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Original Research

Relationship Between Renal Resistive Index and Increased Renal Cortical Stiffness in Patients with Preserved Renal Function

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ABSTRACT

Objective: We aimed to investigate the relationship between cortical stiffness (CS) determined by shear wave elastography (SWE) and conventional ultrasonography (USG) parameters, and identify the determining parameters that increased CS.

Methods: In this study, 229 patients who underwent renal USG were included. In addition to conventional renal USG, SWE was performed. Patients were divided into two groups: with increased CS and without increased CS.

Results: The median CS value of the patients included in the study was 4.92 kPa. The increased CS value was taken as the limit value of 5.0 kPa. The age, creatinine and estimated glomerular filtration rate levels, and presence of diabetes and hypertension were significantly higher in the increased CS group ($p<0.05$). It was found that the cortical echogenicity increase (stage I-II), renal resistance index (RRI), and acceleration time values were significantly higher in patients with increased CS ($p<0.05$). When logistic regression analysis was performed to identify patients with increased CS, we found that RRI, diabetes presence, and cortical echogenicity stages I and II independently predicted an increase in CS ($p<0.05$). According to this analysis, RRI (every-0.1), diabetes, and cortical echogenicity stages I and II increased the risk of increased CS by 2.3 times, 14%, 10.5% and 18.2%, respectively. In ROC analysis for RRI, the area under the curve was 0.719. When the cut-off value for RRI was taken as 0.70, it was found to be 71.1% sensitive and 64.3% specific for increased CS.

Conclusion: The increase in renal echogenicity and RRI obtained by conventional USG studies independently identifies patients with increased CS.

Keywords: Conventional ultrasonography, cortical stiffness, renal resistive index

INTRODUCTION

Conventional renal ultrasonography (USG) can be used to detect kidney size, cortical thickness, and parenchymal echogenicity. It also can reveal indicators of chronic morphologic changes that happen in many renal diseases. However, these changes are not quantitative and clear. Atrophic changes cannot be shown with conventional renal USG in patients with stage 3-4 chronic kidney disease (CKD) with diabetes mellitus (DM), which is the most common reason for CKD. For this reason, conventional renal USG is not informative in determining the progression and stages of CKD (1-3). Shear wave elastography (SWE) is a non-invasive, stable, and cost-effective USG study that has been used in recent years to evaluate tissue elasticity and cortical stiffness (CS) (4-6). The CS evaluation with SWE has been shown to be a simple and practical technique that can be used in the detection of chronic morphological changes in patients with renal transplant, in renal tumors, and in CKD due to DM and in CKD grading (7-10). The SWE is used to screen patients for many organ pathologies in addition to kidneys. These include patients with liver, breast, prostate, pancreas, testicle, thyroid diseases, and renal transplant. However, the SWE examination is not available on every USG device, nor can any radiology specialist do it. In subgroup analyses with patients with renal transplant, a correlation between some conventional USG parameters and CS was reported in literature (10-13). As much as we investigated, no study has evaluated the relationship between CS value determined by SWE and conventional renal USG parameters in patients with preserved renal function who planned to undergo a renal USG screening.

In this study, we aimed to investigate the relation between conventional USG parameters and CS obtained by SWE, and determine the parameters that show an increased CS.

METHODS

Study Protocol and Study Population

Patients who underwent renal USG between September 2017 and May 2018 in University of Health Sciences, Adana Health Practice and Research Center, Radiology Clinic were screened, and 229 patients (173 males, 56 females; and mean age 62.1±12.1

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years) were included in the study. In addition to conventional renal USG (B-mode and Doppler) examinations, SWE examination was performed. The patients who had undergone renal USG examination, those who had estimated glomerular filtration rate (eGFR) <60 mL/min/1.73m^2, or patients who had proteinuria >30 mg/L were excluded from this study. Patients with renal artery stenosis and post-renal renal failure, nephrectomy, malignancy, systemic, or urinary tract infections during renal USG, non-detectable CS, and non-volunteers were excluded. The study was conducted according to the recommendations of the Declaration of Helsinki about biomedical research involving human subjects, and the protocol was approved by the institutional ethics committee. All forms of voluntary consent for all patients were explained in detail, and patients were included in the study after obtaining written consent.

After detailed physical examination of all patients, risk factors were questioned. Age, gender, hypertension (HT), hyperlipidemia, smoking, and DM presence were studied as demographic data of the patients included in the study. Creatinine and blood urea nitrogen levels were measured in all patients. Estimated glomerular filtration rate was calculated using the Modification of Diet in Renal Disease (MDRD) formula. Patients were measured for height and weight, and their body mass index (BMI) were calculated.

Renal USG

All patients underwent renal USG screening using high-resolution USG device with 1-5 MHz high-resolution convex probe (Philips EPIQ 7, Philips Healthcare, Bothell, Washington, USA). Ultrasound scanner setting was made useful for every patient for all B-mode USG examination (gain [55-75 dB]; penetration depth [6-16 cm]; dynamics range [50-60] and zoom range [0.8-2.0]). The USG examinations were performed after minimum 6 h of fasting, and after minimum 20 min of rest. B-mode USG evaluation was first performed on the gray scale, and then quantitative Doppler parameters were obtained. Kidney sizes, cortical thickness, and parenchyma echogenicity were assessed on gray scale. Kidney length was measured in the coronal plane from its upper pole to the lower pole. Renal width was measured from middle pole, and it was recorded as the distance between renal hilum and renal capsule. Cortical thickness was recorded as the distance from the medial section of the renal medullary pyramid base to the renal capsule. Doppler USG was measured with peak systolic velocity (PSV), end diastolic velocity (EDV), and acceleration time (AT) was measured with Doppler USG in Doppler angle 30°-60° in the right and left common renal and interlobular arteries (Figure 1). After the PSV and EDV values were taken, spectral waveform was manually drawn on the device, and the renal resistive index (RRI) value was automatically obtained according to the PSV-EDV/PSV formula. The renal pulsatility index (RPI) was calculated based on the PSV-EDV/average flow rate formula on the spectral waveform. The AT was calculated as the time from the point where the systolic wave began to increase to the first peak point. All measurements were performed three times from the right and left kidney main and interlobular arteries. The arithmetic mean values of RRI, RPI, and AT values obtained from the right and left kidneys were recorded.

The SWE evaluation was performed using 5-1 convex abdominal probe, elastography point quantification (ElastPQ) technique. All measurements were performed as previously described (7). Patients were examined in left and right lateral decubitus position for renal USG. During renal USG, the probe was compressed as lightly as possible, and was placed in a stable position. The patient was asked not to breathe for a few seconds to minimize the movement of the kidney with respiration. The measurement was calculated by placing the region of interest (ROI) on the target (Figures 2 and 3) on the conventional USG image of the renal USG, after the target region was determined. The ROI was placed perpendicular to a vascular-free or cyst-free zone in the renal cortex. The main axis of the ROI was adjusted parallel to the axis of the kidney pyramid (perpendicular to the surface of the kidney). In our study, the ROI target distance was maximum

Figure 1. Renal resistive index measurement by Doppler ultrasonography: Increased RRI in 0.77 is displayed in the lateral right corner

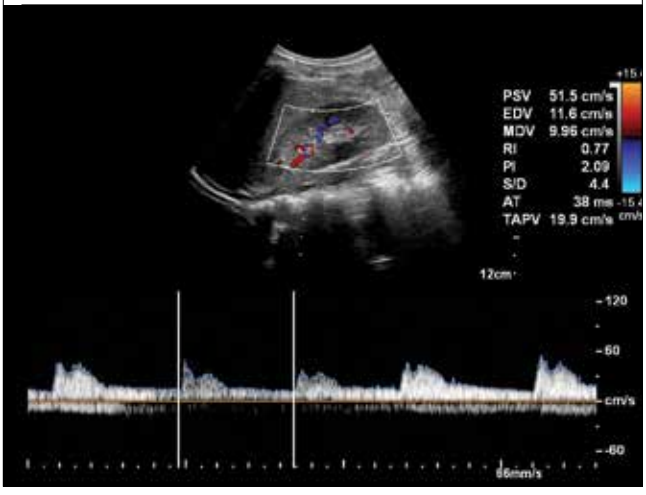


Figure 2. Cortical stiffness measurement by shear wave elastography: Normal shear wave velocity in 2.40±0.86 kPa is displayed in the lower left corner and grade 0 or normal renal parenchymal echogenicity

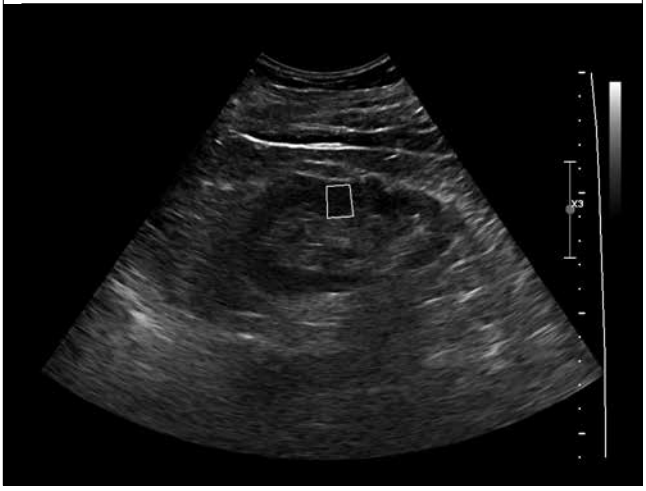


Figure 3. Cortical stiffness measurement by shear wave elastography: Increased shear wave velocity in 15.11±8.11 kPa is displayed in the lower left corner and grade II renal parenchymal echogenicity

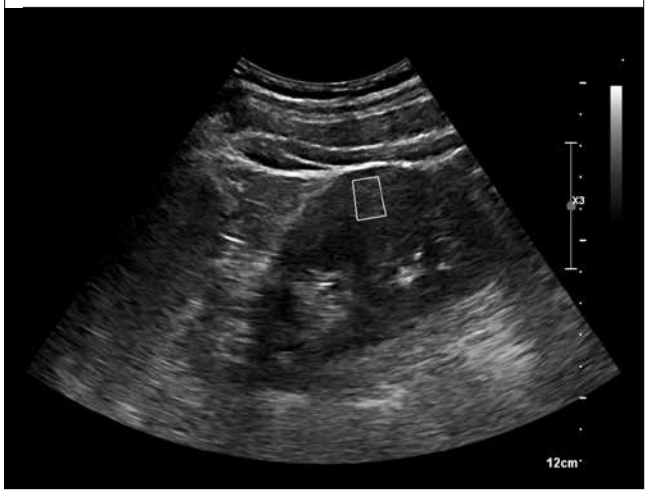
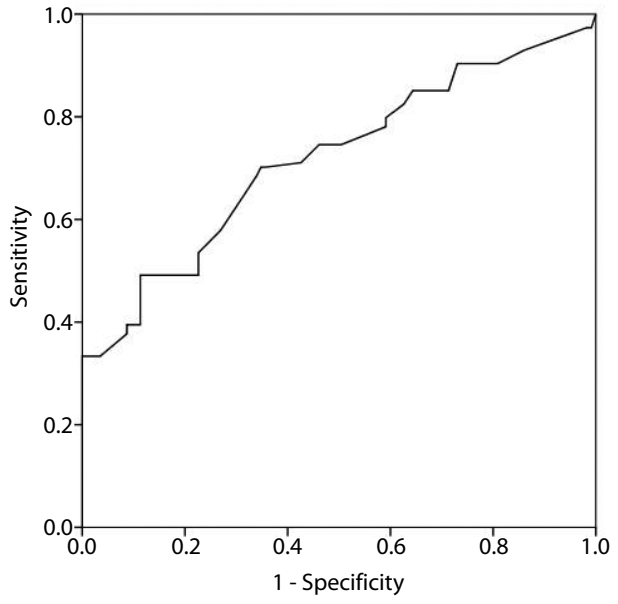


Figure 4. The ROC curve analysis of renal resistive index for predicting increased cortical stiffness



8 cm and the ROI fixed box size was 1 cm-0.5 cm. The compression applied was minimized as much as possible during imaging to avoid mechanical pressure to the kidney. Then, the same examination procedure was repeated for the contralateral kidney. In each case, six valid measurements were obtained for each kidney, and the mean value was calculated. If the measurement reliability is low, a result of 0.00 kPa will be displayed. The result is expressed in kPa value. Subjects were evaluated by a single well-experienced radiology specialist for conventional, Doppler, and SWE examinations. The specialist had more than five years of experience in the SWE studies and had performed at least 500 SWE procedures in a year.

Statistical Analysis

All analyses were performed using the IBM Statistical Package for the Social Sciences 22.0 (IBM SPSS Corp.; Armonk, NY, USA) statistical software package. The normal distribution of continuous variables was assessed using the Kolmogorov-Smirnov test. The continuous variables in the group were expressed as mean±standard deviation. Categorical variables are given in numbers and percentages. Student's t test was used to compare continuous variables between groups. Chi-square (χ^2) test was used to compare categorical variables. For univariate correlation analysis, Pearson correlation analysis was used. Logistic regression analysis was performed to determine the indices independent of the increased CS-related parameters detected in univariate analyses. An ROC curve analysis was performed to re-evaluate the countable parameters that independently determine the patients with elevated CS, and to determine the limit value of these markers. The value of the area under the curve was used as the measure of the accuracy of the test. $P<0.05$ was accepted as statistically significant.

RESULTS

The median and mean CS values of the patients included in the study were 4.92 kPa and 5.33±2.1 kPa, respectively. The increased CS value was taken as the limit value of 5.0 kPa. Patients were divided into two groups: patients with increased CS and without increased CS. All parameters were compared between the two groups, and parameters determining the patient group with increased CS were found.

Demographic Characteristics of Patients with and without Increased CS

When the demographic findings were compared between patients with and without increased CS, the age, creatinine, and eGFR levels as well as the frequency of DM and HT were significantly higher in patients with increased CS (Table 1).

Conventional Renal USG Findings of Patients with and Without Increased CS

When conventional renal USG findings were compared between patients with and without increased CS, among B-mod USG parameters, only cortical echogenity was found to be increased in patients with increased CS (Table 2). In the patient group with increased CS, the RRI and AT values determined with Doppler USG were found to be significantly higher (Table 2).

Identification of Patients with Increased CS

To identify patients with increased CS, when parameters found to be significant in univariate analysis were evaluated with logistic regression analysis, RRI, DM presence, and stage I and II cortical echogenicity independently defines an increased CS risk. According to this analysis, RRI (every 0.1), presence of DM, and stage I and II cortical echogenicity increased the risk of increased CS by 2.3 times, 14%, 10.5%, and 18.2%, respectively (Table 3).

ROC Curve Analysis for the Identification of Patients with Increased CS

When ROC analysis was performed to determine patients with

Table 1. Baseline characteristics and renal functions in patients with increased and normal cortical stiffness

	Increased CS n=119	Normal CS n=110	p
Age (years)	64.4±10.9	59.4±12.7	0.002
Gender (male)	92	81	0.005
Office systolic BP (mmHg)	126.9±9.2	126.9±12.2	0.969
Office diastolic BP (mmHg)	85.5±7.1	83.4±9.5	0.065
Heart rate (beat/min)	77.3±12.4	77.8±10.4	0.719
Weight (kg)	83.8±6.7	81.7±7.6	0.022
Height (cm)	169.1±5.2	167.1±7.6	0.021
Body mass index (kg/m^2)	29.4±2.3	29.3±2.4	0.782
Smoking, n (%)	38 (33.3%)	32 (27.8%)	0.223
Hypertension, n (%)	60 (52.6%)	36 (31.3%)	0.001
Diabetes mellitus, n (%)	49 (43.0%)	11 (9.6%)	<0.001
Hypercholesterolemia, n (%)	25 (21.9%)	16 (13.9%)	0.079
Blood urea nitrogen (mg/dL)	37.1±9.7	36.9±10.3	0.610
Creatinine (mg/dL)	0.91±0.21	0.81±0.16	0.020
eGFR (mL/min/1.73 m^2)	108±22	115±24	0.013

BP: blood pressure; CS: cortical stiffness

Table 2. Conventional renal ultrasound and shear wave elastography imaging parameters

	Increased CS n=114	Normal CS n=115	p
Renal resistive index	0.76±0.08	0.72±0.05	<0.001
Renal pulsatility index	1.94±0.70	1.83±0.49	0.178
Renal AT (m/s)	105.7±44.4	92.4±37.8	0.015
Kidney length (mm)	102.3±9.3	100.9±8.8	0.245
Kidney width (mm)	49.6±6.5	48.9±6.9	0.528
Cortical thickness (mm)	12.1±1.9	11.7±2.0	0.092
Cortical echogenicity Grade 0-I-II, n	70-36-8	90-24-1	0.003
Cortical stiffness (kPa)	7.42±2.11	3.95±1.01	<0.001

AT: acceleration time; CS: cortical stiffness

increased CS of RRI values, the area under the ROC curve was 0.719, which was statistically significant (p<0.001) (Table 4). The ROC curve for RRI is shown in Figure 4. When the cut-off value for RRI was taken as 0.70, it was found to be 71.1% sensitive and 64.3% specific for increased CS (Table 4).

Table 3. According to multivariate regression analysis, independent risk factors for occurrence of increased cortical stiffness

	Odds ratio	95% Confidence interval	p
Renal resistive index (each 0.1)	2.323	1.442-3.758	<0.001
Diabetes mellitus (presence)	0.140	0.068-0.289	<0.001
Cortical echogenicity (Grade I)	0.105	0.012-0.920	0.042
Cortical echogenicity (Grade II)	0.182	0.020-1.658	0.037

Table 4. ROC curve analysis of renal resistive index for predicting increased cortical stiffness

Variable	AUROC Curve	p	Cut-off	Sensitivity	Specificity
Renal resistive index	0.719 (0.653-0.785)	<0.001	0.70	71.1%	64.3%

DISCUSSION

The most important finding of our study is that the CS values obtained by SWE examination in patients with preserved renal function is independently correlated with an increase in conventional USG parameters of RRI and cortical echogenity. It has been found that when the limit value for RRI is taken as 0.70, it independently determines the state of increased CS. Therefore, RRI can be used as an objective parameter in renal CS determination in patients with preserved renal function.

The SWE is a newly developed USG technique that can quantitatively measure tissue stiffness without an invasive procedure. It has been shown that renal fibrosis in patients with CKD increases renal CS, which can be measured by SWE (12-15). In addition to renal elasticity, SWE is used screen patients for many organ pathologies in addition to kidneys. These are patients with liver, breast, prostate, pancreas, testicle, and thyroid diseases, and patients with renal transplant. Studies in patients with renal transplant show the severity of renal histologic changes more clearly in the SWE examinations than B-mode USG (13, 16, 17). Similar findings have been shown in studies performed on native kidneys (18). According to the result of these renal elastography studies, the CS value determined by SWE can be used as a renal parenchymal disease and renal fibrosis indicator in clinical practice. In some studies comparing CS and demographic parameters, there was no significant relationship between CS and age and gender (7, 10, 19). However, another study by Goya et al. (6) found a positive relationship between CS and age in healthy controls (6). In the same study, no significant relationship was observed between CS and gender in patients with CKD and in healthy controls. Similar to the study conducted by Goya et al. (6), in our study, it was found that patients with increased CS were older, but gender is

not related with an increase in CS. In addition, it was found that DM-induced CKD was positively correlated with CS and proteinuria, and negatively correlated with eGFR (7). However, another study did not find a significant association between CS and eGFR in patients with renal transplant (10). In a study, it was reported that there is a close relationship between mean CS value and eGFR and creatinine value (6). In our study, we found that both creatinine level and eGFR were higher in patients with increased CS, but neither of these two parameters are associated with increased CS independently.

With conventional B-mode USG, non-invasive knowledge of renal size and position, renal mass and obstructive uropathy can be obtained, and is still the most reliable examination of screening renal diameter and morphology. The CS values were found to be unrelated to renal length and width in patients with diabetic CKD (7). Furthermore, in the same study, no relationship was observed between CS and renal cortical thickness in the healthy control group, whereas CS and renal cortical thickness were negatively correlated in patients with DM-related CKD (7). In another study conducted on a normal patient group, no relationship was reported between renal length, width, cortical and parenchymal thickness obtained by conventional renal USG, and CS obtained by SWE (19). In our study, kidney sizes were similar in patients with normal and increased CS.

Renal parenchymal changes secondary to systemic diseases, such as DM and HT, increase renal cortex echogenicity, and this finding may provide us with an idea of renal functional impairment (20). This finding, however, does not demonstrate specificity while detecting renal involvement with limited sensitivity (21). In addition, conventional B-mode USG does not provide sufficient information on the degree of renal function abnormality (22). Studies on conventional renal USG have shown that the increase in renal echogenicity is more closely related to renal histologic parameters such as glomerular sclerosis, tubular atrophy, interstitial fibrosis, and interstitial inflammation than renal length and width, cortical or parenchymal thickness (23). However, its power to detect these histopathologic changes of renal echogenicity is very weak. Moreover, the exact cause of increased echogenicity is not clearly predicted to be fibrosis or increased inflammation, and the pathogenesis of parenchymal disease has not been clearly elucidated with increased echogenicity (24). Recently, the SWE method has been developed to detect renal parenchymal diseases and CS non-invasively. In our study, we found that the increase in renal cortical echogenicity was greater in patients with increased CS. Both stage 1 and stage 2 echogenicity increase determines the likelihood of increased CS independently and increases that likelihood 10.5% and 18.2% respectively.

Doppler USG provides information about intra-renal hemodynamic changes resulting from structural and/or functional disorders. RRI detected with Doppler USG is an objective indicator of renal tissue changing that occurs from renal vascular resistance, compliance, arteriolosclerosis, and interstitial fibrosis in both native and transplanted kidneys (25-27). RRI is used as a prognostic and diagnostic parameter for many vascular and renal diseases. An increased RRI that >0.8 in CKD with or without DM is an indicator for worsening kidney functions (28). In patients with renal artery stenosis, RRI>0.73 in the other kidney is an indication that it is more difficult to revert renal function impairment (29). An increased RRI (>0.70) in patients with primary HT is related with worse renal and cardiovascular complications (30). In addition, increased RRI is an indicator for renal transplant success and rejection. Because RRI is a hemodynamic parameter, the relationship between RRI and renal parenchymal diseases is not clearly known. The increased RRI limit value of 0.70, which we use clinically in relation to our study results, is closely related to the increased CS obtained by SWE. Our study suggests that RRI>0.70 may be used as a measure of increased renal parenchymal stiffness as well as increased renal vascular resistance. As we researched, a few studies investigated the relation between CS and RRI in patients with renal transplant (10-13). In a study involving patients with transplanted kidney, it was shown that the renal CS detected by SWE was significantly and positively correlated with the RRI and RPI determined by Doppler USG (10). In another study of patients with renal transplant, both CS and RRI were found to be associated with renal fibrosis separately, but there was no significant association between CS and RRI (13). Another study of 40 patients with renal transplant in the near future found that the RRI from the main, segmental, and interlobar arteries was closely related to CS in the early post-transplant period (11). In this study conducted by Wang et al. (11) reported that the CS increase caused by fibrosis in early stages of kidney transplantation similarly increases RRI with hemodynamic changes. With SWE, CS and related parameters have been compared in end-stage renal patients, such as renal transplant or diabetic nephropathy, and no studies have been conducted on the clinical features of patients with renal USG in everyday practice. We have found a close and independent relationship between RRI and renal CS obtained in routine renal USG examination for patients with HT, DM, coronary artery disease, who do not have kidney function impairment. As we researched, we could not find another study that shows a relationship between CS and RRI in patients with native kidney and patients that not have CKD. Clinical and prognostic significance of RRI>0.70 has been shown in previous studies and in addition to previous studies it has been shown in our study that RRI>0.70 is also associated with increased renal CS.

Histological examination with renal biopsy reveals the ongoing fibrosis clearly, but it cannot be used because of being an invasive examination. For this reason, non-invasive examinations have been preferred. Detection of microalbuminuria is a strong predictor of DM-associated nephropathy, and the urine microalbuminuria is important for the earliest diagnosis of CKD development (31). However, in addition to nephropathy, microalbuminuria is affected by DM, HT, exercise, and blood glucose levels, and it may vary by 40%-50% during the day (32). Because of that, a more objective and stable parameter is needed for the early diagnosis of patients with CKD. The SWE is a promising and non-invasive study that shows the renal elasticity or tissue stiffness objectively, and can be used for this purpose. The CS increase in nephropathy patients compared to the control group in our study may be associated with excessive hyper filtration at the nephron levels or fibrosis that occurs at cellular and mild in-

terstitial level. For this reason, increased CS may be shown with a non-invasive examination, allowing closer follow-up and more aggressive treatment in this patient group.

Our study has several important limitations. Although the number of patients was relatively adequate, more patients could get a more meaningful result. Another limitation was that the study was not conducted in a specific group of patients, and therefore no control group was formed. Patients who came to the Radiology Clinic with routine USG examination were included in this study. In addition, our study did not include patients with known renal disease and patients with eGFR<60 mL/min/1.73m^2. We cannot use our data in this group of patients. In our study, increased RRI was indirectly associated with renal CS. Increased CS due to renal parenchymal diseases as well as renal vascular and intra-renal hemodynamic parameters may be causing an RRI increase. To be able to try something clear in this regard, it is necessary to carry out studies comparing the histological changes obtained with the renal biopsy result and the RRI measurement. Another important limitation of our study is this is not a follow-up study. Especially if patients with high renal CS value were followed up in terms of nephropathy development, they could give information about the relation of high CS value to development of nephropathy in the future.

CONCLUSION

The increase in renal echogenicity and RRI obtained by conventional B-mode and Doppler USG studies with the SWE method independently identifies patients with increased CS. The presence of stage 2 renal echogenicity and RRI>0.70 found with conventional USG was considered to be an increased parameter of renal CS in cases where SWE could not be performed.

Ethics Committee Approval: Ethics committee approval was received for this study from the Ethics Committee of Adana City Health Practice and Research Center (Decision Date: 2018).

Informed Consent: Informed consent was obtained from all patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - A.S.K.; Design - A.S.K.; Supervision - H.E.S.; Writing Manuscript - A.S.K., H.E.S.; Critical Review - H.E.S.

Conflict of Interest: No conflict of interest was declared by the author.

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REFERENCES

1. Gross JL, de Azevedo MJ, Silveiro SP, Canani LH, Caramori ML, Zelmanovitz T. Diabetic nephropathy: diagnosis, prevention, and treatment. Diabetes Care 2005; 28: 164-76.
2. KDOQI Clinical Practice Guidelines and Clinical Practice Recommendations for Diabetes and Chronic Kidney Disease. Am J Kidney Dis 2007; 49(2 Suppl 2): S12-S154.
3. Sicari R, Gargani L, Wiecek A, Covic A, Goldsmith D, Suleymanlar G, et al. The use of echocardiography in observational clinical trials: the EURECA-m registry. Nephrol Dial Transplant 2013; 28: 19-23.
4. Zaffanello M, Piacentini G, Bruno C, Brugnara M, Fanos V. Renal elasticity quantifcation by acoustic radiation force impulse applied to the evaluation of kidney diseases: a review. J Investig Med 2015; 63: 605-12.
5. Samir AE, Allegretti AS, Zhu Q, Dhyani M, Anvari A, Sullivan DA, et al. Shear wave elastography in chronic kidney disease: a pilot experience in native kidneys. BMC Nephrol 2015; 116: 119.
6. Goya C, Kilinc F, Hamidi C, Yavuz A, Yildirim Y, Cetincakmak MG, et al. Acoustic radiation force impulse imaging for evaluation of renal parenchyma elasticity in diabetic nephropathy. Am J Roentgenol 2015; 204: 324-9.
7. Hassan K, Loberant N, Abbas N, Fadi H, Shadia H, Khazim K. Shear wave elastography imaging for assessing the chronic pathologic changes in advanced diabetic kidney disease. Ther Clin Risk Manag 2016; 12: 1615-22.
8. Stegall MD, Park WD, Larson TS, Gloor JM, Cornell LD, Sethi S, et al. The histology of solitary renal allografts at 1 and 5 years after transplantation. Am J Transplant 2010; 11: 698-707.
9. Clevert DA, Stock K, Klein B, Slotta-Huspenina J, Prantl L, Heemann U, et al. Evaluation of Acoustic Radiation Force Impulse (ARFI) imaging and contrast-enhanced ultrasound in renal tumors of unknown etiology in comparison to histological findings. Clin Hemorheol Microcirc 2009; 43: 95-107.
10. Ozkan F, Yavuz YC, Inci MF, Altunoluk B, Ozcan N, Yuksel M, et al. Interobserver variability of ultrasound elastography in transplant kidneys: correlations with clinical-Doppler parameters. Ultrasound Med Biol 2013; 39: 4-9.
11. Wang HK, Lai YC, Lin YH, Chiou HJ, Chou YH. Acoustic Radiation Force Impulse Imaging of the Transplant Kidney: Correlation Between Cortical Stiffness and Arterial Resistance in Early Post-transplant Period. Transplant Proc 2017; 49: 1001-4.
12. Arndt R, Schmidt S, Loddenkemper C, Grünbaum M, Zidek W, van der Giet M, et al. Noninvasive evaluation of renal allograft fibrosis by transient elastographyda pilot study. Transpl Int 2010; 23: 871-7.
13. Stock KF, Klein BS, Vo Cong MT, Sarkar O, Römisch M, Regenbogen C, et al. ARFI-based tissue elasticity quantification in comparison to histology for the diagnosis of renal transplant fibrosis. Clin Hemorheol Microcirc 2010; 46: 139-48.
14. Guo LH, Xu HX, Fu HJ, Peng A, Zhang YF, Liu LN. Acoustic radiation force impulse imaging for noninvasive evaluation of renal parenchyma elasticity: preliminary findings. PLoS One 2013; 8: e68925.
15. He WY, Jin YJ, Wang WP, Li CL, Ji ZB, Yang C. Tissue elasticity quantification by acoustic radiation force impulse for the assessment of renal allograft function. Ultrasound Med Biol 2014; 40: 322-9.
16. Bob F, Grosu I, Sporea I, Bota S, Popescu A, Sirli R, et al. Is there a correlation between kidney shear wave velocity measured with VTQ and histological parameters in patients with chronic glomerulonephritis? A pilot study. Med Ultrason 2018; 1: 27-31.
17. Stock KF, Klein BS, Cong MT, Regenbogen C, Kemmner S, Büttner M, et al. ARFI-based tissue elasticity quantifcation and kidney graft dysfunction: frst clinical experiences. Clin Hemorheol Microcirc 2011; 49: 527-35.
18. Cui G, Yang Z, Zhang W, Li B, Sun F, Xu C, et al. Evaluation of acoustic radiation force impulse imaging for the clinicopathological typing of renal fbrosis. Exp Ther Med 2014; 7: 233-5.
19. Singh H, Panta OB, Khanal U, Ghimire RK. Renal Cortical Elastography: Normal Values and Variations. J Med Ultrasound 2017; 25: 215-20.
20. Neıman HL. The urinary system. In: Textbook of abdominal ultrasound, p. 330. Edited by B. B. Goldberg. Williams & Wilkins, Baltimore 1993.
21. Platt JF, Rubin JM, Bowerman RA, Marn CS. The inability to detect kidney disease on the basis of echogenicity. Am J Roentgenol 1988; 151: 317-9.

22. Soldo D, Brkljacic B, Bozikov V, Drinkovic I, Hauser M. Diabetic nephropathy. Comparison of conventional and duplex Doppler ultrasonographic findings. Acta Radiol 1997; 38: 296-302.
23. Moghazi S, Jones E, Schroepple J, Arya K, McClellan W, Hennigar RA, et al. Correlation of renal histopathology with sonographic findings. Kidney Int 2005; 67: 1515-20.
24. Lee YS, Lee MJ, Kim MJ, Im YJ, Kim SW, Lim NL, et al. Is Increased Echogenicity Related to a Decrease in Glomerular Filtration Rate? Objective Measurements in Pediatric Solitary Kidney Patients--A Retrospective Analysis. PLoS One 2015; 10: e0133577.
25. Bude RO, Rubin JM. Effect of downstream cross-sectional area of an arterial bed on the resistive index and the early systolic acceleration. Radiology 1999; 212: 732-8.
26. Brkljacić B, Mrzljak V, Drinković I, Soldo D, Sabljar-Matovinović M, Hebrang A. Renal vascular resistance in diabetic nephropathy: duplex Doppler US evaluation. Radiology 1994; 192: 549-54.
27. Kim SH, Kim SM, Lee HK, Kim S, Lee JS, Han MC. Diabetic nephropathy: duplex Doppler ultrasound findings. Diabetes Res Clin Pract 1992; 18: 75-81.
28. Radermacher J, Ellis S, Haller H. Renal resistance index and progression of renal disease. Hypertension 2002; 39: 699-703.
29. Bruno RM, Daghini E, Versari D, Sgrò M, Sanna M, Venturini L, et al. Predictive role of renal resistive index for clinical outcome after revascularization in hypertensive patients with atherosclerotic renal artery stenosis: a monocentric observational study. Cardiovasc Ultrasound 2014; 20: 12-9.
30. Doi Y, Iwashima Y, Yoshihara F, Kamide K, Hayashi S, Kubota Y, et al. Renal resistive index and cardiovascular and renal outcomes in essential hypertension. Hypertension 2012; 60: 770-7.
31. Mogensen CE, Schmitz O. The diabetic kidney: from hyperfiltration and microalbuminuria to end-stage renal failure. Med Clin North Am 1988; 72: 1465-92.
32. Schmitz A, Vaeth M, Mogensen CE. Systolic blood pressure relates to the rate of progression of albuminuria in NIDDM. Diabetologia 1994; 37: 1251-8.

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