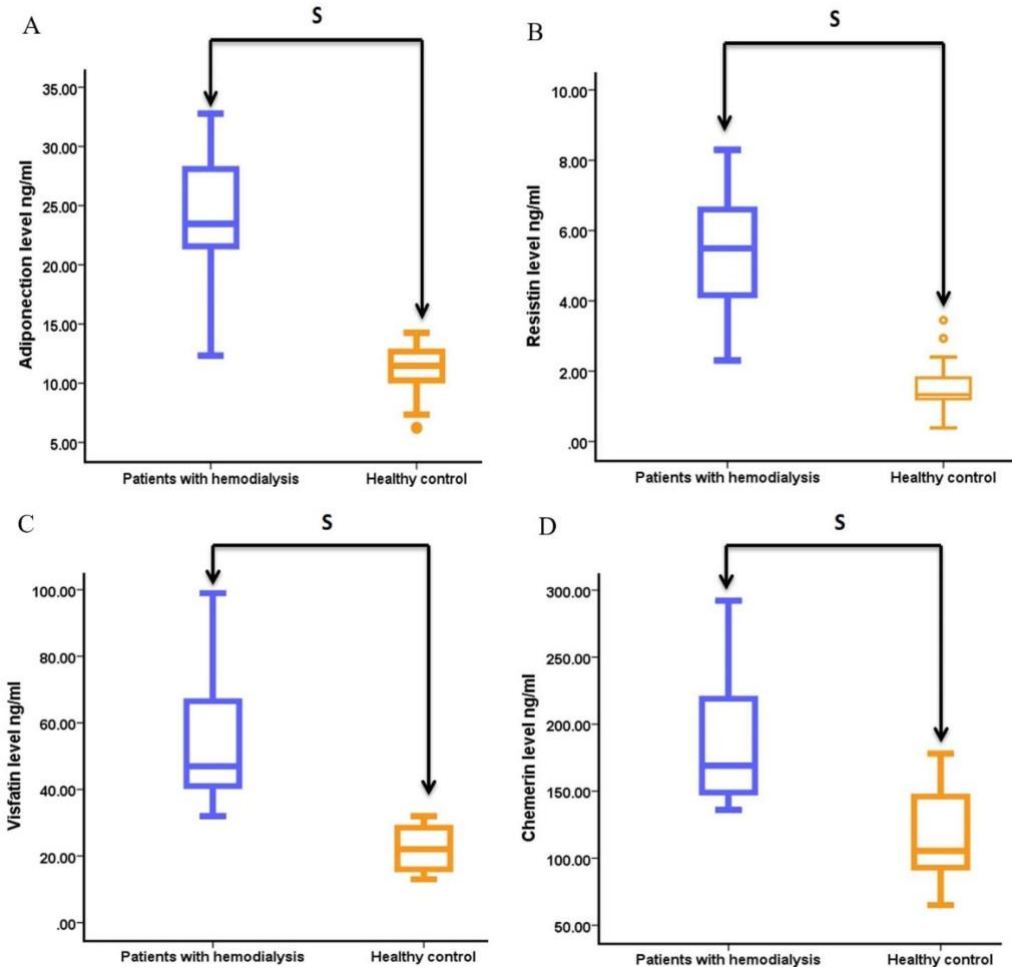

Figure 2. Serum adipocytokines profile concentrations such as (A) levels of adiponectin in HD patients and controls, (B) levels of resistin in HD patients and controls, (C) levels of visfatin in HD patients and controls, and (D) levels of chemerin in HD patients and controls. S indicates $P < 0.05$.

Diagnostic accuracy of adipocytokines profile in HD patients

Table 2 and Figure 3 show the receiver operating characteristic (ROC) analysis results of adiponectin, resistin, visfatin, and chemerin. The optimal cut-off values for adiponectin, resistin, visfatin, and chemerin were 16.79 ng/mL, 2.60 ng/mL, 32.5 ng/mL, and 147.5 ng/mL, respectively (Table 2 and Figure 3).

Table 2. ROC analysis of adipocytokines profile.

Characteristic	Adiponectin	Resistin	Visfatin	Chemerin
Cutoff value	> 16.79	> 2.60	> 32.5	> 147.5
P value	< 0.001*	< 0.001*	< 0.001*	< 0.001*
Sensitivity %	98.3 %	96.7 %	98.3 %	83.0 %
Specificity %	100.0%	95.0%	100.0%	82.5%
PPV %	100.0 %	96.7 %	100.0 %	87.7 %
NPV %	97.6%	95.0%	97.6%	76.7%
AUC (95% CI)	0.995 (0.984- 1.000)	0.991 (0.979- 1.000)	0.998 (0.994- 1.000)	0.910 (0.85-0.966)

* Significant value $p < 0.05$

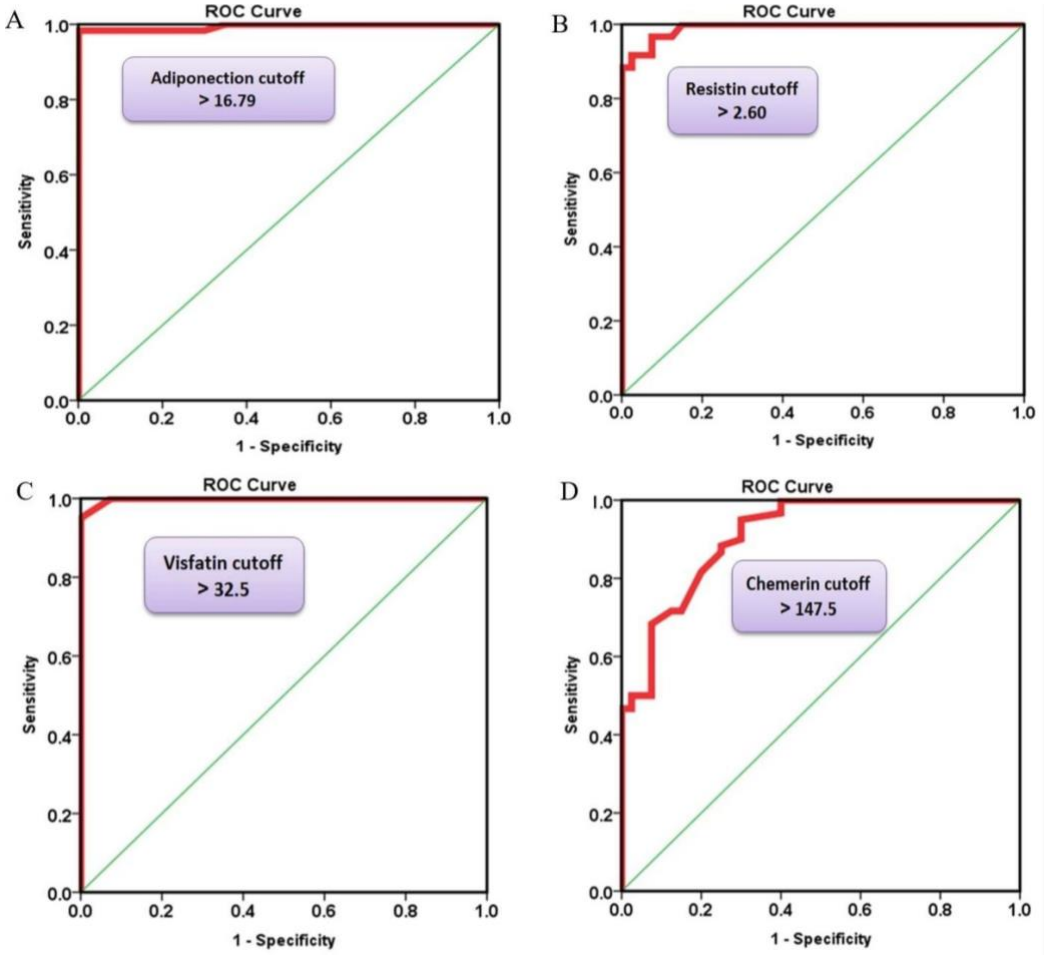

Figure 3. ROC curve of adipocytokines profile for the calculation of possible diagnostic cut-off value (A) Adiponectin ROC curve with cut-off value > 16.79, (B) resistin ROC curve with a cut-off value > 2.60, (C) visfatin ROC curve with cut-off value > 32.5, and (D) Chemerin ROC curve with cut-off value > 147.50.

Correlation between adipocytokines profile and other parameters in HD patients

Table 3 presents the correlations between the adipocytokine profile and other parameters in HD patients. The present results of resistin and HDL levels showed a significant negative correlation ($r = -0.339$, $p = 0.009$). Additionally, a significant positive correlation between visfatin and urea level ($r = 0.279$, $p = 0.031$) as well as between Chemerin and TC level ($r = 0.303$, $p = 0.019$) in HD patients, was found (Table 3). Conversely, all other factors examined did not show significant correlations with one another (Table 3).

Logistic regression correlations between adipocytokines profile in HD patients

Figure 4 illustrates the logistic regression model, highlighting that adipocytokines, particularly adiponectin, have a direct correlation with resistin in HD patients (Figure 4). Additionally, adiponectin also showed direct correlations with both visfatin and chemerin among patients.

Table 3. Correlation between adipocytokines profile and other parameters.

Characteristic	Adipocytokine profile							
	Adiponection		Resistin		Visfatin		Chemerin	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
Adiponectin	1							
Resistin	0.046	0.728	1					
Visfatin	0.139	0.289	0.075	0.569	1			
Chemerin	0.020	0.927	0.106	0.419	0.092	0.484	1	
BMI kg/m^2	0.087	0.509	0.054	0.679	0.036	0.788	0.064	0.628
TC mg/dL	0.128	0.328	0.004	0.979	0.169	0.196	0.303	0.019*
TG mg/dL	0.202	0.121	0.018	0.894	0.244	0.061	0.161	0.220
HDL mg/dL	-0.083	0.527	-0.339	0.009*	-0.096	0.464	0.020	0.877
LDL mg/dL	0.050	0.705	0.078	0.555	0.120	0.360	0.071	0.588
VLDL mg/dL	0.226	0.083	0.082	0.532	0.148	0.258	0.101	0.442
Urea mg/dL	0.233	0.073	0.055	0.678	0.279	0.031*	0.036	0.786
Creatinine mg/dL	0.008	0.949	0.078	0.552	0.123	0.417	0.135	0.305
SOD U/mL	-0.113	0.389	-0.141	0.284	-0.169	0.198	-0.137	0.298
GPx pg/mL	0.043	0.723	0.032	0.805	0.047	0.723	0.126	0.336
MDA mmol/L	0.068	0.604	0.092	0.483	0.036	0.787	0.096	0.464

* Significant value $p < 0.05$

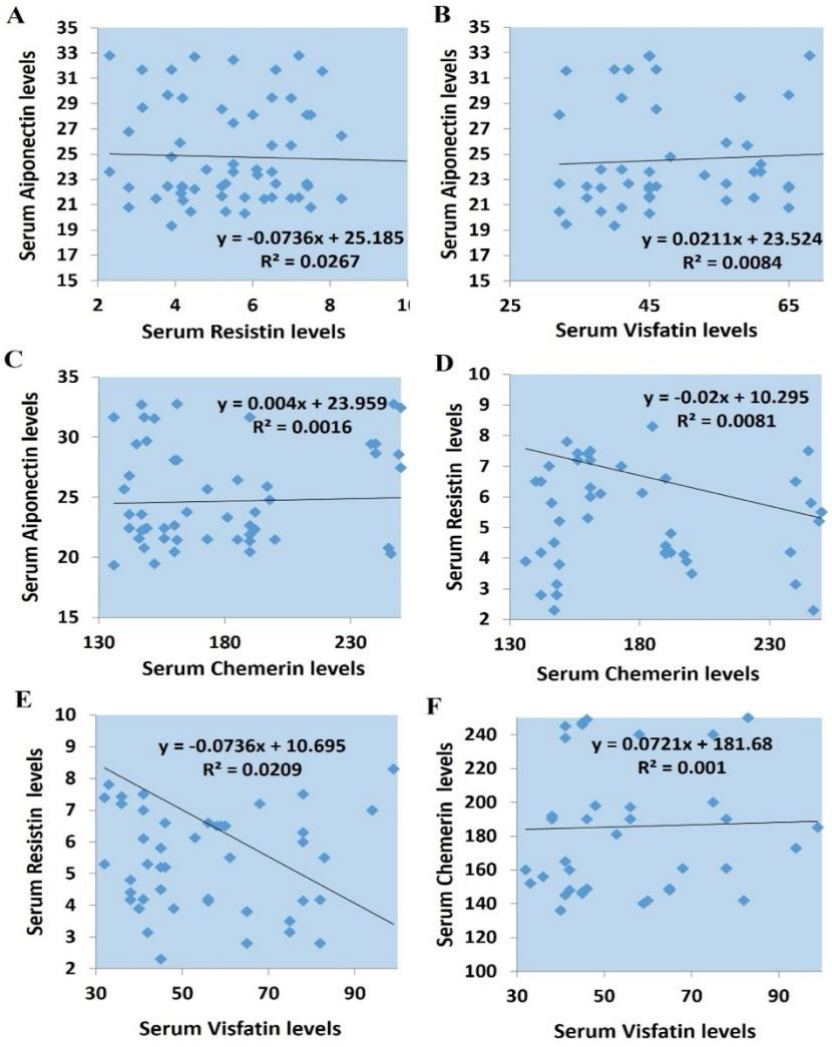

Figure 4. Logistic regression correlations between adipocytokines profile. A) Correlation between adiponectin and resistin, B) correlation between adiponectin and visfatin, C) correlation between adiponectin and chemerin, D) correlation between resistin and chemerin, E) correlation between resistin and visfatin, and F) correlation between chemerin and visfatin.

DISCUSSION

In the current study, oxidative stress biomarkers (SOD, GPx, and MDA), and adipocytokines (adiponectin, resistin, visfatin, and chemerin) had significantly changed in HD-CRF patients. The findings showed an elevated level of oxidative stress biomarkers (except SOD) and adipocytokines, alongside changes in the lipid profile and renal function, while SOD levels were found to be decreased. However, the selection of HD-CRF patients was only on two years HD was not excessively long nor short duration. This approach aimed to obtain data from individuals with the same population. Moreover, the duration of HD can affect various indicators, including oxidative stress and adipocytokines [13, 14].

Variations in cell surface receptor expression and receptor phenotypic abnormalities are frequently observed in CRF. These changes not only affect the bioavailability of the dialysis membranes but also contribute to an accumulation of uremic toxins and a reduction in the kidney's ability to remove harmful substances, which leads to an increase in the synthesis of cytokines, like TNF-a, alongside these receptor alterations [2, 15].

In addition to TNF-α receptors, adiponectin, which is secreted by adipose tissue and enhances CRF, shows a significant association with mortality in patients undergoing HD [16]. Increased levels of TNF-α in individuals with heart failure and renal disease may contribute to pro-apoptotic processes and detrimental inotropic effects, potentially leading to cardiac damage. However, adiponectin levels have been linked to various biological mechanisms, including the regulation of pro- and anti-inflammatory cytokine production, improved insulin sensitivity in liver and skeletal muscle, reduction in atherogenic risk, and the maintenance of endothelial homeostasis [17]. Our results are consistent with Małgorzewicz et al., suggesting that the increase in both resistin and adiponectin may be due to impaired waste removal and biodegradation from the circulatory system rather than excessive secretion [18], and Ayerden et al. suggested that this contributes to the development of left ventricular hypertrophy, as elevated adiponectin levels seem to be associated with increased proinflammatory cytokines. [19]. That is supported by the fact that people with CRF have a 10- to 30-fold increased risk of cardiovascular disease (CVD) [20].

The elevated resistin levels observed in HD patients may be a result of increased inflammatory cytokines, likely linked to impaired renal function [21]. Elevation of serum resistin can cause cardiovascular mortality, where an incident of heart failure was correlated with higher resistin concentrations, especially in the end stages of renal diseases [22], which makes resistin a potential target for atherosclerosis patients [23]. However, there is evidence of a relation between raised resistin concentration and impaired glomerular filtration rate, suggesting that resistin might associated with HD patients with malnutrition [24]. The upregulation of circulating resistin, along with low HDL levels often observed in CRF patients and reflected in our findings, may be linked to HDL's suppression of resistin production from adipocytes. This suppression promotes lipid production by hepatocytes and VLDL-ApoB formation. Reduced HDL levels could thus contribute to the atherosclerotic process [25, 26].

The findings show that visfatin levels are elevated in HD-CRF patients alongside increased urea concentrations. Considering the number of studies linking visfatin to metabolic diseases such as obesity, diabetes, and insulin resistance, there is potential that visfatin plays a pathophysiological role in these conditions, which could have therapeutic implications [27]. Visfatin is a key factor in atherosclerosis's early stages, particularly in endothelial dysfunction. This connection makes the formation of

atherosclerotic plaques, and the inflammation associated with elevated visfatin levels conceivable [28, 29].

Based on chemerin's ability to regulate adipocyte gene expression, insulin resistance, regulation of immune response, and adipose maturation, it could be significantly contributing to nephropathy pathogenesis [30, 31]. Chemerin accumulation in the blood may be induced by reduced renal catabolism or clearance of chemerin, which might be one explanation for the higher serum chemerin levels in CRF [32]. Chemirin's function as a chemoattractant protein might make it play a role in the early stages of inflammation of adipose tissue by attracting macrophages into the tissue [31], and it can also play a role in atherosclerosis and CVD development through the endothelial lipid deposition effect [33].

SOD levels in patients of HD-CRF were found to be significantly decreased in the current study compared to healthy controls. This finding may be related to the increased ROS in plasma and the red blood cells of CRF patients as a result of oxidative stress [34]. However, previous studies have indicated that SOD enzyme activity is significantly decreased in CRF patients and CVD compared to those with CRF alone. This reduction may be due to an imbalance between antioxidant and oxidant enzymes, which, in both animal and human models, contribute to the progression and development of atherosclerotic disease [35, 36]. In contrast to research [37, 38] that found a substantial decrease in GPx activity in CRF, our investigation revealed elevated GPx levels. This increase may represent a defense mechanism for the cells against the excessive generation of free radicals related to CRF. Uremic problems and increased oxidative damage may be attributed to the reduction of antioxidant activity in red blood cells in patients with uremia undergoing CAPD [39]. However, a comparison of GPx concentrations before and after HD showed that both pro- and antioxidant indicators improved after treatment, although they remained significantly different from those in the control group [40, 41]. Compared to healthy controls, MDA concentrations in HD-CRF patients are elevated. MDA, which is generated from the polyunsaturated fatty acids peroxidation in conjunction with proteins, may exhibit atherogenic properties [42]. The result agreed with Sreenivasulu et al. [43] and Sridhar et al. [44], who suggest that the results of oxidative stress from the excessive free radical production are present in the lipid components of cell membranes. Consequently, MDA serves as a valuable biomarker for lipid peroxidation in degenerative diseases such as CRF [45]. As a key marker of CVD, oxidative stress may lead to apoptosis, necrosis, and ultimately thrombosis of atherosclerotic plaques. This occurs due to the action of certain ROS, such as O_2, which is converted into H_2O_2 with the help of SOD and GPx, affecting molecules that support endothelial function [46].

Adipocytokine levels have been associated with oxidative stress parameters in HD patients with various conditions. There was a relationship between plasma MDA and adiponectin levels in HD patients, according to Lim P. et al., who supported the claim that oxidative stress and adiponectin are associated with increased CVD and lipid metabolism in HD patients. Also, insulin resistance and chronic inflammation in diabetes-HD patients may cause adipocytokine dysregulation associated with augmented oxidative stress [47]. That led to the suggestion that oxidative stress and adiponectin expression in adipose tissue may be targets for treatment approaches [48]. However, our results appear to indicate that there is no significant correlation between adipocytokines and either the lipid profile or BMI, indicating that cardiovascular risk factors in patients with HD-CRF are unrelated to malnutrition. These findings are consistent with earlier research that found oxidative stress and the severity of chronic inflammation to be unrelated to BMI [49].

One of the main limitations of this study is that all patients who presented at the hospital were in the late stages of CRF and were already on hemodialysis, making it impossible to compare their pre- and post-HD status. Consequently, other tests could not be conducted, such as those for certain electrolytes, that could have provided comparisons between CRF patients before and after HD.

CONCLUSIONS

The current research offers an overview of some adipocytokines and oxidative stress markers that contribute to the enhanced risk of heart diseases associated with CRF during HD. The majority of parameters that are abnormal in HD-CRF have an influence directly or indirectly on atherosclerosis and heart failure progression. These effects are explained by a rise in the production of proinflammatory cytokines associated with CVD, endothelial lipid deposition, or imbalances in certain antioxidant enzymes.

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None.

AUTHOR CONTRIBUTIONS

SFO and MSH prepared overviews and composed the text. IAR data was analyzed with experiments carried out. MSH prepared the manuscript's first draft. SFO, IAR, and MSH reviewed the scientific material that was presented in the article. The final draft was examined and authorized by all authors before submission.

CONFLICTS OF INTEREST

There is no conflict of interest among the authors.

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