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ATPase level in Iraqi Diabetic Patients with and without Diabetic Kidney Disease

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RESEARCH ARTICLE

ATPase level in Iraqi Diabetic Patients with and without Diabetic Kidney Disease

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ABSTRACT

Complications of diabetes have become one of the main problems that diabetics suffer from, especially during long periods. One of these complications is diabetic nephropathy, as both glomeruli and tubules are exposed to fibrosis due to diabetes. Recent years have witnessed great interest by researchers regarding diabetic nephropathy. This study aimed to determine the level of ATPase and some of the relevant biochemical factors in patients with diabetes and diabetic nephropathy compared with healthy controls. The study included 120 male and female (60 male and 60 female) ranging in age (30-65) years old. Ninety patients with type 2 diabetes are subdivided into three groups on the basis of ACR criteria including normoalbuminuria, microalbuminuria, macroalbuminuria (30 patients for each group). A control group of 30 healthy people was included. This study was conducted at Baghdad Teaching Hospital / Medical City and Al-Yarmouk Teaching Hospital during the period between December 2021 and May 2022 ATPase levels were determined using the ELISA technique, and HbA1c was assessed using the I- chroma device. ATPase levels were found to be significantly increased in patient groups than in healthy control. On the basis of the obtained results in this study, we can be concluded that Na-K ATPase can be applied as a reliable prognosticated factor between disease and normal cases. Furthermore, area under the curve (ROC) analysis data revealed that ATPase was found to be a perfect marker for follow-up diabetic nephropathy disease.

Keywords: ATPase, Albumin to creatinine ratio, Diabetes mellitus, Diabetic nephropathy, Estimation glomerular filtration rate

Introduction

Diabetes mellitus (DM) is defined as multi-etiological metabolic case characterized by chronic hyperglycemia, lipid and protein disorders that increase of insulin secretion, insulin action or both. [1] Tissue damage and dysfunction incidence during a long –term of disease together with failure in kidneys are symptoms of diabetes mellitus, that include thirst, polyuria, blurring of vision, and weight loss. [2] Our previous studies in diabetes and its complications include diabetic nephropathy [3], diabetic neuropathy, diabetic osteoporosis and diabetic periodontitis. [4] Acute hyperglycemia may progress to life-threatening diabetic ketoacidosis while persistent hyperglycemia correlates with macrovascular complications, raising myocardial infarction, stroke and microvascular complications risk that contribute to diabetic nephropathy, retinopathy and neuropathy. The various forms of DM have several tissues and organs. DM is the most prevalent reason for chronic renal disease, leading to terminal renal failure. The kidney include two areas that can be obviously observed an external called the cortex and an inward district called the medulla. [5] The kidney is around 11– 14 cm

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long, 6 cm wide and 4 cm thick. The left kidney is typically slightly larger than the right. [6]

The functional units of the kidney are nephrons that can only be seen microscopically. Every kidney contains roughly 1 million nephrons. [7] Nephrons oust squanders and extra fluid from blood in the form of urine after that the urine flows out of two tubes so-called ureters to the bladder. The wastes come from the breakdown of food or drinks, medicine intake, and normal muscle activity. [8]

Each nephron is a complex system of five basic parts including the glomerulus, which is a capillary: which is a capillary tuft encompassed by the extended end of a renal tubule known as Bowman's container. Each glomerulus is provided by an afferent arteriole carrying the blood in and an efferent arteriole carrying the blood out. The efferent arteriole branches into peritubular capillaries that supply the tubule. In case of fibrosis of the tubules and glomeruli, the secretion of microalbumin is increased in urine. [9] Fibrosis is a process characterized by an excessive accumulation of the extracellular matrix as a response to different types of tissue injuries, which leads to organ dysfunction. [10]

The process can be initiated by multiple and different stimuli and pathogenic factors which trigger the cascade of reparation converging in molecular signals responsible for initiating and driving fibrosis. [11] Though fibrosis can play a defensive role, in several circumstances at a certain stage, it can progressively become an uncontrolled irreversible and self-maintained process, named pathological fibrosis. [12]

The aim of this study is to evaluate ATPase levels in diabetic patients with or without diabetic nephropathy in comparison with healthy subjects to determine whether ATPase can be applied as a predictor factor for diabetic nephropathy complications.

Materials and methods

One hundred and twenty men and women, aged 30-65years participated in the study, 90 patients with diabetes who visited Baghdad Teaching Hospital / Medical City and Al-Yarmouk Teaching Hospital between December-2021 and May-2022 and 30 healthy persons as a control group.

Groups of analysis included

Control group: included 30 healthy persons looking for subjects without any diseases.

Patients groups: included 90 patients were divided into three groups according to ACR criterion:

- The normoalbuminuria group which includes 30 patients with ACR <30 mg/g.
- The microalbuminuria group which includes 30 patients with ACR 30–300 mg/g.
- The macroalbuminuria group which Includes 30 patients with ACR >300 mg/g.

Inclusion criteria

Type 2 diabetes medical history.

HbA1c-based diagnostic criteria for Type 2 diabetes.

A control group of volunteers was formed using the following criteria

Clinically healthy.

Negative for clinical indicators of systemic illnesses.

Negative for diabetes.

Exclusion criteria

Behaviors such as smoking, drinking, and chewing tobacco.

Patients with diabetic neuropathy.

Patients with diabetic retinopathy.

Patients with systemic lupus erythematosus (SLE).

Collection and analyses of sample

Seven ml of blood from the antecubital vein was withdrawn and divided into two parts. Part (1) 5ml in gel tubes coagulated at room temperature for 30 minutes. After 10 minutes of centrifugation, the serum was separated and kept in Eppendorf tubes. The first part was utilized to rapidly identify (FBS, Urea, Creatinine, Na, K) in serum using an Auto Spectrophotometer; a clinical chemistry analyzer performs diagnostic tests. Also, it was utilized after being maintained at –20°C to assess ATPase, which was evaluated using a My BioSource manufactures enzyme–linked immunosorbent test (ELISA) kit, USA. Part (2) test tube containing anticoagulant for HbA1c measurement by I- chroma a device (2 ml).

Statistical analysis

Data was statistically analyzed by SPSS software version 22. The variables were reported as means $\pm$ standard deviation the one-way analysis of variance (ANOVA) is used to determine whether there are statistically significant differences between the means of the four independent studied groups (control, DM with normoalbuminuria, DM with microalbuminuria, and DM with macroalbuminuria). [13]

Table 1. FBS and HbA1C values for both patients and control groups.

Groups	Control N = 30	Normo-albuminuria N = 30	Micro-albuminuria N = 30	Macro-albuminuria N = 30	P value
Parameters					
FBS (mg/dl)	87.46 ± 5.66 [b]	209.03 ± 48.44 [a]	211.16 ± 42.02 [a]	214.03 ± 69.49 [a]	0.0001
HbA1C (mg/dl)	5.18 ± 0.32 [a]	9.68 ± 1.94 [b]	10.08 ± 1.96 [b]	10.49 ± 2.78 [b]	0.0001

Table 2. Levels of urea, creatinine, and in the studied group.

Groups	Control N = 30	Normo-albuminuria N = 30	Micro-albuminuria N = 30	Macro-albuminuria N = 30	P value
Parameters					
Creatinine (mg/dL)	0.74 ±0.11 [b]	0.93 ± 0.24 [b]	1.40 ± 0.7 [b]	3.40 ± 2.89 [a]	0.0001
Urea (mg/dL)	30.66 ± 5.19 [c]	34.43 ± 9.10 [c]	60.12 ± 23.69 [b]	83.33 ± 38.40 [a]	0.0001

Results and discussion

The results of FBG showed a higher significant difference (P = 0.0001) between patients with mean ± SD of (209.03 ± 48.44 a, 211.16 ± 42.02 a, 214.03 ± 69.49 a) mg/dl for normoalbuminuria, microalbuminuria and microalbuminuria, respectively, and control groups. Additionally, there are no significant differences in FBS values among the three patient groups as noticed in Table 1. Also, the results of the glycated hemoglobin HbA1c referred to presence of significance differences (P = 0.0001) between patient groups (9.68 ± 1.94 b, 10.08 ± 1.96 b, 10.49 ± 2.78 b) mg/dl and control(5.18 ± 0.32 a), mg/dl but no significance differences among patient groups. HbA1c considers the best criterion for characterizing diabetic patients than non –diabetic patients that support the values of FBS in all studied groups.

Levels of creatinine and urea for all the studied groups are recorded in Table 2. The results of creatinine showed a significant difference (P = 0.0001) between the macroalbuminuria group (3.40 ±2.89 a) and other groups that include micro, normoalbuminuria and control (1.40 ± 0.7 b), (0.93 ±0.24 b) and (0.74 ±0.11 b), mg/dl respectively. Also, urea levels revealed high significant differences among macro (83.33 ± 38.40 a), mg/dL micro (60.12 ± 23.69 b) mg/dL and normo-albuminuria with control groups (34.43± 9.10 c) mg/dL with (30.66 ± 5.19 c) mg/dL respectively, as shown in Table 2.

The results of the estimated glomerular filtration are rate (eGFR) in all the studied groups, in addition to albumin to creatinine ratio (ACR) in patient groups. the results of eGFR showed highly significant differences among patient groups (macroalbuminuria, micro-albuminuria and normo- albuminuria) and control group, (37.8 ± 25.5 d, 66.33 ± 35.70 c, 89.33 ± 23.24 b and 109.03 ± 11.04 a), ml./min./m^2 respectively. At the same time, the results of ACR revealed highly significant differences among the patient groups that include macro, micro and normoalbuminuria (558.55 ±233.49 a, 111.16±60.44 b and 15.96 ± 5.46 c), mg/g respectively. As shown in Table 3.

The results in Table 4 showed the mean ±SD values of Na for the studied groups including patient (normo -albuminuria, microalbuminuria and macroalbuminuria) and control groups (138.04 ± 4.25 b), (140.32 ± 3.86b), (140.32 ± 3.86 a), and (134.23 ± 5.11 a) respectively. A highly significant (p = 0.0001) was noticed between patient groups (micro and macro) and (normo and control groups). Potassium concentrations were determined in sera of the healthy control group and patient groups as described in Table 4. Significant differences were found between patient macro albumin and both of normo, and control groups, but there is no significant difference between macro and microalbumin group.

ATPase level was determined in sera of the control group and patient groups. The results were described in Table 4. The results showed mean ± SD of ATPase among patient groups including normolalbuminuria, micro and macroalbuminuria (7.53 ± 2.63 a), (7.93 ± 2.59 a), and (8.05 ± 2.69 a) ng/ml respectively. In addition to the control group (4.06 ± 0.83 b) (ng/ml). The results showed a highly significant difference (p = 0.0001) between patient and control groups.

Receiver operating characteristic curve analyses

It is an evaluation method used in analyzing the performance of classification models in information science and statistics to measure and evaluate the efficiency of the model. It is used to distinguish between different categories in classification, such as distinguishing patients with a certain disease from

Table 3. Levels of eGFR and ACR for the studied groups.

Groups	Control N = 30	Normo-albuminuria N = 30	Micro-albuminuria N = 30	Macro-albuminuria N = 30	P value
Parameters					
eGFR (ml./min./m^2)	109.03 ± 11.04 [a]	89.33 ± 23.24 [b]	66.33 ± 35.70 [c]	37.8 ± 25.5 [d]	0.0001
ACR (mg/g)	–	15.96 ± 5.46 [c]	111.16 ±60.44 [b]	558.55 ±233.49 [a]	0.0001

Table 4. Levels of adenosine triphosphate enzyme, Na, and K between patient and control groups.

Groups	Control N = 30	Normo-albuminuria N = 30	Micro-albuminuria N = 30	Macro-albuminuria N = 30	P value
Parameters					
ATPase (ng/ml)	4.06 ± 0.83[b]	7.53 ± 2.63 [a]	7.93 ± 2.59 [a]	8.05 ± 2.69 [a]	0.0001
Na^+ (mg/ml)	134.23 ± 5.11 [b]	138.04 ± 4.25[b]	140.32 ± 3.86[a]	140.27 ± 2.47 [a]	0.0001
K^+ (mg/ml)	4.70 ±0.96[a]	4.39 ±0.49 [a]	4.18 ± 0.45 [ab]	4.06 ± 0.56 [b]	0.001

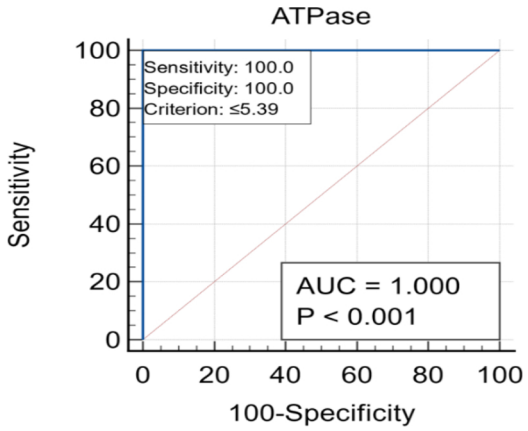

Fig. 1. ROC curve of ATPase in microalbuminuria patients.

statistics. Fig. 1 shows ROC curve of ATPase for patients with microalbuminuria. The results of ROC analysis revealed that ATPase possesses a perfect ability (since AUC is equal to 1.00) to identify diabetic patients with microalbuminuria related to normal control, as shown in Table 5. The significance level is very important ($P < 0.0001$).

Various studies have attempted[14] to identify the better markers for early prediction of damage and fibrosis in glomerulus and subsequently the results of both FBS and HbA1C support the presence of hyperglycemia in patient groups, in addition to their representing as common criteria for diabetes. Diabetic nephropathy is characterized as a silent disease during a long period without any symptoms. However, chronic hyperglycemia affects different types of kidney cells which finally leads to progressive fibrosis glomerular and tubular damage resulting kidney failure. Thus, the difficulty in identifying risks with preclinical kidney disease reflects the challenge in the prevention of diabetic nephropathy in patients with type 2 diabetes mellitus.[15]

The results of serum creatinine and urea in the current study are in agreement with previous studies by Idonije and Baxmann that indicated a strong positive association between the level of blood sugar with urea and creatinine. Plasma creatinine and urea are known indicators of glomerular filtration, with the more sensitive index of kidney function being serum creatinine. Hyperglycemia can affect all forms of renal cells, including podocyte glomerular, mesangial, cells of endothelial, tubular epithelial cells, interstitial fibroblasts and vascular endothelia, which may explain rise both of blood urea, creatinine levels along with chronic hyperglycemia.[16]

The observation from this study shows that estimation of ACR and GFR could be useful markers in this environment for early detection of diabetic nephropathy, prevention of overt nephropathy and progression to end stage renal disease. When creatinine and urea are normal, but there are early changes in glomerular basement membrane, in addition to presence of accumulated matrix materials in the mesangium, with consequent microalbuminuria, the glomerular changes at this stage can reverse pharmacological interference.[17] So, newly detected or known T2DM patients need monitoring for glycemic control, with simultaneous monitoring for early reversible nephropathy, microalbuminuria. Clinical progressions to diabetic nephropathy are defined in terms of changes in urinary albumin excretion rate and decline in glomerular filtration rate.[18]

Sodium and potassium can be used to identify risk factors for decreased kidney function and to understand the mechanisms in the development of chronic kidney disease.[18] Diabetes mellitus type 2 is of particular interest because it is associated with renal sodium retention.[19] The mechanisms of enhanced renal sodium reabsorption have not been clearly established, but evidence has been provided

Table 5. Area under the curve value of ATPase in patients with diabetic microalbuminuria.

Area Under the Curve					
Test Result Variable(s)	Area	Std. Error [a]	Asymptotic Sig. [b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
ATPase	1.000	.000	.000	1.000	1.000

a. Under the nonparametric assumption.
b. Null hypothesis: true area = 0.5.

for the involvement of epithelial sodium channel (ENaC)-mediated sodium reabsorption. This transport appears to be stimulated by the additive effects of aldosterone and the combined actions of hyperinsulinemia and hyperglycemia.[20] In addition to hyperinsulinemia-mediated renal tubular sodium transports, it has also been suggested that the increased glomerular filtration of glucose may enhance the activity of the proximal tubular Na^+-glucose co-transporter and may contribute to sodium retention.[21] when decreased renal perfusion stimulation kidney product renin enzyme. This enzyme can convert angiotensinogen to angiotensin I. Rena endothelium produce angiotensin converting enzyme this enzyme converting angiotensin I to angiotensin II. Angiotensin II acts on tubular Na reabsorption and K excretion.[22]

Our results agreed with Bolignano, et al.[23] The activity of Na-K-ATPase was significantly increased not only by aging, but also in patients with CKD with end-stage renal failure on dialysis and type 2 diabetes mellitus as well. Moreover, CKD patients and hypertensive patients tend to have increased circulating cardiotonic steroid compounds with similar structures functioning as specific inhibitors and ligands of Na-K-ATPase. Activation of Na-K-ATPase signaling stimulates ROS generation and endocytosis of Na-K-ATPase.[24] This signaling mechanism has been demonstrated in the development of CKD. This signaling mechanism also induces other pathophysiological alterations with upregulation of inflammatory cytokines .In preadipocyte 3T3- L1 cells.[25] These studies extensively elucidated that the inhibition of Na-K-ATPase restores systemic redox imbalance and attenuates the release of inflammatory cytokines. This leads to improvement of the diseased phenotype. Furthermore, Na-K-ATPase itself is able to sense changes in ROS.[26] These observations make Na-K-ATPase and its signaling function a potent regulating candidate for systemic oxidative stress and inflammation thus reducing CKD a both males and females.[27]

Conclusion

On the basis of the obtained data in this study, ATPase was found to be a perfect factor for the early Prediction of Glomerular and Tubular Damage and Fibrosis in Diabetic complications of kidney injury. This result was confirmed by many other results such as significant differences among patient groups and control, especially, the results of ROC, that referred to ATPase as a perfect marker to follow the progression of diabetic and diabetic nephropathy complications, as an early predictor.

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Author's declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images, that are not ours, have been included with the necessary permission for republication, which is attached to the manuscript.
- No animal studies are present in the manuscript.
- Authors sign on ethical consideration's approval.
- Ethical Clearance: The project was approved by the local ethical committee at The Medical City.

Author's contribution

R. K., K. K. and A. A. designed the study. R. K. conducted the collection of samples and the test. R. K.,K. K. and A. A. contributed to the design and implementation of the research, to the analysis of the results and to the writing of the manuscript.

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تقييم مستوى ATPase في مرضى السكري العراقيين المصابين أو غير المصابين بمرض الكلى السكري

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[3] قسم الطب، كلية الطب، جامعة بغداد، بغداد، العرا ق.

الخلاصة

أصبحت مضاعفات مرض السكري من المشاكل الرئيسية التي يعاني منها مرضى السكر ، خاصة خلال فترات طويلة. أحد هذه المضاعفات هو اعتلال الكلية السكري ، حيث يتعرض كل من الكبيبات والأنابيب للتليف بسبب مرض السكري. شهدت السنوات الأخيرة اهتمامًا كبيرًا من قبل الباحثين فيما يتعلق باعتلال الكلية السكري. هدفت هذه الدراسة إلى تحديد مستوى ATPase وبعض العوامل البيوكيميائية ذات الصلة في مرضى السكري واعتلال الكلية السكري مقارنة بالضوابط الصحية ، وشملت الدراسة 120 ذكراً وأنثى تتراوح أعمارهم بين (30-65) سنة. تم تقسيم تسعين مريضًا مصابًا بداء السكري من النوع 2 إلى ثلاث مجموعات على أساس معايير ACR تشمل البيلة الألبومينية الطبيعية والبيلة الألبومينية الدقيقة والبيلة الألبومينية الكبيرة (30 مريضًا لكل مجموعة) و 30 شخصًا يتمتعون بصحة جيدة كانوا بمثابة المجموعة الضابطة. ، وبناءً على النتائج التي تم الحصول عليها في هذه الدراسة ، يمكن أن تكون خلص إلى أنه يمكن تطبيق Na-K ATPaseكعامل تنبؤ موثوق بين المرض والحالات الطبيعية. علاوة على ذلك ، كشفت بيانات تحليل ROC أن ATPase وجد أنه علامة مثالية لمتابعة مرض اعتلال الكلية السكري (DN) .

الكلمات المفتاحية: ATPase، نسبة الالبومين الى الكرياتنين، داء السكري، اعتلال الكلية السكري، معدل الترشيح الكبيبي.