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Evaluation of Serum Lipid Profile and Renal Function Levels in Iraqi Patients with Chronic Kidney Disease (CKD)

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Abstract

Chronic Kidney Disease (CKD) has been raised as a public health state worldwide and is associated with increased cardiovascular morbidity and mortality. The aim of the study was to assess serum lipid profile levels, renal function and glucose in CKD patients undergoing hemodialysis. **A total of 50** patients (23 males and 27 females) with CKD attending the hemodialysis at Kidney Unit in Al-Ramadi Teaching Hospital, Anbar Iraq and of ages ranging between 20 -85 years, were sustained for this research. The patients were classified into three categories with their mean age of 65.71±1.98 years. Eleven healthy adults were also recruited in this study with mean age of 37.00±15.56 years as control group. The study included measuring biochemical markers such as glucose, total cholesterol(TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), very-low-density lipoprotein (VLDL) , creatinine and urea by using automated chemistry analyzer by Abbott/USA**.** The outcomes of this study showed that the urea and creatinine levels increased significantly, whereas HDL-C level significantly decreased in the patients as compared with control group. However, TC and LDL-C showed slightly lower levels which were within the normal limits in the control group. While TG, VLDL and glucose levels showed non-significant changes in CKD patients as compared with control ($P \leq 0.05$). In addition, all parameters revealed no significant changes in males as compared with females group ($P \leq 0.05$). Depending on age, only creatinine level showed significant change in 20-40 years age group as compared with 61-85years age group ($p < 0.05$). Finally, abnormal lipid metabolism may probably be an independent risk factor for the progression of renal disease.

Keywords: *Lipid profile, Chronic kidney disease, Renal function tests, Lipoproteins, Dyslipidemia*

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تقييم مستويات الدهون واختبارات وظائف الكلى في المرضى العراقيين المصابين بمرض الكلى المزمن

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الخلاصة:

مرض الكلية المزمن (CKD) هو حالة صحية خطيرة عالميا نتيجة لارتفاع معدل انتشاره وحدوثه في جميع انحاء العالم وارتباطه بزيادة معدل الوفيات والاعتلالات في امراض القلب الوعائية. ان الهدف من بحثنا هو تقييم مستويات مرتسم الدهون،اختبارات وظائف الكلية وسكر الدم في المرضى المشخصين بمراض الكلية المزمن والذين يخضعون لغسيل الكلية في وحدة الديلزة في مستشفى الرمادي التعليمي محافظة الانبار / العراق، ومقارنة نتائجهم احصائيا مع مجموعة من الاشخاص الاصحاء. تم اخذ خمسين مريضا تتراوح اعمارهم ما بين 20 –85 سنة بواقع 23 من الذكور و27) من الاناث، كما تضمنت الدراسة 11 شخصا بالغا سليما من اي امراض كمجموعة سيطرة، وشملت الدراسة قياس المتغيرات الكيموحيوية التالية : سكر الدم ،مرتسم الدهون بانواعها الخمسة (الكوليستيرول الكلي، الدهون الثلاثية، البروتينات الدهنية عالية الكثافة، البروتينات الدهنية واطئة الكثافة والبروتينات الدهنية واطئة الكثافة جدا) ، الكرياتينين واليوريا باستخدام جهاز الكيمياء الحياتية الآلي المجهز من شركة أبوت الأمريكية. أظهرت نتائج الدراسة أن مستويات الكرياتينين واليوريا في المرضى بCKD ارتفعت بشكل معنوي مقارنة بالأصحاء ، بينما انخفض مستوى HDL–C بشكل ملحوظ ، في حين أظهرت (TC و LDL–C انخفاضًا طفيفًا بالرغم من ان مستوياتهما كانت ضمن الحدود الطبيعية مع مجموعة السيطرة، بينما كانت مستويات TG و (VLDL) وسكر الدم غير معنوية فيمرضى CKD مقارنة مع مجموعة الاصحاء ($P \leq 0.05$). بالإضافة إلى ذلك ، أظهرت جميع الاختبارات الكيموحيوية عدم وجود فروقات معنوية في الذكور مقارنة بمجموعة الإناث ($P \leq 0.05$), اما اعتمادًا على العمر ، أظهر الكرياتينين تغيرًا كبيرًا في المجموعة 20– 40 سنة مقارنة بالمجموعة61– 85 سنة عند مستوى احتمالية ($P \leq 0.05$).

1. INTRODUCTION

Chronic kidney disease (CKD) is an increasing public health issue with high incidence of morbidity and mortality that occur commonly throughout a worldwide [1]. CKD may be related with the progression of cardiovascular disease which is the important cause of morbidity and mortality in CKD patients [2] and constitutes greater burden of cost of care especially in developing countries [3]**.** According to the Kidney Disease Outcomes Quality Initiative (K/DOQI) CKD is defined as renal damage or when glomerular filtration rate (GFR) falls below 60 ml/min/1.73m^2 that lasts longer than three months [4]**.** CKD is split into five degrees depending on GFR level [5]. It has been observed that 10-15% of people in the world have CKD that increases with aging [6]**.** The findings of the Third National Health and Nutrition Examination Survey in the USA pointed out that nearly 8.3 million of adults have advanced CKD stages (3–5) [7] and considered it among the twelves common diseases causing prevalence death around the world which is estimated to be 1.1 million deaths per year [8]. Kidneys normally play an important role in the metabolism [9] and if damaged, it can lead to heart attack, stroke and other dangerous situations [10]. This disease also known as a chronic renal failure, occurs mostly in those patients who have a history of hypertension

and diabetes. CKD is a state that causes damage to the kidney or its slow injury relating to the loss of glomerular and tubular function for few months or a year. In the onset period of CKD, the persons do not show any signs and symptoms of the disease. Only after months or year a patient starts showing symptoms like nausea, vomiting, lethargy, edema, confusion and loss of appetite [10]**.**

Lipids are biochemical molecules that in addition to their active functions, are essential in cellular membrane function and produce bile acids, vitamin (D) and steroids hormones. They are transferred by hydro-soluble particles (lipoproteins) that are comprised of esterified cholesterol and nonpolar core of triglycerides, enclosed by apolipoproteins, phospholipids and others polar lipids [11][12]**.** Dyslipidemia is a disturbance in metabolism of lipoprotein, including lipoprotein overproduction or deficiency. Dyslipidemia is characterized by elevation of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) , triglycerides (TG) and decrease of high-density lipoprotein cholesterol (HDL-C) in the blood [13]. Dyslipidemia has been hypothesized to cause renal injury and its development in the early stages of the disease so that play main role in the progression of kidney diseases [14][15]**.** These abnormalities show diversities relying on the level of renal deficiency, etiology of essential disease, existence of nephrotic syndrome (NS) and process of dialysis for subjects undergoing renal replacement therapy [16]**.** Recent KDOQI recommendations suggest that lipid disturbances (TC, LDL-C , HDL-C, and triglycerides) must be estimated in subjects with CKD which was the focus of the current study [17]**.**

2. Materials and Methods

2. 1 Study Population

This was a case-control observational study conducted in kidney dialysis unit at Al-Ramadi Teaching Hospital in Anbar province in Iraq. Fifty patients (23 males and 27 females) with ages between 20-85 years and in different stages of CKD were selected. They received regular treatment at least three times per week at the hemodialysis units. A total of 11 healthy volunteers (8 males and 3 females between 21-65 years ages were taken as control.

2.2 Sample Collection and Analysis

Blood samples were collected after an overnight fasting. Five ml of venous blood samples were collected into disposable plain plastic tubes and then were allowed to coagulate at room temperature to be centrifuged at 3000 rpm for 15 min. Serum was separated and stored in a plain tubes at -20ºC and then used for measuring creatinine, urea, glucose, TC, TG, HDL, LDL and VLDL that were estimated by enzymatic colorimetric method by using multi chemical fully automated chemistry analyzer (Abbott/USA) at the Department of Biochemistry.

2.3 Statistical Analysis

The data was expressed as the mean ± standard deviation. Statistical software (SPSS, version 22.0) was used for data analysis. Comparison of means values was performed by using Student's "t" test. The comparison among three groups of age was performed using One Way ANOVA and then by using "LSD" test. Differences were considered to be significant if the probability was less than $P \leq 0.05$.

3. Results

A total of 61 participated were included in this work, among them were n=50 CKD patients with mean age 53.54±17.33 years, 23 (46%) were men with mean age 54.26 ±15.48 years and 27 (54%) were women with mean age 52.93±19.03 years. Eleven were healthy subjects with mean age 37.00±15.56 years as control. The characteristics of all patients are summarized in Table 1.

Table 1: Characteristics of the study patients with chronic kidney disease

Age (Years)	Males	Females	Total (%)
20-40	5	8	13 (26)
41-60	9	7	16 (32)
61-85	9	12	21 (42)
Total	23	27	50 (100)

The results in this study indicated that the urea (123.52±30.18 mg/dl) and creatinine (7.57±2.96 mg/dl) levels were highly significant in CKD patients compared with healthy subjects (27.60±4.17 mg/dl), (0.74±0.24 mg/dl) respectively. Whereas HDL (35.38±11.43 mg/dl) level reduced significantly in CKD) patients compared with control group (43.55±6.42 mg/dl). TC and LDL showed slightly lower significant levels as compared to the control group. The study also revealed no significant differences in patients group of TG, VLDL and glucose levels compared with control group ($p \leq 0.05$) (Table 2).

Table 2: Comparison of glucose, lipid profile, urea and creatinine between chronic kidney disease patients and the control group

Parameters	Groups	N	Mean ± SD	Sig. (2- tailed)
Glucose mg/dl	patients	50	135.84±113.79	0.192
	control	11	90.27±8.70	
T.C mg/dl	patients	50	157.38±45.56	0.004
	control	11	202.82±48.21	
TG mg/dl	patients	50	142.64±90.92	0.457
	control	11	167.00±125.63	
HDL mg/dl	patients	50	35.38±11.43	0.026
	control	11	43.55±6.42	
LDL mg/dl	patients	50	92.93±35.43	0.011
	control	11	125.87±47.53	
VLDL mg/dl	patients	50	28.71±18.06	0.472
	control	11	33.40±25.13	
Urea mg/dl	patients	50	123.52±30.18	0.000
	control	10	27.60±4.17	
Creatinine mg/dl	patients	50	7.57±2.96	0.000
	control	11	0.74±0.24	

(*$P < 0.05$, statistically significant)

Abbreviations: HDL, high-density lipoprotein cholesterol; LDL, low density lipoprotein cholesterol; VLDL, very-low-density lipoproteins; T.C, total cholesterol; TG, triglyceride.

The results in Table 3, depending on sex differentials of CKD patients, revealed that means values of urea, creatinine, glucose, TC, TG, HDL, LDL and VLDL in males group were non-significant differences as compared with females group ($p \leq 0.05$).

Table 3: Comparison of glucose, lipid profile, urea and creatinine in chronic kidney disease patients according to gender

Parameters	Groups	N	Mean ± SD	Sig. (2-tailed)
Glucose mg/dl	M	23	122.61±66.53	.454
	F	27	147.11±142.72	
T.C mg/dl	M	23	155.43±41.80	.784
	F	27	159.04±49.27	
TG mg/dl	M	23	144.74±112.34	.882
	F	27	140.85±69.95	
HDL mg/dl	M	23	36.74±11.00	.444
	F	27	34.22±11.87	
LDL mg/dl	M	23	89.75±33.07	.563
	F	27	95.64±37.74	
VLDL mg/dl	M	23	29.34±22.24	.822
	F	27	28.17±13.99	
Urea mg/dl	M	23	121.70±30.81	.697
	F	27	125.07±30.13	
Creatinine mg/dl	M	23	7.97±2.87	.380
	F	27	7.23±3.05	

(*P < 0.05, statistically significant).

Abbreviations: HDL, high-density lipoprotein cholesterol; LDL, low density lipoprotein cholesterol; VLDL, very-low-density lipoproteins; T.C, total cholesterol; TG, triglyceride.

The comparison of the results based on age in CKD patients showed that there were not any significant differences in three categories of age in all parameters, except creatinine level in 20-40 years age patients which was a significant difference (9.14±3.22 mg/dl) compared with 61-85 years age group (6.51 ± 2.28mg/dl) ($p \leq 0.05$) (Table 4).

Table 4: Comparison of glucose, lipid profile, urea and creatinine in chronic kidney disease patients according to age groups

Parameters	Group	Mean ± SD	Groups	Mean Difference (I-J)	Sig.
Glucose mg/dl	20-40	157.62 ± 195.31	41-60	29.8654	.492
			61-85	29.0916	.479
	41-60	127.75±70.32	61-85	-.7738	.984
TC mg/dl	20-40	140.62±29.13	41-60	-27.3221	.112
			61-85	-19.0989	.238
	41-60	167.94±42.29	61-85	8.2232	.586
TG mg/dl	20-40	107.69±63.70	41-60	-57.6827	.093
			61-85	-39.2601	.222
	41-60	165.38±95.95	61-85	18.4226	.540
HDL mg/dl	20-40	39.00±15.50	41-60	4.0000	.354
			61-85	5.5714	.174
	41-60	35.00±10.28	61-85	1.5714	.681
LDL mg/dl	20-40	78.00±20.08	41-60	-21.8625	.101
			61-85	-18.8952	.133
	41-60	99.86±32.84	61-85	2.9673	.799
VLDL mg/dl	20-40	22.23±12.43	41-60	-10.8442	.112
			61-85	-7.1597	.264
	41-60	33.08±19.19	61-85	3.6845	.539
Urea mg/dl	20-40	134.23±28.50	41-60	6.6058	.551
			61-85	20.4689	.055
	41-60	127.63±13.71	61-85	13.8631	.163
Creatinine mg/dl	20-40	9.14±3.22	41-60	1.45010	.174
			61-85	2.63813*	.011
	41-60	7.69±3.10	61-85	1.18804	.210

(*P < 0.05, statistically significant). (Age: 20-40, n=13) (Age: 41-60, n=16) (Age: 61-85, n=21).

Abbreviations: HDL, high-density lipoprotein cholesterol; LDL, low density lipoprotein cholesterol; VLDL, very-low-density lipoproteins; T.C, total cholesterol; TG, triglyceride.

4. Discussion

Kidney failure occurs when the kidneys are unable to eliminate or perform their regulatory role of body metabolic waste [18]. Advanced chronic kidney disease can cause a decline in urinary excretion which leads to retention of metabolites e.g., electrolytes , urea, creatinine and water in human body [19].

In the current study, the mean of the urea and creatinine levels showed highly significant increase in patients with CKD compared with healthy individuals ($p \leq 0.05$) (Table 2). These findings agree with several previous studies [20] [21][18]. This increase in urea and creatinine levels occurs because of reduction in the number of nephrons in patients with renal failure which causes decrease in the GFR that is responsible for a significantly low level in water and solutes**.** Due to this the kidney loses its efficiency to remove blood nitrogenous wastes which causes accumulation of these components in the bloodstream [18][22]**.** In the urea cycle, the urea produced by transformed ammonia, is then conveyed by means of blood to the kidneys for its removal outside the body [20]. The high levels of urea in the blood are mainly caused by the development of the disease, but is heavily influenced either by a catabolic condition or by excessive protein intake which leads to increased production of protein catabolism waste [18].
However, there was no significant difference in the mean values of urea and creatinine levels between male and female groups ($p \leq 0.05$) (Table 3). On other hand, regarding age, there no significant difference in the mean values of urea was detected, whereas the mean values of creatinine significantly ($p \leq 0.05$) increased in 20-40 years age group as compared with 61-85 years age group (Table 4). Our data was in accordance with a study done by Paudel *et al.*[23] which reported that the creatinine was relatively higher in younger but when compared with another age groups in their study no significant differences were found [24]. As mentioned previously in cross-sectional studies that starting at 40 years of age the creatinine clearance declined by 0.87 cc/min/year. Interestingly, this hypothesis does not apply to everyone [25]. The glucose levels were non-significant statistically according to the gender and age of CKD patients, likewise, patients with CKD compared with healthy subjects ($p \leq 0.05$) as most of patients in the current study were without diabetes mellitus [22] (Tables 2, 3, 4)**.**

Results of this study revealed that there was a significant ($p \leq 0.05$) decrease in the mean values of HDL-C level in patients with CKD as compared with healthy subjects (Table 2). Our findings were similar to previous study done by Kumar [12]. HDL-C stimulates on a reverse cholesterol transport from circulating macrophages, feature that is well known to be atheroprotective [26]. Patients with CKD have reduced HDL-C levels which may be due to several mechanisms. Patients with CKD usually exhibit decreased levels of apolipoproteins AI and AII, diminished activity of LCAT, also high activity of protein (CETP) which facilitates the convey of cholesterol esters from HDL to triglyceride-rich lipoproteins thus reducing of HDL-C [16]**.** Hemodialysis and peritoneal dialysis procedure may also have a contributory role in the reduced HDLC [16]**.** The HD-procedure includes factors that may influence the lipoprotein metabolism [27]**.** HDL-C decreases because of loss molecules for maturation of HDL, rise of monocyte count which may cause foamy cells, also causing proinflammatory effects that lead to progression of (CKD)[28] **.**

In addition, T.C and LDL-C levels were slightly significant lower in CKD patients, whereas TG and VLDL levels were statistically insignificant ($p \leq 0.05$) as compared with healthy subjects (Table 2) .The current results are partially similar to some previous studies [29] [30]. It is believed that malnutrition is an agent participating into both the decreased levels of lipids and increased mortality in patients with dialysis [22]. Another study reported that serum TC and LDL-C levels are commonly in normal range or lower in subjects who had end-stage renal disease (ESRD) and continued on hemodialysis [31]**.** TG in the serum is one

of the most lipid kinds changing in renal diseases. The most dramatic increase occurs in patients with nephrotic syndrome and other renal diseases because of both unusual production and lower catabolism of TG [28]. The CRF induced hypertriglyceridemia, unnatural composition and decline clearance of triglyceride rich lipoproteins and their remnants are generally because of downregulation of lipoprotein lipase, hepatic lipase, and the VLDL receptor, also upregulation of hepatic acyl-CoA cholesterol acyltransferase (ACAT) [32] **.** On other hand, TC, TG, HDL-C, LDL-C and VLDL levels in males patients with CKD showed non-significant changes as compared to females group ($p \leq 0.05$) (Table 3). Also no significant differences in three groups of age were found (Table 4)**.** The results agree with study done by Giandalia *et al.*[33] which found no significant differences in males and females in the lipid profile except TG. Whereas another study found that the percentage of TC, LDL-C, TG and low HDL-C in CKD patients consistently non-statistically increases with age [17].

5. Conclusion

This study was done to identify the lipid abnormalities and renal function tests in patients with CKD. Our data demonstrated that the creatinine and urea highly significantly increased, while HDL significantly decreased in CKD patients. While other biomarkers were either within normal levels or slightly changed compared with control. Lipids abnormalities in serum were probably independent risk factors for progression of renal disease.

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