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DCE MRI Measurement of Renal Function in Patients Undergoing Partial Nephrectomy: Preliminary Experience

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Abstract

Objectives—To evaluate changes in single-kidney glomerular filtration rate (SK-GFR) using low-dose dynamic contrast-enhanced magnetic resonance renography (MRR) in patients undergoing partial nephrectomy for renal masses.

Materials and Methods—In this Health Information Patient Protection Act -compliant prospective study, 18 patients with renal masses underwent preoperative MR imaging at 1.5 T for renal mass evaluation and low-dose gadolinium-enhanced MRR. MRR was repeated approximately 48–72 hours and 6 months after partial nephrectomy. SK-GFR was calculated from MRR images, and right and left kidney values summed for total MR-GFR. Postoperative changes in SK-GFR and MR-GFR were compared with changes in eGFR calculated using modification of diet in renal disease (MDRD) formula, renal lesion characteristics, and ischemia type (warm versus cold), and ischemia time.

Results—A decrease in the operated kidney SK-GFR was seen in 15 of 18 patients, with a mean loss of 31% ($\pm$ 23%), while eGFR decreased in 13 of 18 patients with mean decrease of 19% ($\pm$ 14%). Decrease in SK-GFR was greatest in patients with warm ischemia time greater than 40 minutes and least in patients with cold ischemia. In the immediate-postoperative period, 6 of 7 (86%) patients with pre-operative MR-GFR less than 60 mL/min/1.73 m² failed to demonstrate compensatory increase in SK-GFR in the non-operated kidney, while 5 of 11 patients with baseline MR-GFR over 60 mL/min/1.73 m² showed compensatory increase in non-operated kidney SK-GFR.

Conclusion—MRR can demonstrate functional loss in the operated kidney and compensatory increase in the function of the contralateral kidney, thus enabling evaluation of various surgical techniques on kidney function.

Keywords

MR Renography; Functional Renal MRI; Partial nephrectomy

Introduction

Most renal masses are discovered incidentally on imaging studies. Early stage neoplasms (T1a < 4 cm in size) now account for 70% of all newly diagnosed renal cancers (1, 2). Partial nephrectomy (PN) has been shown to provide equivalent oncologic control to radical nephrectomy (RN) for stage T1a tumors while significantly reducing the risk of chronic kidney disease (CKD) and non-oncologic morbidity and mortality (3–5). Preserving renal function in these patients is important as most patients with renal neoplasms present in the 6th and 7th decades with medical comorbidities, and up to 30% of elderly patients have underlying CKD despite normal serum creatinine (6). Preoperative CKD contributes to worsened outcomes in renal function and overall mortality (5).

Despite the advantages of PN over RN in preserving renal parenchyma, up to 50% of patients undergoing PN will ultimately develop CKD over time (5). A variety of non-modifiable factors predict worsened kidney functional following surgery: advanced age, baseline kidney function, and tumor location and size. However, there are modifiable factors, such as surgical technique, which may impact kidney function following surgery.

Since the kidneys receive nearly 25% of the body's cardiac output, PN frequently requires clamping of the renal vasculature for safe tumor excision, resulting in temporary ischemia. Ischemia time during PN has been shown to play a role in post-operative kidney functional outcomes (7). It is also widely believed, though not well studied, that applying renal hypothermia (cold ischemia) allows for longer safe ischemia times and improves kidney functional outcomes. Currently, cold ischemia can only be performed with open PN as laparoscopic hypothermia techniques have not been sufficiently developed for routine clinical use.

The impact of PN ischemia time and type on kidney functional is poorly understood due to lack of available tools for accurate assessment of single kidney function (SK-GFR). Serum creatinine and creatinine-based formulas of eGFR assess global kidney function and thus are incapable of discerning changes in the operated kidney in patients with two functioning kidneys. Inulin clearance is considered the most accurate measure of SK-GFR, but is invasive and time-consuming. Tc 99m-diethylenetriamine-pentaacetic acid (Tc 99m-DTPA) plasma clearance combined with scintigraphy has also been used as a reference standard for measuring SK-GFR, but is clinically impractical for routine use due to radiotracer injections, blood sampling, potentially long acquisition time, and minimal provided anatomic information (8–10).

Dynamic contrast enhanced (DCE) magnetic resonance renography (MRR) has been shown to reliably estimate individual kidney GFR with a low dose (4 mL) of gadolinium contrast agent (11–13). MRR can be performed as an adjunct to routine renal MRI both pre and post-operatively and help evaluate the impact of surgical technique on kidney functional outcomes. In this prospective study, our purpose was to demonstrate feasibility and evaluate changes in single-kidney glomerular filtration rate (SK-GFR) using low-dose dynamic contrast-enhanced magnetic resonance renography (MRR) in patients undergoing partial nephrectomy for renal masses.

Materials and Methods

Patients

Written informed consent was obtained from all patients in this Health Insurance Portability and Accountability Act-compliant, institutional review board-approved study. Between October 2009 and December 2011, 31 consecutive patients with renal masses under

consideration for partial nephrectomy were referred from the Urology division at our institution for preoperative renal mass MR imaging. We excluded one patient whose MRI demonstrated no renal mass, nine patients who underwent surgery outside our institution, and three patients who required radical nephrectomy. Eighteen patients were included in the final study cohort (nine men (mean age 59.0 yrs, range 43 – 73); nine women (mean age 53.1 yrs, range 33–72), overall mean age 56.0 yrs, range 33 – 73).

MR Imaging

All MR imaging was performed at 1.5 T (Magnetom Avanto; Siemens Medical Solutions, Erlangen, Germany). Patients underwent preoperative clinical MR imaging for renal mass evaluation with addition of MRR. Postoperative MRR examinations were performed at 48–72 hours after surgery and six months after surgery. The standard preoperative clinical protocol for renal mass evaluation included coronal and axial T2-weighted half-Fourier single-shot turbo spin-echo (HASTE), diffusion weighted imaging, axial T1-weighted gradient echo in- and opposed-phase, and axial 3D T1-weighted fat suppressed gradient echo, volume interpolated breath-hold exam (VIBE) pre- and post-contrast acquisition in the corticomedullary, nephrographic, and excretory phases of enhancement. MRR acquisition was performed (as described below) prior to pre and post-contrast VIBE imaging, using 4 mL gadolinium-DTPA contrast medium. Pre- and post-contrast VIBE acquisitions were subsequently performed using remainder of the standard weight-based dose of gadolinium-DTPA contrast.

MRR was performed using a 3D spoiled T1-weighted gradient echo sequence with a view-sharing undersampling technique called time-resolved angiography with stochastic trajectories (TWIST) (14, 15). Imaging was initiated 5-seconds after administration of 4 mL gadolinium-DTPA contrast medium (Magnevist, Berlex Laboratories, Wayne, NJ) at a rate of 2 mL/s followed by 20 mL saline flush at the same rate. Imaging was performed in coronal plane covering the abdominal aorta and both kidneys with the following parameters: TR/TE (msec) = 2.33/0.77, flip angle 12°, slice thickness 2.5 mm, bandwidth 650 Hz/voxel, voxel size 2.4 × 1.7 × 2.5 mm, parallel imaging factor 3, 40 slices acquired per measure. Total acquisition time for the first initial full-k space sampling was 5.1 seconds. Each repeat measurement of the TWIST sequence updates 20% of central k-space area and 20% of outer k-space lines at a frame rate of 1.2 seconds. This sampling scheme for TWIST has been shown to minimize error in GFR estimation (14). Acquisitions included 21 initial post-contrast measures at 1.2 seconds temporal resolution in a long breath hold (29 sec), and subsequent 3 to 5 measures performed (acquisition time 8 to 10 seconds) every 30 seconds for 4 minutes.

All patients tolerated pre-and post-operative MRI. No patients were excluded from analysis.

MR Image Postprocessing

MRR images were independently processed by two readers (_ and _), with 2 years and 3 months of experience with the software, who were blinded to the clinical data and surgical technique. Co-registration of dynamic data sets was performed using mutual information algorithm, followed by segmentation of the cortex, medulla, and collecting system using validated semi-automated software to produce aortic, renal cortical and medullary signal intensity versus time curves (16). To calculate arterial input function, an ROI was drawn in the center of the abdominal aorta lumen at the level of the renal arteries. The signal intensities were converted to T1 values, which in turn were converted to gadolinium tracer concentration [Gd] versus time curves using the relationship $[Gd] = (1/T1 - 1/T1_0)/r_1$, where r_1 is the relaxivity of gadolinium ($4.5 \text{ mM}^{-1} \cdot \text{s}^{-1}$) and $T1_0$ represents the measured values for aorta, renal cortex and medulla before contrast (17).

To calculate the single kidney GFR, we applied a tracer kinetic model with an impulse retention function (IRF) compartmental approach. The IRF for the cortex and medulla are convolved with the arterial input function to derive the gadolinium tracer-time curve in the renal parenchyma. Specifically, a three-compartment tracer kinetic model fitting this convolution follows movement of the tracer from the renal vascular bed to the proximal tubule, and from the proximal tubule into the loop of Henle, which yields SK-GFR as well as perfusion parameters for each kidney (12, 13, 17–19). All parameters were calculated by minimizing residual discrepancies between the measured data and the model fitting using the Levenberg-Marquardt algorithm in Matlab (MathWorks, Natick, Massachusetts). SK-GFR for both kidneys was summed for the total MR-GFR. Both SK-GFR and MR-GFR were normalized to the patients' body surface area. The renal parenchymal volume of each kidney was calculated by summing the volumes of the segmented cortex and medulla that were automatically generated (16).

Laboratory and Clinical Parameters

For each patient, we obtained serum creatinine within 24 hours of preoperative MR imaging and on the day of each postoperative imaging test. We calculated eGFR using the MDRD formula. The ischemia type (warm or cold) and ischemia time were recorded by the operating urologists. Medical comorbidities such as diabetes mellitus, hypertension and pertinent medications were also recorded for each patient.

Statistical Analyses

Inter-reader agreement in SK-GFR and MR-GFR were assessed by calculating the coefficient of variation and concordance correlation coefficient. Unequal variance t tests were used to compare preoperative MR-GFR and SK-GFR values and tumor characteristics of patients in the cold and warm ischemia groups (Table 1). Unequal variance t tests were also utilized to compare operated kidney changes in SK-GFR with respect to ischemia types (warm vs. cold). We also examined the changes in operated kidney SK-GFR with warm ischemia time less than or at least 40 minutes as previously suggested for a threshold ischemia time associated with increased risk of irreversible damage (7). The association of ischemia time with changes in SK-GFR of the operated kidney as measured by each reader, and the association of renal parenchymal volume loss and operated kidney SK-GFR changes were also examined.

Pearson correlation was used to examine the association of preoperative MR-GFR with postoperative MR-GFR, 48–72 hour post-operative normal SK-GFR and operated kidney SK-GFR.

The normality assumptions underlying the parametric t tests and Pearson correlations were found through residual plots and Shapiro-Wilk tests to be reasonably met. All statistical tests were conducted at the two-sided 5% significance level using SAS 9.3 (SAS Institute, Cary, NC).

Results

Eighteen patients underwent preoperative and immediate postoperative MR imaging. Six patients underwent open PN while the remaining 12 patients had laparoscopic PN. Eleven patients (four open PN and seven laparoscopic PN) returned for postoperative imaging at approximately 6 months (mean 220 days, range 166–407 days). Mean preoperative MR-GFR, mean operated kidney SK-GFR, ischemia time, maximum tumor diameter, and estimated blood loss did not differ significantly between the open and laparoscopic PN groups (Table 1).

Inter-reader Agreement

The between-reader concordance correlation coefficient for SK-GFR and renal volume changes in the affected and contralateral kidney ranged from 0.92–0.98 in pre-operative and both post-operative measurements. The coefficient of variation ranged from 4.5 – 9.6% for SK-GFR measurements. These results suggest excellent agreement between readers for GFR measurements and volume changes in each kidney.

Post-operative Changes in MR-GFR and eGFR

Mean pre-operative MR-GFR was 72.6 mL/min/1.73 m², range 43–112 mL/min/1.73 m², and decreased postoperatively to 58.9 mL/min/1.73 m², range 32–94 mL/min/1.73 m². Mean pre-operative eGFR was 86.5 mL/min/1.73 m², range 58–136 mL/min/1.73 m², and remained higher than MR-GFR postoperatively at mean 70.2 mL/min/1.73 m², range 49–136 mL/min/1.73 m². Pre-operative MR-GFR resulted in CKD upstaging compared with eGFR in seven patients: from stage 1 to 2 in two patients, 1 to 3 in one patient, and 2 to 3 in four patients.

Operated kidney SK-GFR decreased in 15 of 18 patients with a mean decrease of 31% ± 23% (Figure 1). Meanwhile, the calculated eGFR decreased in 13 of 18 patients (mean eGFR change 19% ± 14%) with undetected change in two patients whose MR-GFR decreased by more than 30%. The MR-GFR underestimated the eGFR by approximately 25%, which is similar to previously reported findings (13). At 6 months, operated kidney SK-GFR was decreased by a mean of 18% (±19%) compared with baseline values.

Effect of Ischemia Type and Time on Operated Kidney GFR and Renal Volume Loss

Comparison of the post-operative decrease in operated kidney GFR between ischemia types showed that warm ischemia was associated with greater mean loss of 36% (± 24%) compared with 22% (± 18%) in the cold ischemia group (Figure 2). Three patients with warm ischemia time of at least 40 minutes had greater operated kidney loss than warm ischemia less than 40 minutes (Table 2). There was moderate correlation between SK-GFR functional loss and warm ischemia time ($r=0.55$; $p=0.06$). There was no correlation between warm ischemia time and MR-GFR or eGFR ($r=0.05$ and -0.05 respectively). Within the cold ischemia group, one patient had cold ischemia time of 40 minutes with operated kidney GFR loss of 49%, compared with mean loss of 17% in the remainder of the group. Overall, cold ischemia yielded the smallest decrease in operated kidney SK-GFR at 48 hours, while warm ischemia of at least 40 minutes was associated with the greatest loss of operated kidney SK-GFR and parenchymal volume loss (Table 3), though differences were not statistically significant ($p > 0.20$).

Contralateral Kidney Compensatory Effects

Compensatory increase in the contralateral non-operated kidney occurred in six patients 48–72 hours after surgery (Figure 1) with mean increase of 22.5 ± 13.6%. Five of the six patients demonstrated preoperative MR-GFR of greater than 60 mL/min/1.73 m², and all six patients had MR-GFR values greater than 50 mL/min/1.73 m². Preoperative MR-GFR demonstrated a correlation with increased normal kidney GFR at 48–72 hours, significant for one reader ($r = 0.41$ – 0.47 , $p = 0.048$, 0.090). Three of the six patients with compensatory SK-GFR increases returned for follow up imaging at six months, and all three of these patients demonstrated sustained increase in SK-GFR of the non-operated contralateral kidney (Figure 3).

Of the patients who did not show compensatory increases in contralateral kidney function, eight patients had stable non-operated kidney SK-GFR and four patients demonstrated decreased SK-GFR, by a mean of 26 ± 7% (Figure 1).

Renal Parenchymal Volume Loss and Operated Kidney GFR

Mean parenchymal volume loss for the operated kidney was 13.8% at 48–72 hours and 13.1% at 6 months. Volume loss was correlated with operated kidney GFR loss 48–72 hours after surgery, significant for one reader with a strong trend for the second reader ($r = 0.45$, $p < 0.06$), but no correlation was found at 6 months ($r = -0.13$).

Discussion

Over the past decade, PN has become the standard of care for treatment of small kidney tumors, with data showing that PN reduces risk of developing new-onset postoperative CKD and associated morbidity and mortality (3, 5). CKD, however, is highly prevalent in patients with kidney tumors, and up to one-half of patients undergoing PN will ultimately develop CKD (5). Considerable attention has been paid to identifying factors that impact kidney functional outcomes following PN (20–22). Although some factors cannot be modified, such as pre-operative GFR and tumor size and location, surgical factors such as ischemia type and duration can be altered in order to provide maximum preservation of renal function.

The significance of ischemia duration and type remains poorly understood. Prior studies of renal functional change following partial nephrectomy using eGFR have suggested that warm ischemia time does not affect renal function (22, 23, 24). Using MRR, we found significant correlation of prolonged ischemia time with operated kidney functional loss in the immediate postoperative period, despite the small number of patients in our study. We also found that warm ischemia of any time was associated with greater SK-GFR loss than cold ischemia, and that extended warm ischemia resulted in greater loss than ischemia less than 40 minutes; a recent study using Tc 99m-DTPA scintigraphy also recently reported greater affected kidney functional loss after prolonged warm ischemia time (25). No definite conclusions could be drawn regarding the effect of ischemia type and time at 6-month follow-up in our study due to the small number of patients.

eGFR failed to provide information about the surgical impact on the operated kidney, and showed similar decrease in function (18% vs. 19.5%) irrespective of the ischemia time. Furthermore, eGFR demonstrated no correlation with ischemia time, although SK-GFR was moderately correlated with ischemia time. However, eGFR decreased more in laparoscopic PN than open PN, though not significantly, and consistently provided higher values than MR-GFR both immediately after surgery and at six months. MR-GFR underestimates the eGFR which has been noticed in prior studies (13), and may be related to neglecting water-exchange kinetics or flow effect in the aorta in MR-GFR measurement. Furthermore, there are concerns regarding overestimation of renal function by eGFR due to inability of serum creatinine based formula to assess acute kidney insult in immediate post-operative period.

MRR can also provide insight into changes in the contralateral non-operated kidney. Post-operative compensatory increases in non-operated kidney SK-GFR were detectable in one-third of patients just 2–3 days after surgery. This can mask the GFR loss in the operated kidney when using serum creatinine to measure global renal function. Three of six patients with increased immediate post-op SK-GFR in the non-operated contralateral kidney returned for follow-up MRR at six months and demonstrated sustained increase in SK-GFR. Similar results were also noted using Tc 99m-DTPA renal scintigraphy that demonstrated increased GFR of the unaffected kidney at 3 months (26). These interesting results are not well understood and long-term effects of persistent increased filtration are unclear.

In our study, compensation in the contralateral kidney GFR immediately after surgery showed positive correlation with preoperative baseline MR-GFR. This suggests better ability of patients with normal baseline function to increase GFR in the non-operated kidney to

compensate functional loss in the operated kidney. However, even in patients with MR-GFR $> 60 \text{ mL/min/1.73 m}^2$, nearly half the patients did not demonstrate this compensatory increase in function. In four patients, postoperative decreases in the contralateral kidney GFR may have been related to surgical factors, hydration status, heart disease, or other perioperative effects related to medication, which could be further studied as surgical risk factors for CKD. Thus MRR can act as a tool for studying various factors that can potentially impact not only the functional loss in the operated kidney but also compensatory effect in the contralateral kidney, while providing more diagnostic information than renal scintigraphy (26, 27).

Renal parenchymal volume loss was significantly associated with operated kidney GFR decrease only immediately after surgery but not at 6 months, possibly due to the ability of the remaining functional parenchyma to recover from the ischemic episode or compensate for lost parenchyma. A recent study in the urologic literature reported the association of functional volume preservation and not ischemia time with long-term conserved renal function but was limited by an insensitive estimate of GFR using the MDRD formula (24). Our findings suggest that even in the setting of postoperative volume loss, individual kidneys vary in ability to compensate and may eventually recover in function (28).

Limitations of our study include small sample size. A larger study would allow for multivariate analysis of postoperative renal GFR changes related to ischemia type, time, lesion characteristics, and preoperative renal function. Image analysis for MRR involves use of validated, semi-automated segmentation software and application of a tracer kinetic model, which entails a post-processing time of approximately 30 minutes. Lack of standardization in acquisition and post-processing of the perfusion data limits its wide spread use across different institutions. The injection of gadolinium contrast prior to a conventional contrast-enhanced MR examination of the kidneys has the potential to confound the detection of small, slightly enhancing renal masses. Given that the conventional dose is approximately 4 to 5 times the dose used for MRR, the likely impact on lesion detection is low, although this needs to be investigated further.

A potential risk of DCE MRR in the perioperative setting is the need for gadolinium-based contrast, which carries a risk of nephrogenic systemic fibrosis in patients with severe chronic kidney disease (29). No patients in our study who completed pre- and post-operative MRR had eGFR or MR-GFR less than $30 \text{ mL/min/1.73 m}^2$. Although we used a linear gadolinium agent in our study, use of macrocyclic agent with higher stability can further alleviate the concerns for NSF and can potentially permit use of this technique in subjects with GFR less than $30 \text{ mL/min/1.73 m}^2$. Development of non-contrast functional MRI techniques such as diffusion tensor imaging (DTI), blood oxygen level-dependent (BOLD), and arterial spin labeling (ASL) may also provide insight into renal insult related to surgery while avoiding the potential harm of NSF in patients with severe chronic kidney disease (30–33).

We have demonstrated the utility of MRR pre- and post-operatively as an adjunct to clinical MR imaging for renal mass evaluation and postoperative surveillance, adding less than 10 minutes of additional scanning time and a low dose of gadolinium contrast. To aid clinical management of early stage renal neoplasms, MRR may eventually be used to risk stratify patients for chronic kidney disease and select surgical approach, including modifiable risk factors such as ischemia type and time, to minimize post-surgical renal functional loss.

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Figure 1. Operated and contralateral kidney pre and postoperative SK-GFR. Line is an identity line ($y = x$). MR renography showed SK-GFR decrease in 15 of 18 operated kidneys. In 6 patients the contralateral kidneys increased SK-GFR in response to functional loss in the operated kidneys.

Figure 2. Box plot demonstrating operated kidney GFR loss in warm ischemia is greater compared to cold ischemia. Whiskers extend to maximum and minimum values.

Figure 3.

61 year old woman with a left renal mass. (A) Preoperative axial post-contrast image depicts a 1.8 cm mass in the anterior left kidney (arrow). (B) Baseline MRR gadolinium concentration vs. time curves for the left renal cortex and medulla. After application of tracer kinetic model this yielded preoperative left SK-GFR of 45 mL/min/1.73 m². Total MR-GFR (summed over both kidneys) was 75 mL/min/1.73 m² and MDRD eGFR was 73 mL/min/1.73 m². (C) Immediate post-operative gadolinium concentration vs. time curve of the left kidney. Application of tracer kinetic model yielded left kidney SK-GFR of 10 mL/min/1.73 m², a decrease of 78%. Significant arterial bleeding and prolonged ischemia time of 49 minutes occurred during surgery. Total MR-GFR (summed over both kidneys) was 47 mL/min/1.73 m². Right kidney SK-GFR showed compensatory increase to 37 mL/min/1.73 m² from baseline SK-GFR of 30 mL/min/1.73 m². MDRD eGFR decreased to 56 mL/min/1.73 m² but underestimated the degree of insult to the left kidney and could not assess compensatory increase in function of the right kidney. (D) 6 month post-operative gadolinium concentration vs. time curve of the left kidney. There was near-complete recovery of renal function on the basis of eGFR which returned to baseline value of 73 mL/min/1.73 m². However, at MRI left kidney SK-GFR did not reach the baseline and remained diminished to 39 mL/min/1.73 m². Right kidney continued to demonstrate sustained increase in function with SK-GFR of 45 mL/min/1.73 m². Although total MR-GFR also returned to normal, evaluation of left and right kidney function separately provided more insight into the degree of renal damage in the operated kidney and response in the contralateral kidney.

Figure 4.

Figure 5.

Figure 6.

Table 1

Patient and surgical characteristics of patients undergoing partial nephrectomy.

Characteristic	OPN	LPN	All	P value
Number of Patients	6	12	18	---
Preoperative MR-GFR (mL/min/1.73 m ²)	68.0 ± 23.2	74.7 ± 18.7	72.5 ± 19.9	0.557
Preoperative Affected Kidney GFR (mL/min/1.73 m ²)	29.9 ± 7.8	35.4 ± 9.0	33.6 ± 8.8	0.206
Ischemia Time (min)	24.3 ± 16.2	28.9 ± 12.0	27.4 ± 13.2	0.557
Maximum Tumor Diameter (cm)	4.5 ± 1.0	3.3 ± 1.7	3.7 ± 1.6	0.097
Estimated Blood Loss (mL)	220 ± 195	327 ± 558	294 ± 469	0.580

OPN = open PN, LPN = laparoscopic PN. Values are mean ± standard deviation unless otherwise indicated

Table 2

Distribution of patients by ischemia time within each ischemia type

Time (min)	Warm Ischemia (# patients)	SK-GFR Loss (%)	Cold Ischemia (# patients)	SK-GFR Loss (%)
0–9	0	--	1	20.9
10–19	2	29.0	1	31.1
20–29	6	28.2	1	5.5
30–39	1	42.1	2	14.4
40–50	3	55.2	1	49.7

Table 3

SK-GFR decrease and parenchymal renal volume changes in the operated kidney in immediate post-operative period after PN.

	Cold Ischemia	Warm Ischemia < 40 min	Warm Ischemia > 40 min
Operated SK- GFR Mean Decrease (%)	22.8 ± 18.6	29.3 ± 20.6	55.2 ± 30.6
Parenchymal Volume Loss (cc)	1.9 ± 7.6	16.1 ± 10.4	23.9 ± 3.9

Values are mean ± standard deviation.