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Original Article *Özgün Araştırma*

# QUANTITATIVE ASSESSMENT OF RENAL STEATOSIS AND ITS RELATIONSHIP WITH CLINICAL STAGE IN CHRONIC RENAL FAILURE USING CHEMICAL SHIFT MRI

## *Kronik Böbrek Hastalığında Renal Steatozun ve Klinik Evre ile İlişkisinin Kimyasal Şift MRG ile Kantitatif Olarak Değerlendirilmesi*

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## ABSTRACT

**Objective**: Quantitative measurement of renal parenchymal lipid accumulation in chronic renal disease using chemical shift magnetic resonance imaging and evaluation of its relationship with clinical stages.

**Material and Methods:** In this retrospective study, the groups were designed as in chronic renal disease (n=46), diabetes without chronic renal disease (n=31), and control (n=59). Chronic renal disease group also divided into two subgroups as diabetic chronic renal disease (n=25) and non-diabetic chronic renal disease (n=21). A total of 272 kidneys of 136 patients were evaluated. Chronic renal disease clinical staging was performed according to e-GFR values. All magnetic resonance imaging examinations were performed with a 1.5 Tesla device. Chemical shift imaging was used in this study to quantify fat in the renal parenchyma (in-phase, out-of-phase, Dixon-water and Dixon-fat). Measurements were made from kidney and spleen by two different methods as whole parenchyma (first method) and focal parenchymal (second method). The fat fraction, and spleen-to-renal chemical shift imaging ratio were calculated.

**Results**: In the control group, parenchymal fat fraction according to first and second measurement methods were calculated as 0.05±0.01 and 0.05 ± 0.02, respectively. In the chronic renal disease groups, fat fraction measurements were 0.07±0.02 and 0.07±0.04, respectively, and they were found to be significantly higher from chronic renal disease stage 3 compared to the control group ($p<0.001$). No significant difference was observed in fat fraction and spleen-to-renal chemical shift imaging ratio values in diabetes patients ($p>0.05$).

**Conclusion:** In chronic renal disease, starting from stage 3, there is a significant renal parenchymal lipid accumulation compared to the control group and diabetic patients.

***Keywords**: Magnetic resonance imaging, chronic renal disease, diabetes mellitus*

## ÖZ

**Amaç**: Kronik böbrek hastalığında renal parankimal lipid birikiminin kantitatif olarak kimyasal şift manyetik rezonans görüntüleme ile ölçülmesi ve klinik evreler ile ilişkisinin değerlendirilmesi amaçlandı

**Gereç ve Yöntemler**: Bu retrospektif çalışmada gruplar kronik böbrek hastalığı (n=46), kronik böbrek hastalığı olmayan diyabet hastaları (n=31) ve kontrol (n=59) olarak tasarlandı. Kronik böbrek hastalığı grubu kendi içinde diyabetik kronik böbrek hastalığı (n=25) ve non-diyabetik kronik böbrek hastalığı (n=21) olarak iki alt gruba ayrıldı. Yüz otuz altı hastanın toplam 272 böbreği değerlendirildi. Kronik böbrek yetmezliği klinik evrelemesi e-GFR değerlerine göre yapıldı. Tüm manyetik rezonans incelemeleri 1.5 Tesla cihazla yapıldı. Böbrek parankimindeki yağ miktarını ölçmek için, kimyasal şift görüntüleme (Faz içi, faz dışı, Dixon-su ve Dixon-yağ) sekansları kullanıldı. Böbrek ve dalaktan, tüm parankim (Birinci yöntem) ve fokal parankimal (İkinci yöntem) olmak üzere, iki farklı yöntemle ölçümler yapıldı. Yağ fraksiyonu ve dalak-böbrek kimyasal şift görüntüleme oranı hesaplandı.

**Bulgular**: Birinci ve ikinci ölçüm yöntemlerine göre, kontrol grubunda parankimal yağ fraksiyonu değerleri sırasıyla 0.05±0.01 ve 0.05±0.02 olarak hesaplandı. Kronik böbrek hastalığı gruplarında ise yağ fraksiyonu ölçümleri sırasıyla 0.07±0.02 ve 0.07±0.04 olup, kontrol grubuna göre kronik böbrek hastalığı Evre 3'ten itibaren anlamlı yüksek bulundu ($p<0.001$). Diyabet hastalarında yağ fraksiyonu ve dalak-böbrek kimyasal şift görüntüleme oranı değerlerinde anlamlı farklılık gözlenmedi ($p>0.05$).

**Sonuç**: Kronik böbrek hastalığında renal parankimde evre 3'ten itibaren, kontrol grubu ve diyabetik hastalara göre anlamlı lipid birikimi olmaktadır.

***Anahtar Kelimeler**: Manyetik rezonans görüntüleme, kronik böbek hastalığı, diabetes mellitus*

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## INTRODUCTION

The kidneys have a low-fat content, and the triglyceride content accounts for approximately 0.6% of the renal weight (1, 2). Abnormal accumulation of triglycerides in the kidney has been described as renal steatosis, with a suggested etiology of obesity, metabolic syndrome, hypertension, and diabetes mellitus (DM) (2-4). Renal steatosis may cause the development and/or progression of chronic kidney disease (CRD) (4, 5). Renal fat accumulation is pararenal, perirenal, renal sinus, and parenchymal (6). Renal fat accumulation is thought to lead to hemodynamic changes and increased intraglomerular pressure in the renal arteries by mechanical pressure. This leads to the development of hypertension and/or renal parenchymal damage (7). The term, “Fatty Kidney Disease”, has been proposed to express the local and systemic effects of ectopic fat accumulation in the kidney. Thus, as in non-alcoholic fatty liver disease (NAFLD), it has been reported that renal steatosis can be treated as a separate disease and the necessary studies for the treatment can be conducted more effectively (8).

The gold standard assessment method of renal steatosis is a biopsy and quantitative enzymatic measurement of triglycerides and qualitative "oil red O" staining. However, these methods are invasive and may lead to an increased risk of complications (2). Non-invasive measurement of the lipid amount is also possible with fatty tissue sensitive magnetic resonance imaging (MRI) methods (9). Chemical shift imaging (CSI) is a MRI technique used to detect small areas (voxels) that contain both water and fat protons. This technique makes use of the difference in resonance frequencies of water and fat protons. Images are obtained at the in-phase (IP) and the out-of-phase (OP) times of the water and fat protons. In areas containing both water and fat, there will be a loss of signal in OP images. In areas containing only fat protons and areas containing only water protons, there will be no signal difference between the IP and OP images (10, 11).With the simple addition and subtraction of the two images, an image containing only water (Dixon Water) and an image containing only fat (Dixon Fat) are obtained. With Dixon-based CSI, it is possible to show microscopic (intracellular) fat presence and measure the amount of fat. Moreover, CSI is unaffected by underlying fibrosis (12). A few MRI studies have shown that normal human kidneys contain low amounts of lipids (Fat Fraction= 0.4-0.7%) (9,13). Although it has been reported in the literature that renal steatosis may lead to the development and/or progression of CRD, data are lacking on this subject (4,5,14). This study aimed to quantitatively evaluate renal parenchymal lipid accumulation and its relationship with clinical stages using CSI in CRD subjects. It was also investigated whether diabetes contributed to renal steatosis in CRD.

## MATERIALS AND METHODS

*Study Population*

In retrospective archive search, it is not obligatory to obtain patient consent therefore all procedures in the study were carried out in accordance with the Declaration of Helsinki. Approval for the study was obtained from the local ethics committee (Süleyman Demirel University University Clinical Research Ethics Committee, date: 27.02.2020, issue number: 2020/66).

This retrospective study was conducted on individuals registered in our hospital's picture archiving and communication system (PACS) between 2015-2018. A total of 272 kidneys of 136 patients older than 18 years of age, consisting of 46 CRD patients, 31 diabetes who had not yet developed CRD (microalbuminuria <30 mg/dl), and 59 control individuals were evaluated (15). According to the information recorded in PACS, CRD patients were divided into two groups as diabetic (dCRD) (n=25) and non-diabetic CRD (non-dCRD) (n=21). CRD was staged from 1 to 5 according to e-GFR values (16). In addition, to evaluate the effect of chronic illness duration on parenchymal steatosis, CRD patients were divided into three groups (<5 years, 5-10 years and

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>10 years) according to the duration of the disease. The control group consisted of individuals who did not have diabetes, urinary disease, malignancy or any chronic disease, but had upper abdominal MRI recorded in PACS. Cancer patients and pregnant women were not included in the study.

The power analysis of the study was performed by PASS 13.0.6 software (NCSS, LLC, 2014). The sample size was determined by Two-way Repeated Measure ANOVA. The mean and standard deviations of chemical shift imaging ratio (SRR) and fat fraction (FF) measurements for two types of methods, determined in a preliminary study, were used to calculate the effect size. The effect size values were determined as 1.063 for SRR, and 2.391 for FF measurements in the F test. The sample size was determined as 20 for each groups (dCRD, non-dCRD and Control) for smaller effect size. Therefore, the overall sample size was determined as 60 for two measurement methods considering the power as 90% (actual power 91.2%) and the type-I error 5%.

*MR Examination Parameters*

MRI examinations were performed using a 1.5-T MRI system (Magnetom Avanto, Siemens Healthcare, Erlangen, Germany), with a 16-channel "body coil", with the patient in a supine position without the need for sedation. Axial and coronal plane turbo spin-echo T2-weighted (T2W) without fat saturation (FS) and a breath-hold, gradient-echo sequence FS T1 volumetric interpolated breath-hold examination (VIBE) images based on the Dixon two-point method were acquired IP and OP to detect intracellular lipid. The chemical shift MRI examination parameters are presented in Table 1.

**Table 1**: Magnetic resonance examination parameters.

| | **In phase (IP)** | **Out of phase (OP)** | **Dixon Water (DW)** | **Dixon Fat (DF)** |
|---|---|---|---|---|
| Voxel size (mm) | 0.6 x 0.6 x 3 | 0.6 x 0.6 x 3 | 0.6 x 0.6 x 3 | 0.6 x 0.6 x 3 |
| FOV (mm) | 261 x 380 | 261 x 380 | 261 x 380 | 261 x 380 |
| Matrix size (mm) | 288 x 320 | 288 x 320 | 288 x 320 | 288 x 320 |
| TR (ms) | 7.08 | 7.08 | 7.1 | 7.1 |
| TE (ms) | 2.39 | 4.77 | 2.4 | 2.4 |
| Flip angle (°) | 10 | 10 | 10 | 10 |
| Bandwidth (Hz/Px) | 490 | 490 | 490 | 490 |
| Slice thickness (mm) | 3 | 3 | 3 | 3 |
| Next | 1 | 1 | 1 | 1 |
| Aquisition time (s) | 16 | 16 | 16 | 16 |
| Slice gap (mm) | 20 | 20 | 20 | 20 |

FOV: Field of view TR: Repetition time, TE: echo time

*Evaluation of Images*

Abdominal MRI images were evaluated on a clinical workstation (iMac Pro retina, Apple) with an Osirix DICOM Viewer by a single observer (AS, 3 years of abdominal MRI experience). The observer evaluated the images in a single session, unaware of the clinical and laboratory information. Morphological evaluation of the area to be measured in terms of an artifact, cyst, mass foreign body, and vascular structure was performed in both kidney T2W axial MRI sections. Measurements were made in the axial plane by two different methods, from the most suitable single section, which passes through the renal hilus level and does not contain any space-occupying lesions. In the first method, the region

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of interest (ROI) was drawn by hand to cover the entire renal parenchyma. In the second method, three measurements were taken and averaged, using a 30 $mm^2$ circular ROI from the areas where the corticomedullary junction was best tracked. The same ROI methods were used for spleen measurements. Renal parenchyma measurements were made in IP, OP, Dixon Fat (DF), Dixon Water (DW) sequences (Figure 1). The spleen-to-renal ratio (SRR) was modified from the CSI-Ratio formula used for adrenal adenoma detection. The SRR and renal FF values from these measurements were calculated with the following formulas:

$$SRR = \frac{\text{Renal SI}_{OP}/\text{Spleen SI}_{OP}}{\text{Renal SI}_{IP}/\text{ Spleen SI}_{IP}} \quad (17). \qquad FF = DF / DF + DW \quad (10).$$

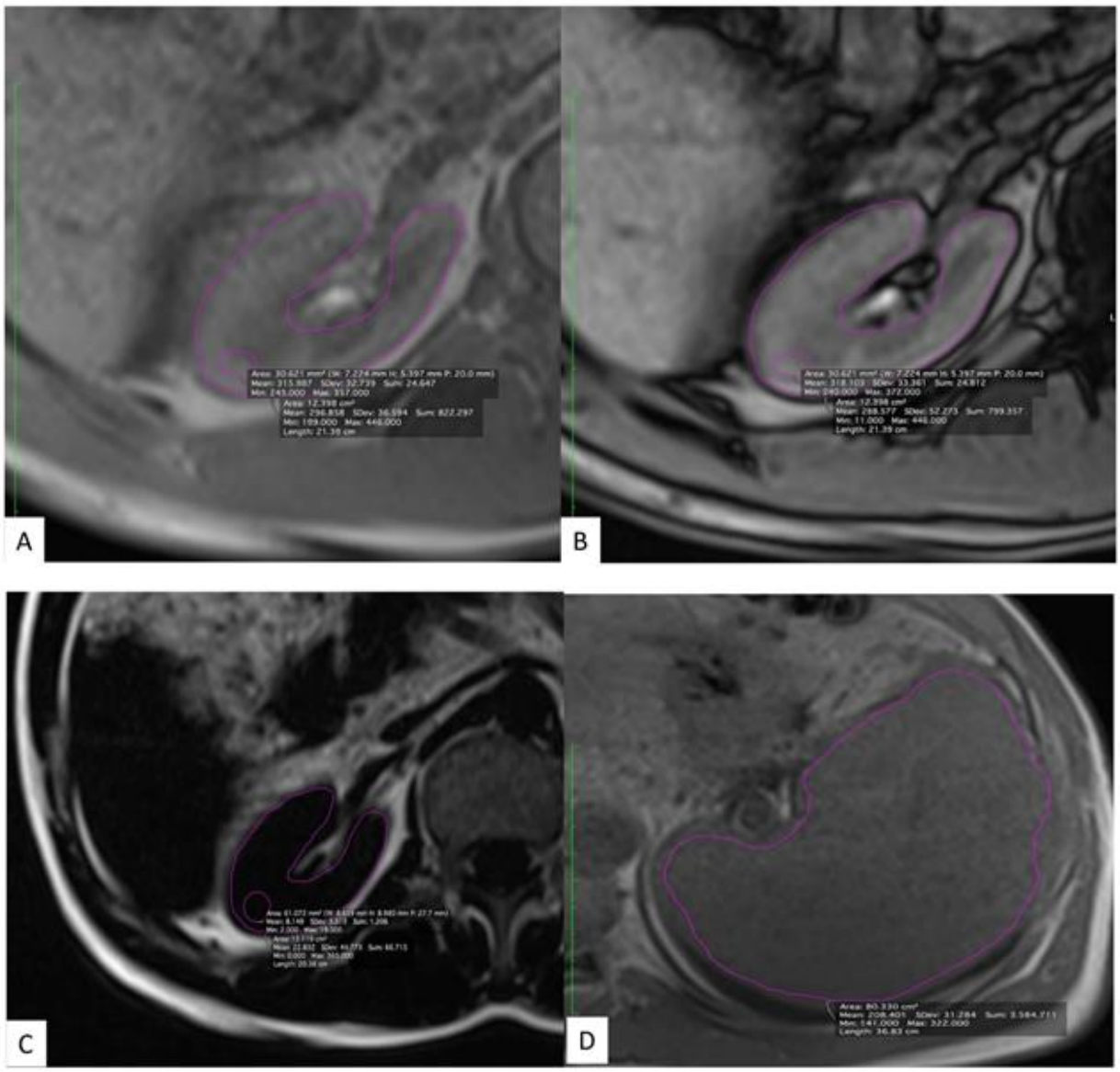


**Figure 1**: Measurement methods from the renal parenchyma are shown. In the first method, the region of interest (ROI) was drawn by hand to cover the entire renal parenchyma. In the second method, three measurements were taken and averaged, using a 30 $mm^2$ circular ROI from the areas where the corticomedullary junction was best tracked. Measurements were made in In Phase (A), Out of Phase (B), Dixon Fat (C), Dixon Water (not shown) sequences. The same ROI methods were used for spleen measurements (D).

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*Statistical Analyses*

The statistical analyses of the study were performed using SPSS vn. 20.0 software (IBM Inc, Chicago, IL, USA). Descriptive statistics were presented as mean±standard deviation (SD) values and frequency (percentage). The Student's t-test was used in the comparison of two independent groups, and One Way ANOVA for multiple groups with Tukey HSD post-hoc test since the distribution of the variables was normal. ROC analysis was performed to predict the diagnostic values. Relationships between the variables were examined with Pearson correlation analysis. A value of $p<0.05$ was considered a statistically significant result in all analyses with a 5% type-I error.

## RESULTS

In the study, 272 kidneys of 136 adults were evaluated. The mean age of CRD, diabetes, and control groups were 61.12±11.24, 45.44±14.23, and 53.11±12.23 years, respectively. Gender ratios were equal. There was no effect of gender difference on measurement values. Disease duration was <5 years in 39% of the CRD patients, 5-10 years in 23%, and >10 years in 37%.

The renal FF and SRR values of both measurement methods in the study groups are presented in Table 2. Fat fraction values were significantly higher in the whole CRD group compared to diabetes and control groups ($p<0.001$). The FF measurement did not show a significant difference between diabetes and the control group.

**Table 2**: Comparison of measurements between study groups.

| | **Splenorenal CSI Ratio (SRR)** | | **Fat Fraction (FF)** | |
|---|---|---|---|---|
| | 1st Method | 2nd Method | 1st Method | 2nd Method |
| | (mean±SD) | | | |
| All CRD | 1.02±0.18 | 1.02±0.18 | 0.07±0.02 | 0.07±0.04 |
| Control | 1.11±0.7 | 1.06±0.08 | 0.05±0.01 | 0.05±0.02 |
| *p* | 0.21 | 0.086 | *<0.001** | *<0.001** |
| Diabetes | 1.02±0.07 | 1.06±0.10 | 0.05±0.01 | 0.05±0.02 |
| Non-dCRD | 1.03±0.17 | 1.03±0.16 | 0.08±0.03 | 0.08±0.05 |
| *p* | 0.935 | 0.291 | *<0.001** | *0.004** |
| Non-dCRD | 1.03±0.17 | 1.03±0.16 | 0.08±0.03 | 0.08±0.05 |
| dCRD | 1.01±0.20 | 1.03±0.18 | 0.07±0.02 | 0.07±0.03 |
| *p* | 0.692 | 0.919 | 0.38 | 0.227 |
| Diabetes | 1.02±0.07 | 1.06±0.10 | 0.05±0.01 | 0.05±0.02 |
| Control | 1.11±0.7 | 1.06±0.07 | 0.05±0.01 | 0.05±0.02 |
| *p* | 0.098 | 0.9 | 0.232 | 0.101 |
| Diabetes | 1.02±0.07 | 1.06±0.10 | 0.05±0.01 | 0.05±0.02 |
| dCRD | 1.01±0.20 | 1.03±0.18 | 0.07±0.02 | 0.07±0.03 |
| *p* | 0.654 | 0.258 | *<0.001** | *0.012** |

*: significant at $p<0.05$ level according to Independent Sample t-test. mean±SD

All-CRD: All chronic renal disease; Non-dCRD: non-diabetic chronic renal disease; dCRD: diabetic chronic renal disease; FF: Fat Fraction.

From the 3rd stage of CRD, parenchymal FF values were found to be significantly higher in both measurement methods (p<0.001) (Table 3, Figure 2).

In respect of the SRR values, there was no statistically significant difference between the control, CRD, and diabetes groups. It was observed that there was a tendency for the SRR measurement values to decrease in all patient groups, which was more pronounced in diabetic patients in the first method (p=0.098).

The measurement methods were compared in each group (Table 4). FF measurements according to both methods showed a significant difference in all groups. In the first method, FF measurements were lower in controls and diabetes, however, in the non-dCRD group, the FF value was higher in the first method than in the second method (p<0.001). With the first measurement method, SRR values were significantly lower in patient groups (p<0.001). For SRR in the control group, no significant difference was observed between the two measurement methods.

According to the duration of the CRD, there was only a significant increase in SRR values (p=0.038), and a tendency to increase was observed in FF values (p=0.082).

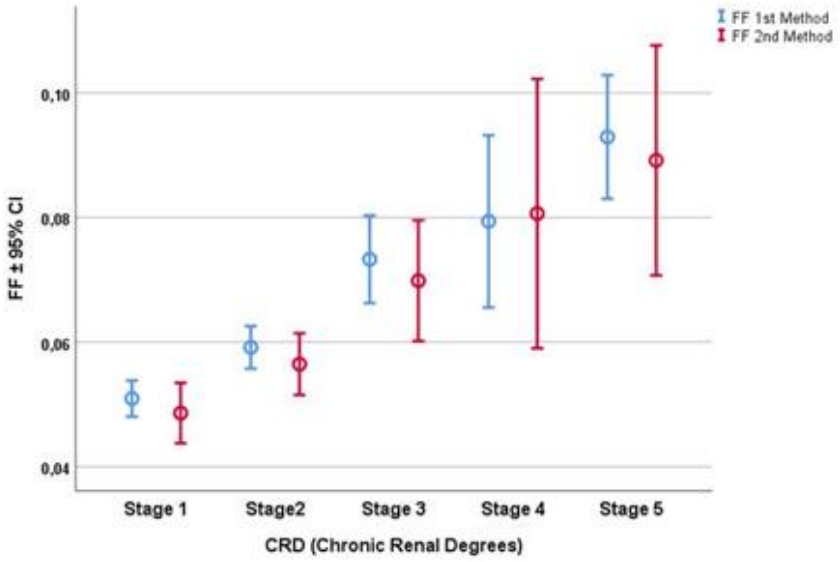


**Figure 2**: Error bar graph of renal parenchyma FF values on CRD stages (FF: Fat fraction, CRD: Chronic renal disease).

**Table 3**: Comparison of fat fraction (FF) measurements with clinical stages of CRD.

| (n=272) Stage | 1st Method (mean±SD) | 2nd Method (mean±SD) |
|---|---|---|
| 1 (84) | 0.051±0.013 a.b.c | 0.048±0.022 a.b.c |
| 2 (82) | 0.059±0.015 d.e.f | 0.056±0.022 d.e |
| 3 (65) | 0.073±0.028 a.d.g | 0.069±0.038 a |
| 4 (19) | 0.079±0.025 b.e | 0.081±0.041 b.d |
| 5 (22) | 0.092±0.023 c.f.g | 0.089±0.043 c.e |
| *p* | *<0.001** | *<0.001** |

*: significant at 0.05 level according to ANOVA. a, b, c, d, e, f, g: same superscript letters denote significant pairwise comparisons according to Tukey HSD test. CRD: Chronic renal failure

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**Table 4**: Comparison of measurement methods in groups.

| | **1st Method** (mean±SD) | **2nd Method** (mean±SD) | **p** |
|---|---|---|---|
| Control (n=118) | | | |
| SRI Ratio | 1.16 ± 0.86 | 1.06 ± 0.07 | *0.310* |
| FF | 0.05 ± 0.01 | 0.05 ± 0.02 | *<0.001** |
| Diabetes (n=62) | | | |
| SRI Ratio | 1.02 ± 0.07 | 1.06 ± 0.10 | *<0.001** |
| FF | 0.05 ± 0.01 | 0.05 ± 0.02 | *<0.001** |
| CRD (n=92) | | | |
| SRI Ratio | 1.03 ± 0.17 | 1.03 ± 0.16 | *<0.001** |
| FF | 0.08 ± 0.03 | 0.08 ± 0.05 | *<0.001** |

*: *significant at p<0.05 level according to Paired Sample t-test*
CRD: *Chronic renal failure.* FF: *Fat Fraction.* SRR*: The spleen-to-renal ratio*

## DISCUSSION

The results obtained from this study showed that there was statistically significant lipid accumulation in the renal parenchyma of CRD patients from stage 3 onwards. There was no significant increase in FF values in diabetes compared to the control group. Moreover, there was no significant difference between dCRD and non-dCRD FF values. These findings suggest that diabetes does not significantly contribute to renal steatosis in patients with CRD.

Kidney lipotoxicity and its role in the pathogenesis of kidney disease are not fully understood (4). Different results have been reported in a limited number of studies investigating the relationship between obesity and renal parenchymal fat accumulation (13,18-22). Using a 3T device, Yokoo et al. found that the FF value (2.38%) was high in diabetic patients, independent of serum creatinine, BMI, and HbA1c (9). Renal FF was also determined as 0.82% in the control group and this was stated to be quite close to the results reported in the literature (FF=0.6%). In this study, the absence of a significant difference in FF values between the diabetic group and the control group was incompatible with the high FF rate stated by Yokoo et al. This disagreement between our study and Yokoo et al. study may be due to differences in the magnetic field strength of the device used, measurement techniques, difference of the detalied parameters such as duration, severity, and age of onset of diabetes, and the number of patients that make up the groups. In a recent study, diabetes without marked renal failure was reported to have increased extra parenchymal renal lipid accumulation and increased renal vascular resistance (7). When these results are evaluated together with those of the current study, the need for more studies to be able to understand to what extent lipid accumulation in the parenchymal and extra parenchymal compartments causes renal lipotoxicity in diabetes is evident.

In this study, to measure renal intracellular fat accumulation, the adrenal-spleen CSI ratio used in the diagnosis of adrenal adenomas was modified as the spleen-renal CSI ratio (SRR). To the best of our knowledge, this is the first use of this approach in the literature. In the renal parenchyma SRR results, there was no significant difference between the control and patient groups. However, a statistically significant increase was found in the SRR value in those with advanced disease duration (>10 years). In other words, it may be said that the SRR measurement is inadequate compared to Dixon sequences (DW and DF) in determining the amount of renal parenchymal lipid, which is normally extremely low. However, since the

duration of diabetes were not evaluated in this study, it may be misleading to draw such a conclusion for the SRR. Therefore, controlled studies with large numbers of subjects according to their clinical stages are needed. The results of the measurement methods were compared. In the first method using ROI containing the entire parenchyma, SRR values were lower and FF measurements were higher compared to the second method using focal ROI. While stage 2 could be distinguished from stage 3 by the first measurement method, this distinction could not be made according to FF values in the second method. Since the first measurement method includes a larger sample area to represent the entire parenchyma, it can be thought that it gives results closer to the mean renal parenchyma values. In addition, although the measurement results are very close to each other, the statistically significant difference can be explained by the very low amount of lipid in the normal parenchyma. That is, even a slight increase in the amount of renal parenchymal lipid can be detected due to the high sensitivity of chemical shift imaging. Therefore, it may be possible to accurately determine the clinical staging of CRD with MRI. In this regard, multicenter studies are needed to determine FF cut-off values according to clinical stages and test intra-observer and interobserver reliability.

This study had some important limitations: 1) Histopathological results of the kidney were not obtained as a reference standard. 2) The single measurement by the single observer raises doubts about the reliability of the measurements. 3) Other parameters that may affect renal steatoses, such as obesity, hypertension, and metabolic syndrome were not included in the study, so it was not possible to evaluate the steatosis status of CRD alone. 4) As it was a retrospective study, the relationship between the duration and severity of the disease and renal steatosis could not be evaluated, and the diabetic patients could not be categorized according to their clinical status in detail.

In conclusion, even trace amounts of renal parenchymal lipid accumulation can be quantitatively measured by chemical shift MRI. In patients with CRD, there was a significant increase in parenchymal lipid accumulation from stage 3 onwards. Further studies can be performed using chemical shift MRI in large patient groups; to evaluate the relationship between CRD and renal steatosis, disease stages, duration, and possible etiological factor parameters. Thus, we believe that the concept of renal steatosis will be developed and new therapeutic approach studies will be pioneered.

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