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Diagnostic potential of soluble TNF alpha receptor 1 in diabetic and hypertensive patients with renal impairment and UTI.

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ABSTRACT

Background: Chronic kidney disease is one of the most common diseases. Health care workers in all countries of the world are concerned with the early detection and prevention of kidney diseases. Several novel diagnostic markers are being under investigation nowadays. Tumor necrosis factor-alpha and its receptors are examples.
Aim: The present study was conducted to evaluate the role of tumor necrosis factor α receptor 1 (TNFR1) as a biomarker for detection of renal dysfunction.
Materials and Methods: The study was carried out for the period from February to June 2019 and included 180 patients (their ages were between 19 and 85 years old) and were divided into 60 patients with renal impairment, 60 hemodialysis patients, and 60 patients with normal renal function (as a control group). Each group included patients with hypertension, patients with diabetes mellitus, and hypertensive- diabetic patients.
The patients were attended to Center of Kidney Disease and Transplantation, Dialysis Unit of Baghdad Teaching Hospital – Medical City , Dialysis Unit of Tikrit Teaching Hospital and private laboratory in Samarra City.
Urine sample was collected from each patient for bacteriological study and detection the level of TNFR1.
Results: The most common pathogen isolated from cultured samples was *Escherichia coli.* Concentration of urinary TNFR1 in hypertensive and or diabetic with normal kidney function compared with hypertensive or and diabetic renal impairment did not differ statistically significant.
Conclusion: Urinary level of tumor necrosis factor receptor 1 (TNFR1) is not important in the diagnosis of renal impairment with the presence of hypertension and or diabetes mellitus. Through statistical comparisons of patients with urinary tract infection (UTI) group and those without UTI group , it seems that UTI does not affect the diagnostic ability of urinary TNFR1. We recommend future studies focusing on serum level of the receptors mentioned above to test their diagnostic potential in renal impairment. In addition, investigating the effect of the immunological causes of renal impairment on the level of TNFR, both in urine and serum.

Introduction

Chronic kidney disease is a change in kidney structure and function resulting to a progressively and perpetual leakage of renal function[1]. The most common reasons of CKD are diabetes and hypertension in addition to urinary tract infection, autoimmune disease and others[2]. Diagnostic markers of renal function test can be components of serum or urine. Urine biomarkers serve to detect early renal impairment, so it could be used for early diagnosis, identification of mechanism disorders and

severity of dysfunction[3]. Novel diagnostic markers are being investigated including Tumor Necrosis Factor Alpha (TNF α) and its receptors, Cystatin C[4], β-Trace Protein[5], β2-Microglobulin [6], iohexol and inulin[7], Arginase and Carbonic Anhydrase. [8], α1-microglobulin [9] , retinol binding protein [10], neutrophil gelatinase-associated lipocalin [11], netrin-1 [12], fibroblast growth factor 23 [13], kidney injury molecule-1 [14] etc…

Tumor necrosis factor (TNF)α is one of an important proinflammatory cytokine and essential factor of inflammatory tissue injury. In addition,'it has important immune regulatory functions. Most researchers reported a role of TNF in acute and chronic renal disease pathogenesis. Thus, after renal injury the early proinflammatory mediator is TNF-α, which release by dendritic cells (DCs) in the renal interstitium [15]. Production of tumor necrosis factor in the kidney may be increased by infiltrating immune cells, essentially macrophages [16]. Renal hemodynamics and nephron transport can be alter by TNF-α and changing on activity and expression of transporters. It stimulates immune cell infiltration and cell death which lead to organ damage [17]. In chronic kidney disease (CKD) TNF-α is increased, which is characterized by deterioration of renal function, renal damage, and hypertension [18]. Tumor necrosis factor receptor 1 or p55 and TNFR2 or p75 mediate the actions of TNF-α. Thus, two distinct actions of TNF-α signaling in the kidney. Tumor necrosis factor receptor 1 acts on protective functions in the kidney by decreasing hyperfiltration but enhancing natriuresis, and thus regulating blood pressure. TNFR2 enhance renal tissue damage by proinflammatory pathways [19]. Tumor necrosis factor receptor 1 and TNFR2 are expressed in the collecting ducts, proximal tubules and endothelial cells of the renal vasculature of the kidney, but only TNFR1 is present in the smooth muscle cells of the renal vasculature [20]. Tumor necrosis factor receptor 1 can be found in the proximal tubule, collecting duct, vascular endothelium, and vascular smooth muscle of the kidney. Renal hemodynamic and excretory function mostly related to TNFR1 activity. Reduces GFR and renal blood flow, as well as promotes natriuresis and dieresis mediate by TNFR1 activation [21]. Moreover, results from the Joslin Kidney study concluded that elevated concentration of circulating TNFR1 and TNFR2 showed a very strong predictors of the development of diabetic nephropathy to chronic kidney disease-stage 3 or end stage renal disease [22]. These bio-markers could prove very useful in terms of early detection and prognosis in CKD. Recently, serum TNFR1 and TNFR2 have been associated to progression of renal function [23].

Materials and Methods

A cross-sectional study from February to June 2019. Blood and urine samples were collected from 180 patients (age 19 – 85 years old) at center of kidney disease and transplantation, hemodialysis unit of Baghdad teaching hospital–Medical City, hemodialysis unit of Tikrit Teaching Hospital and private laboratory in Samarra City. After taking informed consent, patients were divided into: 60 renal failure patients under hemodialysis divided into hypertensive patients, diabetic patients and hypertensive - diabetic patients. Another group of 60 renal impairment patients which divided into hypertensive patients, diabetic patients and hypertensive - diabetic patients. The third group 60 controls with normal kidney function patients who were divided into hypertensive patients, diabetic patients and hypertensive - diabetic patients. Patients with autoimmune disease(s) were excluded. Data were collected by direct interview using a questionnaire designed for this study. These information included name, age, gender, medical history, duration of disease, control of disease, complication of disease, and ask patient about UTI symptoms. The samples which were collected from the patients included urine and blood. Urine sample was collected for macroscopic, bacteriological and immunological examination. A midstream urine (MSU) sample was collected (30 ml of urine) in sterile cap and transported to the laboratory within 30 minutes for chemical and bacteriological study and preparing urine to immunological study by centrifuge and the supernatant was transferred to Eppendorf tubes, labeled and kept frozen at 20^0C. Blood was withdrawn from the veins and transferred to sterile gel & clot activator tube, The blood in gel & clot activator tube was centrifuged and only the clear serum was used. For quantitative measurement of urinary TNFR1, a kit used enzyme-linked immune sorbent assay (ELISA) based on biotin double antibody sandwich technology. Biochemical test for serum creatinine was determined by using Autoanalyser Mindray BS-200 .

Statistical analysis: Was done by using SPSS version 24, namely Man Whitney test and student t-test for two-mean comparison. Analysis of variance was used to compare more than two means. Finding of *P value* < 0.05 was regarded significant.

Results

Urine culture was done for all urine samples of 180 patients who were included in this study. Only 18% of samples had positive bacterial growth. The most common organisms were *E.coli.*

The present study revealed that the mean of TNFR1in patients of renal impairment with positive Urine culture was high than that found in those with negative urine culture. However, the difference was statistically significant, as shown in Table 1.

Table 1: Comparison of TNFR1 between patients of renal impairment with positive and those with negative urine culture.

Patients of renal impairment		No.	TNFR1			*P value*
			Mean	S.D.	S. Error Mean	
Urine culture	Positive	13	2.106231	0.61853687	0.171551261	0.027027
	Negative	47	1.622128	0.769006548	0.112171134	

The present study revealed that the mean of TNFR1 was higher in hemodialysis patients with positive urine culture as compared with those with negative urine culture. The difference was statistically non-significant, as shown in Table 2.

Table 2:Comparison of TNFR1 between hemodialysis patients with positive and those with negative urine culture.

Hemodialysis Patients		No.	TNFR1			*P value*
			Mean	S.D.	S. Error Mean	
Urine culture	Positive	15	1.308466667	0.412931829	0.10661854	0.090652
	Negative	42	1.54281	0.52806	0.081481	

The present study suggests that the mean of TNFR1 was higher in patients with positive urine culture and normal renal function as compared with those of negative urine culture and renal impairment. The difference was statistically non- significant as shown in Table 3.

Table 3: Comparison of TNFR1 between patients with positive urine culture and normal renal function with those of negative urine culture and renal impairment.

Group	No.	TNFR1			*P value*
		Mean	S.D.	S. Error Mean	
positive Urine culture normal renal function	4	2.0675	0.958348	0.479174	0.99625
Negative Urine culture normal renal function	56	2.07	0.769726	0.102859	

The current work illustrates that in the mean of TNFR1 in the patients with positive urine culture was higher than those with negative culture. The difference was statistically non- significant.. Table 4.

Table 4: Comparison of TNFR1 between the patients with positive urine culture and those with negative culture.

Patients		No.	TNFR1			*P value*
			Mean	S.D.	S. Error Mean	
Urine culture	negative	145	1.616131	0.678300319	0.056329775	0.759681
	Positive	32	1.654094	0.621151758	0.109805155	

The present study revealed that the difference in the mean of TNFR1 between hypertensive patients with normal renal function and those with renal impairment was statistically non- significant.. Table 5.

Table 5: Comparison of TNFR1 between hypertensive patients with normal renal function and those with renal impairment.

Hypertensive Patients	No.	TNFR1			*P value*
		Mean	S.D.	S. Error Mean	
Normal renal function	24	1.615	0.546194901	0.111491567	0.15997
Renalimpairment	24	1.881958	0.733624384	0.14975045	

The present study views that the difference in the mean of TNFR1 between hypertensive control and hypertensive- hemodialysis was statistically non-significant.. Table 6.

Table 6: Comparison of TNFR1 between hypertensive control and hypertensive- hemodialysis patients.

Hypertensive Patients	No.	TNFR1			*P value*
		Mean	S.D.	S. Error Mean	
Normal renall function	24	1.615	0.546194901	0.111491567	0.290954
Hemodialysis	24	1.482375	0.261102148	0.053297253	

The difference in the mean of TNFR1 between diabetic control and diabetic renal impairment patients was statistically non- significant.. Table 7.

Table 7: Comparison of TNFR1 between diabetic control and diabetic renal impairment patients.

Diabetic Patients	No.	TNFR1			*P value*
		Mean	S.D.	S. Error Mean	
Normal renal function	18	1.468722	0.756760807	0.178370233	0.299976
Renal impairment	13	1.204308	0.633546681	0.175714234	

The difference in the mean of TNFR1 between diabetic control and diabetic hemodialysis patients was statistically non- significant.. Table 8.

Table 8: Comparison of TNFR1 between diabetic control and diabetic hemodialysis patients.

Diabetic Patients	No.	TNFR1			*P value*
		Mean	S.D.	S. Error Mean	
Normal renal function	18	1.468722	0.756760807	0.178370233	0.657251
Hemodialysis	11	1.604091	0.802233564	0.24188252	

The difference in the mean of TNFR2 between the control group and renal impairment patients was statistically non- significant.. Table 9.

Table 9: Comparison of TNFR1 between the control group and renal impairment patients.

Group	No.	TNFR1			*P value*
		Mean	S.D.	S. Error Mean	
Control	60	1.65373	.686322	.088604	0.581
Renal impairment patients	60	1.72702	.761139	.098263	

The difference in the mean of TNFR1 between the control group and hemodialysis patients was statistically significant.. Table 10.

Table10: Comparison of TNFR1 between the control group and hemodialysis patients.

Group	No.	TNFR1			*P value*
		Mean	S.D.	S. Error Mean	
Control	60	2.06983	.773957	.099917	0.004379
Hemodialysis patients	57	1.660965	0.747327046	0.098985885	

Comparison of urinary levels of TNF1 among the three groups (control, renal impairment and those on hemodialysis) using ANOVA test showed that the difference was not significant (p=0.458).

Discussion

In the present study it is significant to test ability of using urinary TNFR1as early marker for renal impairment in hypertensive and diabetic patients, and examined the effect of urinary tract infection on this diagnostic ability. Urine culture of all 180 patients was made, which was positive for 18% patients. The prevalence of UTI was higher among female than male patients and this was almost similar to that of Chih-Yen *et al* [24].

The commonest organism isolated from the urine in this study was *E.coli*. This finding goes with that of [25, 26].

Urine culture was done to see how the infection affects the level of urinary TNFR1 and how it relates with its diagnostic ability. Data of this study in this regard revealed that UTI doesn't affect the level of urinary TNFR1 except in case of renal impairment group. Corresponding studies stated that macrophages produce proinflammatory cytokines, such as tumor necrosis factor (TNF), within bacterial infections, neutrophils and epithelia cells can produce TNF [27,28,29,30,31]. Tumor necrosis factor is produced by recruited immune cells in UTI, also in infections of other organs [32]. Djojodimedjo and Soebadi, [33] concluded in their research that UTI increased the expression of TNF And TNFR-1 by nephrectomy and histological examination.

Engel and colleagues showed that TNF is increased in the bladder during UTI [34].

Mohkam et al [35] concluded in patients with acute pyelonephritis urinary TNF-α/creatinine ratio was high and after empirical treatment that urinary TNF-α/creatinine ratio was decreased and also showed that sensitivity of TNF-α/C ratio for diagnosis of acute pyelonephritis is 91%. Davidoff and coworkers[36] showed in patients with cystitis the TNF-α was significantly elevated compared with healthy individuals. Sadeghi and colleagues[37] revealed in kidney transplant patients level of urinary cytokines including TNF-α during bacteriuria was increased. In contrast to the results mentioned above, Olszyna and coworkers [38] reported that concentrations of serum and urine TNF were below the limit of detection in the vast majority of controls and pyelonephritic patients, and no significant differences between these two groups were found. They showed that only TNF receptors had higher concentrations in urine of pyelonephritic patients. Kim and colleagues[39] showed the same results, too.

Comparison of urinary TNFR1 levels between those with hypertensive on hemodialysis and hypertensive normal renal function (control), revealed that the differences were not significant and showed that the

differences were not significant in the urinary TNFR1 concentration in renal impairment hypertensive and in hypertensive patients with normal renal function (control). This finding was consistent with Puszkarska et al [40]. On the other hand, Xun and Zhao, [41] observed that level of circulating serum TNFR1 was significantly higher in CKD group than normotensive control. They did not take into account if patients had hypertensive disease which is considered to be an inflammatory condition so an elevated concentration of inflammatory cytokines includes TNF [42]. Also the current study examined urinary level of TNFR1 but not the serum concentration .

Other groups in the present study diabetic renal impairment, diabetic on hemodialysis and diabetic with normal renal function. The concentration of urine TNFR1 of each group and comparison between them showed that the differences were not significant. Again, Islam *et al* [43] concluded a strong correlation of serum, but not urine, TNFR1 concentrations with renal functions represented by GFR. Furthermore, Griffin et al [44] concluded that there was a strong association between serum TNFR-1 and GFR in patients with diabetic kidney disease and urine TNFR-1 correlated less closely with GFR.

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الامكانية التشخيصية لمستقبل ألفا 1 عامل نخر الورم البولي في مرضى السكري وارتفاع ضغط الدم للمصابين باعتلال الكلية والتهاب المجاري البولية

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الملخص

مرض الكلى المزمن هو واحد من أكثر المضاعفات شيوعا لمرض السكري وارتفاع ضغط الدم، والتي تعتبر حالة التهابية لذلك يمكن توقع تركيز مرتفع من السيتوكينات الالتهابية في مرضى ارتفاع ضغط الدم ومرضى السكري. في هذه الدراسة، تم استخدام مستقبل ألفا 1 عامل نخر الورم البولي كعلامة تشخيصية مبكرة لضعف الكلية في مرضى ارتفاع ضغط الدم ومرض السكري، وفحصت تأثير عدوى المسالك البولية على القدرة التشخيصية لمستقبل ألفا 1 عامل نخر الورم البولي. شملت الدراسة 180 عينة من مرضى ارتفاع ضغط الدم والسكري مقسمين إلى مجموعات تبعا لوظيفة الكلى عن طريق اختبار الكرياتينين في مصل الدم والاستبيان، شملت مجموعة مرضى ارتفاع ضغط الدم ومرضى السكري الذين لديهم وظيفة كلوية طبيعية، وشملت مجموعة ضعف الكلى مرضى ارتفاع ضغط الدم ومرضى السكري الذين يعانون من اختلال وظائف الكلى وشملت مجموعة الفشل الكلوي مرضى ارتفاع ضغط الدم ومرضى السكري من وحدة غسيل الكلى. تم اخذ عينات دم وعينات بول من كل هذه المجموعات. واستخدمت عينات الدم لاختبار الكرياتينين وعينات البول لاختبار الزلال والسكر عن طريق شريط فحص الادرار، تم فحص مستقبل ألفا 1 بواسطة جهاز الاليزا. بعد زرع وحضانة البول، تم تشخيص النمو عن طريق الفحص الكيميائي الحيوي الذي استخدم الوسائط التفاضلية. كانت البكتيريا الأكثر شيوعًا المعزولة هي *E. coli*. من خلال المقارنات الاحصائية للنتائج تبين أن مستقبل ألفا 1 عامل نخر الورم البولي لا يمكن استخدامه كعلامة تشخيصية لضعف الكلى في مرضى ارتفاع ضغط الدم ومرضى السكري مع أو بدون عدوى المسالك البولية حيث لا تؤثر على القدرة التشخيصية على مستقبل ألفا 1 عامل نخر الورم البولي.