Original Article

Correlation of Interleukin-17A and Beta-2-Microglobulin in Early Diagnosis of Patients with Hypertension Related Chronic Kidney Disease

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Abstract

Background: Interleukin-17A (IL-17A) plays a harmful role in the inflammation associated with hypertensive renal disease, investigation conducted to discover therapeutic strategies that specifically target this cytokine to mitigate the risk of kidney impairment generated by hypertension. **Objective:** To evaluate the role of IL-17A and β2-MG, as markers of inflammation in hypertensive CKD patients and assess possible usage of these markers as a predictive index for CKD occurrence. **Materials and Methods:** The total number of participants are 100 participants including 35 CKD patients with hypertension, 35 CKD patients without hypertension, and 30 controls without hypertension or CKD, 48 individuals were male and 52 individuals were female, their ages ranged from 20 to 70, all study participants were evaluated with respect to serum IL-17A and β2-MG levels by enzyme-linked immune sorbent assay test. **Results:** The results showed a high significant differences in concentrations of IL-17A and β2-MG in study patient (CKD patients with hypertension, and CKD patients without hypertension) in comparison with the control group. **Conclusion:** Serum IL-17A and β2-MG can be used as markers of inflammation and predicators for early onset of CKD in hypertensive patient more accurately than routine diagnostic tests.

Keywords: Beta-2-microglobulin, chronic kidney disease, hypertension, interleukin-17A

Introduction

Hypertension is a prevalent chronic medical disorder defined by the sustained rise of arterial pressure,[1] which is the primary variables associated with kidney disease, and it is widely regarded as a prominent "silent killer" disease that contributes to premature mortality worldwide.[2]

According to recent studies, hypertension is the single most common risk factor for ill health, contributing to a higher worldwide disability load. Many patients with hypertension may be unaware of it early on, because it is rarely accompanied by symptoms and is usually detected through screening or when seeking health care for an unrelated condition. Symptoms such as headache, vertigo, dizziness, and palpitation only appear when blood pressure is extremely high.[3]

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Pathophysiological hallmarks of the condition are age-related vascular dilatation and stiffness in elastic arteries, which manifests as vasomotor dysfunction. Other factors that contribute to the development of hypertension include renal aging, neurohormonal dysfunction, and autonomic dysregulation.[4]

The risk factors for disease do not exist on their own; rather, they are the outcome of numerous factors coming together. When several factors contribute to a result,

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the influence of a single element may be increased or decreased due to the interaction of other factors.[5]

Through both blood pressure-dependent and blood pressure-independent processes, obesity, and a high consumption of salt have been shown to contribute to the acceleration of chronic kidney disease (CKD).[2]

CKD represents a worldwide public health problem, with a global prevalence of 9.1%. It is defined as abnormalities of kidney structure or function present for more than 3 months, with implications for health. CKD increases the risk of all-cause mortality, cardiovascular disease, and progression to end-stage renal disease (ESRD). Thus, identifying the risk factors for CKD or a decrease in estimated glomerular filtration rate (eGFR) may help in understanding the mechanism of CKD and provide new strategies for its prevention.[6]

Interleukin-17 (IL-17) is thought to be an essential connecting molecule between the adaptive and innate immune systems because it is noticeably a pro-inflammatory cytokine which cause release of other pro-inflammatory cytokines such IL-6 and IL-8, matrix metalloproteinase, and granulocyte colony-stimulating factor.[7]

Interleukin-17A (IL-17A) is primarily produced by T helper 17 (Th17) cells, innate lymphoid cells, natural killer cells, and other innate and adaptive immune cell. IL-17A raises blood pressure through inhibiting the production of endothelial nitric oxide, The augmentation of reactive oxygen species generation is seen to facilitate the progression of vascular fibrosis, enhancing renal salt retention, and causing glomerular injury.[8]

Beta-2-microglobulin (ß2-MG) is a polypeptide which is a part of the major histocompatibility complex (MHC) class I, may function as a local and systemic inflammatory signal. Endothelial damage and a persistent inflammatory response were caused by the elevated level of ß2-MG. Plasma ß2-MG levels rose with CKD stage and were therefore highest in hypertensive individuals.[9]

Materials and Methods

Patients

The case-control study has been conducted at Al-Imam Al-Sadiq Teaching Hospital in Babylon Governorate, spanning from January 2023 to April 2023. The study population consisted of seventy individuals diagnosed with CKD, with 35 of them presenting with CKD and Hypertension, while the remaining 35 had CKD without Hypertension. Additionally, a control group of 30 controls was included for comparison. The ages of the individuals ranged from 20 to 70 years. 48 of study participants were males and the other 52 were females.

Methods

Data collection and blood sampling

Five mL of venous blood was drawn in an aseptic condition using a plastic syringe and the standard safety procedures used during venipunctures. Blood were taken in a gel tube and left for 30 min to clot, then centrifugations were carried out for 20 min at 1500 rpm, and serum was separated for analyzing parameters.

Serum IL-17A and ß2-MG levels were measured by using ELISA kits from Elyue® catalogs number: FY-EH6678 for IL-17A and FY-EH5209 for ß2-MG and measurement was done according to the manufacturer's recommendations.

Exclusion criteria

Patients were excluded from the study if they had any of the following: hepatitis, autoimmune disease, pregnant women, elevated blood pressure due to cancer, diabetic patients, kidney stones disease, and other types of renal diseases, except CKD with hypertension.

Statistical analysis

Independent *t* Test and Mann–Whitney *U* tests have been utilized to conduct a comparative analysis between two groups on the same continuous variable and the results have been considered to have statistical significance at ($P \leq 0.05$).

Ethical approval

This study was approved by the Ethical Committee at College of Applied Medical Science/University of Kerbala, Kerbala, Iraq. All subjects involved in this work were informed and agreement was obtained verbally from each participant before the collection of samples. The study protocol and the subject information and consent form were reviewed and approved by a local ethics committee according to the document number 3 (including the number and the date on January 1, 2023) to get this approval.

Results

The demographic characters of all study groups

The current study involved 48 males and 52 females in regard to sex, whereas it involved 36 participants in age group within 20–40 and 64 participants in age group within 41–70, while the participants distribution according to BMI was divided into 20 of them were was normal weight, 41 of them were Overweight and 39 of them were severely obese. As presented in Table 1.

Interleukin -17 A(IL-17A)

According to the findings of Table 2 CKD patients with hypertension had higher concentrations of IL-17A than CKD patients without hypertension, while the lowest concentration was in control group. In the same table, it is also indicated significant differences ($P \leq 0.05$) in both patient groups compared with control.

According to the findings of Table 3, when CKD patients with hypertension in comparison with CKD patients without hypertension indicated to IL-17A concentrations was higher in CKD with HBP group more than CKD without HBP group but there were nonsignificant differences between them.

Beta-2- microglubolin (β2-MG)

According to the findings of Table 4, CKD patients with hypertension had higher concentrations of β2-MG than CKD patients without hypertension, while the lowest concentration was in control group. In the same table, it is also indicated significant differences ($P \leq 0.05$) in both patient groups compared with control.

According to the findings of Table 5, when CKD patients with hypertension in comparison with CKD patients without hypertension indicated to ß2-MG concentrations was not extremely higher in CKD with HBP group than CKD without HBP group also there was nonsignificant differences between them.

Urea and creatinine

Results of Table 6 demonstrated that there were significant changes ($P \leq 0.05$) in the Urea and Creatinine concentration in both patient group when compared to the control group. The Urea and Creatinine concentration highly increased in CKD patients with hypertension than

Table 1: Distribution and characteristics of patients and control according to the study subjects

Variable	Level	CKD with HBP		CKD without HBP		Control		Total
		No.	Percentage (%)	No.	Percentage (%)	No.	Percentage (%)	
Sex	Male	17	48.6%	16	45.7%	15	50.0%	48
	Female	18	51.4%	19	54.3%	15	50.0%	52
Age group	20–40	12	34.3%	14	40.0%	10	33.3%	36
	41–70	23	65.7%	21	60.0%	20	66.7%	64
BMI groups	Normal weight	3	8.6%	8	22.9%	9	30.0%	20
	Over weight	14	40.0%	16	45.7%	11	36.7%	41
	Severely obese	18	51.4%	11	31.4%	10	33.3%	39

Table 2: IL-17A mean levels among study groups according to age group and sex

Disease status	Variable	Variable level	Patients		Control		*P* value
			Mean	Std. deviation	Mean	Std. deviation	
CKD with HBP	Case	Case/control	27.61	9.31	5.58	2.01	0.004*
	Sex	Male	29.16	10.43	5.42	1.66	0.001*
		Female	26.14	8.15	5.75	2.36	0.001*
	Age group	20–40	28.63	10.03	4.99	1.77	0.006*
		41–70	27.08	9.10	5.88	2.10	0.008*
CKD without HBP	Case	Case/control	22.80	9.35	5.58	2.01	0.007*
	Sex	Male	24.22	10.21	5.42	1.66	0.002*
		Female	21.60	8.65	5.75	2.36	0.007*
	Age group	20–40	22.86	8.79	4.99	1.77	0.003*
		41–70	22.76	9.92	5.88	2.10	0.002*

NS: no significance

*Means significant differences at $P \leq 0.05$

Table 3: Comparison of biomarker levels (IL-17A) among patients, stratified by gender and age group

Disease status	Variable	Variable level	Mean	Std. deviation	*P* value
CKD with HBP	Gender	Male	29.16	10.43	0.621
		Female	26.14	8.15	
	Age group	20–40	28.63	10.03	0.602
		41–70	27.08	9.10	
CKD without HBP	Gender	Male	24.22	10.21	0.417
		Female	21.60	8.65	
	Age group	20–40	22.86	8.79	0.975
		41–70	22.76	9.92	

*The mean difference is significant at the 0.05 level

Table 4: Beta 2 Micro globulin mean levels among study groups according to age group and sex

Disease status	Variable	Variable level	Patients		Control		*P* value
			Mean	Std. deviation	Mean	Std. deviation	
CKD with HBP	Case	Case/control	6.45	2.00	2.39	0.74	0.002*
	Sex	Male	7.00	1.69	2.31	0.65	0.006*
		Female	5.92	2.18	2.48	0.83	0.002*
	Age group	20–40	5.96	2.37	2.25	0.47	0.002*
		41–70	6.70	1.79	2.47	0.84	0.001*
CKD without HBP	Case	Case/control	5.98	2.38	2.39	0.74	0.001*
	Sex	Male	5.73	2.24	2.31	0.65	0.002*
		Female	6.19	2.54	2.48	0.83	0.004*
	Age group	20–40	6.21	2.66	2.25	0.47	0.002*
		41–70	5.82	2.23	2.47	0.84	0.006*

NS: no significance

*Means significant differences at $P \leq 0.05$

Table 5: Comparison of biomarker levels (ß2-MG) among patients, stratified by gender and age group

Disease status	Variable	Variable level	Mean	Std. deviation	*P* value
CKD with HBP	Gender	Male	7.00	1.69	0.113
		Female	5.92	2.18	
	Age group	20–40	5.96	2.37	0.306
		41–70	6.70	1.79	
CKD without HBP	Gender	Male	5.73	2.24	0.703
		Female	6.19	2.54	
	Age group	20–40	6.21	2.66	0.661
		41–70	5.82	2.23	

*The mean difference is significant at the 0.05 level

Table 6: Distribution of urea and creatinine levels among study groups according to age group and sex

Disease status	Parameters	Variable level	Patients		Control		*P* value
			Mean	Std. deviation	Mean	Std. deviation	
CKD with HBP	Urea	Case/control	22.82	10.99	4.62	1.40	0.005*
		Male	25.86	11.83	4.77	1.24	0.002*
		Female	19.95	9.59	4.47	1.58	0.002*
		20–40	23.43	10.98	4.26	1.16	0.007*
		41–70	22.51	11.23	4.80	1.55	0.001*
	Creatinine	Case/control	216.05	114.93	65.82	11.51	0.005*
		Male	247.79	125.69	66.73	13.28	0.002*
		Female	186.08	97.93	64.90	9.81	0.006*
		21–40	235.49	133.58	66.97	10.55	0.001*
		42–70	205.91	105.73	65.24	12.18	0.002*
CKD without HBP	Urea	Case/control	19.64	10.17	4.62	1.40	0.003*
		Male	21.07	11.89	4.77	1.24	0.006*
		Female	18.43	8.61	4.47	1.58	0.001*
		20–40	20.58	11.16	4.26	1.16	0.002*
		41–70	19.01	9.68	4.80	1.55	0.001*
	Creatinine	Case/control	213.46	99.64	65.82	11.51	0.003*
		Male	221.14	126.44	66.73	13.28	0.005*
		Female	206.99	73.03	64.90	9.81	0.009*
		20–40	216.69	96.68	66.97	10.55	0.005*
		41–70	211.31	103.87	65.24	12.18	0.003*

NS: no significance

*Means significant differences at $P \leq 0.05$

CKD patients without hypertension when compared to the control group.

Discussion

The data in Table 1, demonstrates the distribution and characteristics of patients and control according to sex, age, and BMI in this study.

A study showed higher hazard ratios for CKD among males, younger than 40 years, compared to those among females, younger than 40 years.[10]

Another study was conducted with an average age of 75 years old among those with stage three CKD demonstrated that there was strong relationship between increased age and high blood pressure in elderly patients with CKD.[11]

While study showed a significant relationship between CKD and hypertension ($P = 0.01$) in obesity.[12]

The role of the pro-inflammatory cytokine IL-17A has been investigated in the pathogenesis of hypertension. The current data in Table 2, demonstrates a high statistically significant in serum IL-17A levels in all groups of CKD patients, both with and without hypertension, as compared to the control group.

IL-17A is important in human hypertension and vascular disease; it can be used as a predictive index for early diagnosis of CKD occurrence and follow-up in those patients.

A study found that IL-17A contributes to further increases in blood pressure and end-organ damage via effects on vascular and renal cells. The increase in Th17-like cells and IL-17A production were observed in experimental animal models and humans with hypertension.[8] Also, another study found that IL-17A is closely correlated with the progression of renal damage these results of study agree with present study.[13]

While a study shows the systemic IL-17A infusion in mice increased systolic blood pressure over control values $P < 0.05$. However, IL-17A infusion did not modify renal function as assessed by serum urea and creatinine. These data suggest no significant impact of IL-17A infusion on parameters of kidney function or injury that are commonly used in the clinic.[14] which was agreed with the result in Table 3 when compared CKD patients, both with and without hypertension without control group.

The result of Table 3 shows no significant impact of IL-17A on comparison groups, which may be related to numerous factors, for example, the total number of participants in this study should be increased to make higher comparison or in collection of samples the CKD with HBP group had drug that effect on results.

A study found that β2-MG level contributes to the process of atherosclerosis formation, chronic inflammation process, and endothelial injury. Further mechanisms include macrophage and lymphocyte recruitment, which accelerate the atherosclerosis plaque formation and blood vessel rigidity. which found a significant difference in β2-MG level based on CKD stages;[9] which agree with the present study findings that shows the concentration of *β*2-MG in Table 4 in CKD patients with hypertension was significantly higher more than CKD patients without hypertension compared to control groups in this work.

Also, this study findings corroborate with another study that showed the β2-MG in the hypertensive nephropathy group were higher than those in the control group and the simple hypertension group ($P < 0.05$).[15]

Other studies also observed a significant correlation between B2M and eGFR, even when renal function was only slightly impaired.[16,17]

Whereas the result of Table 5 shows no significant impact of β2-MG on comparison groups, which may be related to numerous factors, such as in collection of samples the CKD without HBP group had drug that irritate β2-MG level.

According to the most recent findings in Table 6, had considerably higher urea and creatinine in both patient groups concentrations than the control group. The present investigation observed a noteworthy elevation in the levels of urea and creatinine in the sera of patients with CKD, both with and without hypertension, in comparison to the control group. This finding aligns with research that found higher treated BP was associated with early kidney function decline (defined as a rise in serum creatinine ≥ 0.6 mg/dL).[18]

A study observed the levels of the routine chemistries (creatinine and urea) were consistent with the expected pattern in CKD and were significantly different from those of the control group.[19] This is in agreement with the findings of this study.

Also, the present study's findings align with another study, which also examined renal function tests. The results showed statistically significant differences ($P < 0.01$) in serum urea, creatinine, and glomerular filtration rate (GFR) across all groups studied.[20]

Conclusions

The study concludes that serum IL-17A and *β*2-MG levels is significantly higher in both CKD patients with hypertension and CKD patients without hypertension which indicated that can be used as markers of inflammation and predicators for early onset of CKD in hypertensive patient more accurately than routine diagnostic tests (urea and creatinine).

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Conflicts of interest
There are no conflicts of interest.

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