

Role of Renal Resistive Index in early detection of Diabetic Nephropathy in Type II Diabetic Patients**Nihal O.Youssif Ali^a, Ahmed M. Khalaf-Awad^a, Fayed HM^b, Abd elkader Ahmed Hashim^c, Ghada M. Abd ElRazik^a**^aDiagnostic and Intervention Radiology Department, Faculty of Medicine-South Valley University, Qena, Egypt^bClinical and Chemical Pathology Department, Faculty of Medicine-South Valley University, Qena, Egypt.^cInternal Medicine Department, Faculty of Medicine, South Valley University, Qena, Egypt**Abstract****Background:** Diabetic nephropathy (DN) burdens health services; renal resistive index (RI) indicates atherosclerotic changes.**Objectives:** Assessing renal artery RI for early detection of DN in type II diabetes (T2DM) patients.**Patients and methods:** A cross-sectional study analyzed 82 T2DM patients, divided into two subgroups 27 normoalbuminuric (10 males; 17 females), 55 hyper-albuminuric (26 males; 29 females), and 18 age and sex-matched healthy volunteers. All participants were evaluated by clinical examination, gray-scale renal ultrasound, Doppler evaluation of renal RI, and laboratory evaluation of glycemic control and renal functions.**Results:** T2DM without DN had a mean age of 48.6±3.9 years, while T2DM with DN had a mean age of 52.9 ±6.3 years. T2DM with DN significantly increased BMI and higher HbA1c levels compared to T2DM without DN and normal groups. T2DM patients with DN have higher renal artery RI compared to the control group. RI values showed a positive correlation with albuminuria. Renal impairment and RI increase with disease duration over 5 years. T2DM patients with DN have significantly higher renal RI (0.71 ±0.015), compared with T2DM without DN (0.639 ±0.017), and compared to the control group (0.56 ±0.02) (p < 0.001). The RI at a cutoff level of > 0.68 had an AUC of 1.0, which can discriminate T2DM with DN from that without DN with 100% in all sensitivity, specificity, PPV, and NPV, (P<0.001).**Conclusion:** Poor glycemic control and obesity negatively impact renal function in T2DM; renal RI > 0.68 is a useful test for early diabetic nephropathy evaluation.**Keywords:** Diabetic nephropathy, renal hemodynamics, Renal Resistive index***Correspondence:** nihalyoussef1994@yahoo.com**DOI:** 10.21608/SVUIJM.2023.236371.1700**Received:** 1 September, 2023.**Revised:** 21 September, 2023.**Accepted:** 30 September, 2023.**Published:** 27 May, 2024**Cite this article as:** Nihal O.Youssif Ali, Ahmed M. Khalaf-Awad , Fayed HM , Abd elkader Ahmed Hashim, Ghada M. Abd ElRazik.(2024). Role of Renal Resistive Index in early detection of Diabetic Nephropathy in Type II Diabetic Patients. *SVU-International Journal of Medical Sciences*. Vol.7, Issue 1, pp: 1011-1021.

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Introduction

By 2030, it is predicted that the prevalence of diabetic nephropathy (DN) would have increased to 44% globally, making it a significant contributor to end-stage renal disease (Zhang et al., 2020). In the absence of other renal disorders, it is characterized by an increased excretion of urine albumin, an accelerated escalation of proteinuria, and a decline in the estimated glomerular filtration rate (eGFR) (Bjornstad et al., 2019).

DN results from a complex combination of inflammatory, metabolic, and hemodynamic alterations, as well as metabolites connected to the energy system, such as fatty acids and Krebs cycle intermediates. (Zhang et al., 2020). The risk of kidney damage can be reduced by up to 50% with early detection and therapy of DKD, and slowing the progression of the disease requires the capacity to identify asymptomatic renal failure (Wettersten and Weiss, 2013).

Increased albumin excretion rate is thought to be the first and most distinct clinical symptom of DN and is currently utilized in clinical practice to pinpoint the individuals who most require the implementation/optimization of preventative interventions. It is caused by microvascular illness that causes glomerulopathy. Strict metabolic and blood pressure control, quitting smoking, modest protein restriction, and renin-angiotensin system inhibitors have all shown promise in avoiding or delaying DN, especially when used early in the course of the illness (Masulli et al., 2009).

Renal resistive index (RI) has been widely used to quantify renal blood flow as a semi-quantitative parameter measured using Doppler ultrasonography. In terms of

hemodynamics, RI stands for renal flow reserve and indicates both arterial stiffness and endothelial function (Bruno et al., 2012). Previous studies found a link between RI and the onset of a severe form of chronic kidney disease (CKD) with interstitial fibrosis (Jinadu et al., 2022). Renal artery RI is a ratio of the difference between peak systolic velocity and end-diastolic velocity to peak systolic velocity. Duplex sonography provides an easily applicable, noninvasive, and well-established method for investigating renal functional or structural changes in DN (Afsar and Elsurur, 2017). We aimed to determine the diagnostic value of measuring renal RI in the early detection of DN in type 2 diabetes mellitus (T2DM) patients.

Patients and methods

This was a cross-sectional study conducted on 82 T2DM adult patients and 18 age and sex-matched healthy volunteers (normoglycemic subjects with normal renal function) as a control group. Patients were selected randomly from the outpatient clinics of the Internal Medicine department at Qena University Hospitals from June 2021 to December 2022.

Participants were divided into 3 groups:

- Group 1: 27 T2DM patients with ACR<30mg/g.
- Group 2: 55 T2DM patients with ACR>30mg/g.
- Group 3: 18 age and sex-matched healthy subjects.

I. Inclusion criteria: Patients with T2DM. Age > 18 years old.

II. Exclusion criteria:

- Previously diagnosed renal anomalies and chronic renal diseases.
- Treated for any known renal pathology in the last 1 year.
- Patients with ESRD on hemodialysis

- Patients with known CKD such as chronic glomerulonephritis.
- Patients with major comorbidities such as cardiovascular disease or malignancies.
- Patients with a history of renal artery stenosis.
- Patients with evidence of hydronephrosis with symptoms or signs of urinary tract infection.

This study was accepted by the ethical committee of the Qena Faculty of Medicine, and all members signed a written informed consent before their inclusion in this study with ethical approval code: SVU – MED-RAD- 028-1-22-2-346.

All participants underwent the following:

I. History and Clinical Examination: -

- 1- Complete history taking including the history of comorbid conditions and risk factors such as hypertension, cardiac disease, smoking, and drug history.
- 2- Full clinical examination: with a focus on manifestations of CKD.
- 3- Anthropometric measurements: height and weight were measured, and body mass index (BMI) was calculated.

III- Laboratory Investigations

Blood sampling: 5 milliliters of venous blood were collected after a fasting period of ≥ 10 hours and allocated into 2 vacutainer tubes; three ml in a plain tube, blood left to clot and then centrifuged and the resulting sera were used for biochemical investigations, 2ml was collected into EDTA tube used for CBC and HbA1c assay.

1. Complete blood picture by Erma Automated Cell Counter (Tokyo, Japan).
2. Estimation of HbA1c using high-performance liquid chromatography (HPLC) Bio-Rad D-10 (Bio-Rad Laboratories, USA).
3. Estimation of serum chemistry; fasting blood glucose

(FBG), urea, and creatinine were measured by the Beckman Coulter (Synchron CX 9 ALX) Clinical Auto analyzer (Beckman Instruments, Fullerton, Ca, USA).

4. Morning fasting urine spot samples were collected for estimating albumin and creatinine by (Synchron CX 9 ALX) auto analyzer (Beckman Instruments, Fullerton, Ca, USA) then albumin-to-creatinine ratio (ACR) was calculated; and classified as follow: normal (ACR < thirty mg/g), microalbuminuria (30-300 mg/g), and macroalbuminuria (>300 mg/g) (Miller et al., 2018).

5. **The estimated glomerular filtration rate (eGFR:** serum creatinine levels were used to assess eGFR using the CKD Epidemiology Collaboration (CKD-EPI) equations (Levey et al., 2009). Impaired kidney function eGFR < 60 mL/min/1.73 m² or ACR > 30mg/g). The diagnosis of diabetes was defined as FBG ≥ 126 mg/dl and/or HbA1c > 6.5% or using hypoglycemic agents or by self-reported history of diabetes, according to American Diabetes Association (ADA) guidelines (ADA, 2012).

Glycemic Control: The ADA suggests measuring HbA1c for glycemic control. If HgbA1c is unavailable, an average FBS is used (ADA, 2019).

IV. Imaging: All measures were made after an overnight fast in a supine position at the end of inspiration using:

1. Conventional real-time gray-scale B-mode imaging ultrasound US was performed on all subjects per protocol with logic 3, LSD 30269WS5, General Electric, USA system with 3 MHz curve linear transducer with a wide 7 cm contact surface. The scanning was performed from the posterior-lateral direction. This provides a cross-sectional

anatomy illustration in grayscale. The depth of a pixel in the image corresponds to the separation of a tissue from the transducer, and its intensity to the tissue's echogenicity, or capacity to reflect the US signal. Echogenicity is influenced by tissue surfaces and microstructures, capturing underlying tissue differences (Szabo et al.,2014).

2 Duplex Doppler US evaluation of renal RI, using Philips iU22 x Matrix DS Ultrasound (Philips Medical System Corporation-Eindhoven-Netherlands). A longitudinal image of the kidney was made by gently positioning the ultrasound probe on the flank in oblique projection. The main track of the renal arteries' Doppler beam was replaced. The renal size, cortical thickness, cortico-medullary differentiation, and echogenicity were assessed. The US equipment automatically estimated the renal RI. Intra-renal resistance was recorded at interlobar arteries in 3 different zones of both kidneys (inferior, middle, and superior zones), and the mean value was estimated. Then, a mean RI was determined and derived from six measures for each patient. RI can be calculated using the built-in software as follows: $RI = [\text{peak systolic velocity} - \text{end-diastolic velocity}] / \text{peak systolic velocity}$ (Bruno et al., 2011).

Statistical analysis

The data were studied using the Statistical

Software for Social Sciences (SPSS) version 26.0. All continuous variables were tested for normality using the Kolmogorov–Smirnov test. Numerical variables were shown as mean \pm standard deviation(M \pm SD) while categorical variables were shown as frequency and percentage number (%), Mann-Whitney U test (MW): when comparing two means (for normally distributed data). Kruskal-Willis test (KW): when comparing between more than two means (for abnormally distributed data). Chi-square test: was used when comparing non-parametric data. Receiver Operating Characteristic (ROC) Curve was used to detect cutoff value, sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and diagnostic accuracy of RI in discrimination between groups by determining the area under the curve (AUC) at the specified cutoff value. Statistical analyses were two-sided, and a $P < 0.05$ was considered significant.

Results

The study was carried out on 100 subjects, involving 3 groups: Group 1: T2DM without DN, 27 patients (10 males and 17 females) with a mean age of 48.6 ± 3.9 years (ranges from 44-52 years), and a mean BMI of 31.5 ± 4.9 kg/m². Group 2: T2DM with DN, 55 patients (26 males and 29 females), with a mean age of 52.9 ± 6.3 years (ranges from 46-58 years), and mean BMI of 34.9 ± 6.6 kg/m². Group 3: control group: 18 healthy age and sex-matched non-diabetic subjects (6 males and 12 females), mean age of 47.4 ± 4.5 years (range from 43-51 years), and mean BMI of 30.6 ± 3.7 kg/m², (Table.1).

Table 1. Demographic data in the studied groups.

Variables		Groups						Test	P-value
		T2DM without DN (No = 27)		T2DM with DN (No = 55)		Normal (No = 18)			
Age (years) Mean \pm SD		48.6	± 3.9	52.9	± 6.3	47.4	± 4.5	KW = 16.5	< 0.001*
Sex	Male N (%)	10	37%	26	47.3%	6	33.3%	$\chi^2 = 1.45$	0.483 NS

	Female N (%)	17	63%	29	52.7%	12	66.7%		
	BMI (Kg/m²) Mean ± SD	31.5	± 4.9	34.9	± 6.6	30.6	± 3.7	KW = 6.07	0.048*
	Disease duration (months)	47.3	± 11.5	61.7	± 15.4	---	---	MW = 281	< 0.001*
TTT regimen	Oral N (%)	18	66.7%	41	74.5%	---	---	X ² = 1.21	0.545 NS
	Insulin N (%)	5	18.5%	10	18.2%	---	---		
	Both N (%)	4	14.8%	4	7.3%	---	---		
	Hypertension N (%)	7	25.9%	20	36.4%	6	33.3%		0.640
	Smoking N (%)	7	25.9%	18	32.7%	0	0%		0.021

*: significant; KW: Kruskal Willis test; MW: Mann Whitney U test; X²: Chi-square test.

Demographic data

T2DM with DN were significantly older (52.9 ± 6.3 years) compared with T2DM without DN (48.6 ± 3.9 years) and normal (47.4 ± 4.5 years) ($P < 0.001$), with significantly higher BMI compared with T2DM without DN ($31.5 \pm 4.9 \text{ kg/m}^2$) and normal ($30.6 \pm 3.7 \text{ kg/m}^2$) ($P = 0.048$), and significantly longer disease duration (61.7 ± 15.4 months) compared with T2DM without DN (47.3 ± 11.5 months) ($P < 0.001$), (**Table.1**).

T2DM with DN had a significantly higher percentage of smokers (32.7%) compared with T2DM without DN (25.9%) and normal (0%) ($P = 0.021$), (**Table.1**).

Poor glycemic control is evident as high risk for DN, as T2DM with DN had significantly higher FBS (212.2 ± 22.9 mg/dl) compared with T2DM without DN (157.6 ± 21.7 mg/dl) and normal (91.7 ± 25.3 mg/dl) ($P < 0.001$). T2DM with DN had significantly higher HbA1c ($8.2 \pm 0.5\%$) compared with T2DM without DN ($6.9 \pm 0.2\%$) and normal ($5.3 \pm 0.5\%$) ($P < 0.001$).

T2DM with DN had significantly higher ACR (222.8 ± 54.6 mg/g) compared with T2DM without DN (19.9 ± 6.2 mg/g) and normal (17.3 ± 5.9 mg/g) ($P < 0.001$). T2DM with DN had significantly higher s. urea (40.4 ± 11.2 mg/dl) compared with T2DM without DN (16.8 ± 2.1) and normal (15.6 ± 1.8 mg/dl) ($P < 0.001$). T2DM with DN had significantly higher s.

creatinine (1.38 ± 0.28 mg/dl) compared with T2DM without DN (0.93 ± 0.14 mg/dl) and normal (0.9 ± 0.13 mg/dl) ($P < 0.001$). T2DM with DN had significantly lower GFR (61.8 ± 20.1 ml/min/1.73m²) compared with T2DM without DN (99.0 ± 12.7 ml/min/1.73m²) and normal (102.6 ± 14.8 ml/min/1.73m²) ($P < 0.001$). T2DM with DN had significantly higher ALB in urine ($P < 0.001$), where 33 patients (60%) had (+) ALB and 22 patients (40%) had (++) ALB in urine while T2DM without DN had no ALB in urine, (**Table.2**).

T2DM with DN had a significantly higher percentage of echogenic grade I left kidney in 23 (41.8%) patients when compared without DN (0%) and normal (0%) ($P < 0.001$), with insignificant differences concerning the right kidney grayscale ($P = 0.073$), (**Table 3**). T2DM patients with DN have significantly higher renal RI, therefore they have significantly higher values in the right kidney (0.71 ± 0.015), left kidney (0.71 ± 0.02), and both kidneys (0.71 ± 0.015) when compared with T2DM without DN, right kidney (0.640 ± 0.016), left kidney (0.637 ± 0.019), and both kidneys (0.639 ± 0.017), and when compared to control group right kidney (0.57 ± 0.03), left kidney and both kidneys (0.56 ± 0.02) ($P < 0.001$), (**Table 4**.)

Table 2: Laboratory values in the studied groups

Laboratory parameters Mean \pm SD	Groups						KW	P-value	
	T2DM without DN (No = 27)		T2DM with DN (No = 55)		Normal (No = 18)				
Hb (g/dl)	13.6	± 1.2	13.5	± 1.1	14.1	± 1.3	3.76	0.152	
PLTs ($\times 10^3$ /ul)	194	± 44.3	177	± 39.6	171	± 16.6	2.08	0.353	
WBCs ($\times 10^3$ /ul)	5.9	± 0.6	5.8	± 0.6	5.9	± 0.5	0.6	0.738	
FBS (mg/dl)	157.6	± 21.7	212.2	± 22.9	91.7	± 25.3	68.5	< 0.001*	
HbA1c (%)	6.9	± 0.2	8.2	± 0.5	5.3	± 0.5	79.5	< 0.001*	
ACR (mg/g)	19.9	± 6.2	222.8	± 54.6	17.3	± 5.9	73.9	< 0.001*	
Urea (mg/dl)	16.8	± 2.1	40.4	± 11.2	15.6	± 1.8	70.3	< 0.001*	
Creatinine (mg/dl)	0.93	± 0.14	1.38	± 0.28	0.90	± 0.13	62.9	< 0.001*	
eGFR (ml/min/1.73m ²)	99.0	± 12.7	61.8	± 20.1	102.6	± 14.8	55.6	< 0.001*	
Urine ALB	Nil N (%)	27	100%	0	0	18	100%	X ² = 100	< 0.001
	+ N (%)	0	0	33	60%	0	0		
	++ N (%)	0	0	22	40%	0	0		

*: significant; KW: Kruskal Willis test; X²: Chi-square test

Table 3. Kidney grey scale results in the studied groups

Kidney Gray-scale Echogenicity N (%)		Groups						Test	P-value
		T2DM without DN (No = 27)		T2DM with DN (No = 55)		Normal (No = 18)			
Rt. side	Normal	27	100%	49	89.1%	18	100%	X ² = 5.2	0.073 NS
	Grade I	0	0%	6	10.9%	0	0%		
Lt. side	Normal	27	1 0 0 %	32	58.2 %	18	100%	X ² = 24.4	< 0.001 HS
	Grade I	0	0 %	23	41.8 %	0	0%		

X²: Chi-square test.

Table 4. Renal artery Resistive Index in the studied groups

Resistive Index (RI) Mean \pm SD	Groups						KW	P-value
	T2DM without DN (No = 27)		T2DM with DN (No = 55)		Normal (No = 18)			
Right kidney	0.640	± 0.016	0.71	± 0.015	0.57	± 0.03	80.2	< 0.001
Left kidney	0.637	± 0.019	0.71	± 0.02	0.56	± 0.02	79.6	< 0.001
Both kidney	0.639	± 0.017	0.71	± 0.015	0.56	± 0.02	79.8	< 0.001

*: significant; KW: Kruskal Willis test.

Table 5. Diagnostic performance of RI in discriminating T2DM without DN from the control group

RI	Cut off	AUC	Sensitivity	Specificity	PPV	NPV	p-value
		> 0.62	0.97	85.2%	88.9%	88.5%	85.7%

AUC: Area under the curve; NPV: negative predictive value; PPV: positive predictive value

Table 4. Renal artery Resistive Index in the studied groups

Resistive Index (RI) Mean ± SD	Groups						KW	P-value
	T2DM without DN (No = 27)		T2DM with DN (No = 55)		Normal (No = 18)			
Right kidney	0.640	± 0.016	0.71	± 0.015	0.57	± 0.03	80.2	< 0.001
Left kidney	0.637	± 0.019	0.71	± 0.02	0.56	± 0.02	79.6	< 0.001
Both kidney	0.639	± 0.017	0.71	± 0.015	0.56	± 0.02	79.8	< 0.001

*: significant; KW: Kruskal Willis test.

Table 5. Diagnostic performance of RI in discriminating T2DM without DN from the control group

RI	Cut off	AUC	Sensitivity	Specificity	PPV	NPV	p-value
	> 0.62	0.97	85.2%	88.9%	88.5%	85.7%	< 0.001

AUC: Area under the curve; NPV: negative predictive value; PPV: positive predictive value

The RI at a cutoff level of > 0.62 had an AUC of 0.92, which can discriminate T2DM without DN from normal with 85.2% sensitivity,

88.9% specificity, 88.5% PPV, and 85.7% NPV, (P < 0.001 (Table.5, Fig.1).

RI (DM without DN & Normal groups)

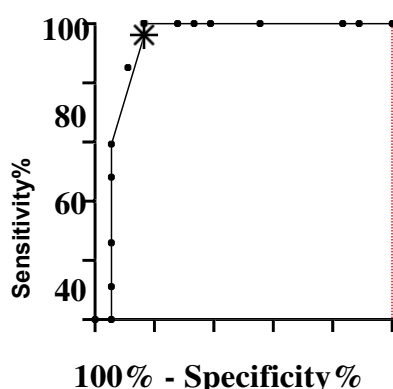


Fig.1. ROC curve showing the discriminatory point of RI differentiation between normal from T2DM without DN

Table 6. Diagnostic performance of RI in discriminating T2DM without DN from T2DM with DN

	Cut off	AUC	Sensitivity	Specificity	PPV	NPV	p-value
RI	> 0.68	1.0	100%	100%	100%	100%	< 0.001

AUC: Area under the curve; NPV: negative predictive value; PPV: positive predictive value

The RI at a cutoff level of > 0.68 had an AUC of 1.0, which can discriminate T2DM with DN from that without DN with 100% in all

sensitivity, specificity, PPV, and NPV, (P< 0.001).Table (Table.6, Fig.2).

RI (DM without DN & DM with DN groups)

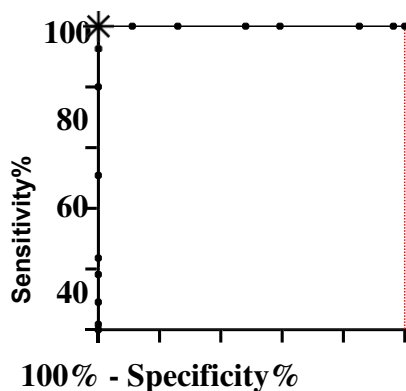


Fig. 2. ROC curve showing the discriminatory point of RI differentiation RI between T2DM without DN from T2DM with DN

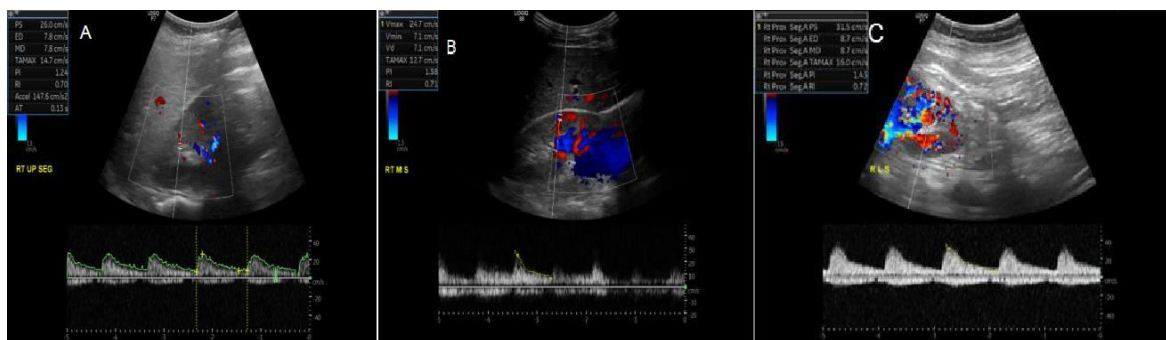


Fig.3. A 49 year-old male patient known to be diabetic for 49 months, not hypertensive, smoker, HbA1c: 7.9 % and Al/ Cr Ratio: 240 mg/gm , renal Doppler was done revealed right kidney mean RI +/- .71 and left kidney mean RI +/- .71 (Figure III) : A) right upper segmental artery Doppler showing RI.7.B)RightmiddlesegmentalarteryDopplershowingRI.72. C) Right lower segmental artery Doppler showing RI. 72.

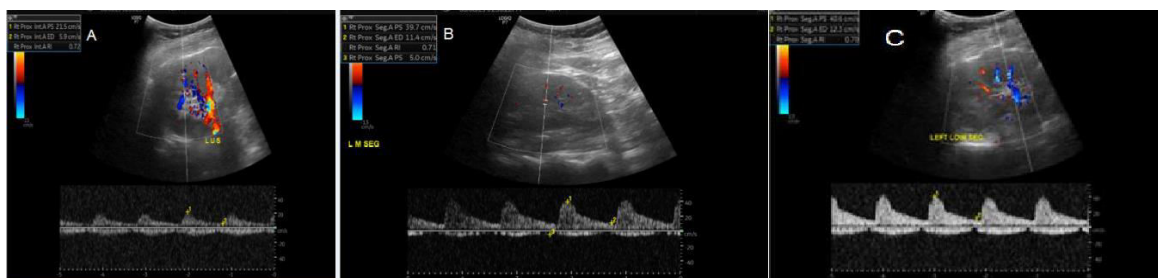


Fig. 4. A 45-year-old female patient known to be diabetic for 84 months, not hypertensive, smoker, HbA1c: 8.1 % and Al/ Cr Ratio: 241 mg/gm, renal Doppler was done revealed right kidney mean RI +/- .7 and left kidney mean RI +/- .71 (Figure IV): A) left upper segmental artery Doppler showing RI .72. B) left middle segmental artery Doppler showing RI .72. C) left lower segmental artery Doppler showing RI .7.

Discussion

Diabetes mellitus (DKD) is a significant global health issue, that affects quality of life and economic costs. Risk factors include hyperglycemia, predisposing genes, and factors like smoking, ethnicity, and age (Liang et al., 2017). This study aimed to determine the diagnostic value of the renal resistivity index in the early detection of DN in T2DM patients.

Results of the current study showed that T2DM with DN had significantly higher BMI ($34.9 \pm$

66 kg/m^2) compared with T2DM without DN ($31.5 \pm 4.9 \text{ kg/m}^2$) and control group ($30.6 \pm 3.7 \text{ kg/m}^2$). Since obesity harmed renal function in diabetic patients as it was associated with significant proteinuria, this agreed with the findings of (Chen et al., 2013; Abougambou et al., 2016).

In the current study, we found that poor glycemic control has a significantly high risk for DN. T2DM with DN group had significantly higher FBG ($212.2 \pm 22.9 \text{ mg/dl}$) and HbA1c ($8.2 \pm 0.5\%$) compared with T2DM without DN ($157.6 \pm 21.7 \text{ mg/dl}$) and ($6.9 \pm 0.2\%$), and control group ($91.7 \pm 25.3 \text{ mg/dl}$) and ($5.3 \pm 0.5\%$), ($p < 0.001$). This agreed with (Al-Rubeaan et al., 2014).

In this study, T2DM with DN were significantly older with longer disease duration and high BMI, higher blood chemistry of FBS, HbA1c, S. urea, S. creatinine, ACR, and albuminuria that were correlated with renal artery RI, with insignificant sex difference which emphasizes the importance of tight diabetic control to avoid or postpone nephropathy. Hamano et al reported a significant association between urinary albumin excretion and RI (Hamano et al., 2008). Also, it was observed that renal RI was highest in patients with increased 24-h urinary albumin (Afsar and Elsurer, 2012).

In this study, we utilize a Gray-

scale US for the assessment of DM impact on kidney morphology, we found that T2DM with DN had a significantly higher percentage of echogenic grade I left kidney when compared with DM without DN. This agreed with (Jastaniah et al., 2013) who reported that there was abnormal renal echogenicity with nephropathy Grade 1 which was so greater than Grade 2 that showed decreased renal size among diabetic patients.

In this study, DM with DN had significantly higher renal RI (0.71 ± 0.015) when compared with DM without DN group (0.639 ± 0.017) and normal group (0.56 ± 0.02) ($P < 0.001$). This agreed with (Sari et al., 1999; and Yamaguchi et al., 2019), and in type 1 DM (Abdel Dayem et al., 2016; Maksoud et al., 2019).

In our study, the analysis of the ROC curve indicates that the RI at a cutoff level of > 0.62 had an AUC of 0.92, which can differentiate between normal and T2DM without DN with sensitivity 85.2%, specificity 88.9%, PPV 88.5%, and NPV 85.7%, ($P < 0.001$). In our study, the ROC curve showed that the renal RI at a cutoff level of > 0.68 had an AUC of 1.0, which is a predictor of DN in T2DM with 100% in all sensitivity, specificity, PPV, and NPV, ($P < 0.001$), this was in line with the findings (Rui et al., 2012; Shirin et al., 2015; Jinadu et al., 2022).

Conclusion

Poor glycemic control and obesity negatively impact renal function in T2DM; renal RI > 0.68 is a valuable adjunct test for assessing functional abnormalities in renal hemodynamics in the early stages of diabetic nephropathy.

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