# Prospective Measurement of Urinary Microalbumin in Living Kidney Donor Nephrectomy: Toward Understanding the Renal Functional Recovery Period

Young Eun Yoon, Kwang Suk Lee, Kyung Hwa Choi, Kwang Hyun Kim, Seung Choul Yang and Woong Kyu Han*

*From the Department of Urology, Urological Science Institute, Yonsei University College of Medicine (YEY, KSL, KHC, SCY, WKH) and Department of Urology, Ewha Womans University Mokdong Hospital (KHK), Seoul, Korea*

**Abbreviations and Acronyms**

CKD = chronic kidney disease

DTPA = diethylenetriamine pentaacetic acid

eGFR = estimated glomerular filtration rate

IB = implantation biopsy

IgA = immunoglobulin A

MA = microalbuminuria

MDRD = Modification of Diet in Renal Disease

UACR = urinary albumin-to-creatinine ratio

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* Correspondence: Department of Urology, Urological Science Institute, Yonsei University College of Medicine, 50 Yonsei-ro, Seodaemun-gu, Seoul, 120-752, South Korea (telephone: +82-2-2228-2310; FAX: +82-2-312-2538; e-mail: hanwk@yuhs.ac).

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**Purpose:** We determined the clinical implications of perioperative urinary microalbumin excretion in relation to renal function after living donor nephrectomy.

**Materials and Methods:** Between August 2010 and January 2013, 259 donors undergoing live donor nephrectomy were enrolled in the study. The donor urinary albumin-to-creatinine ratio was measured perioperatively, and changes in perioperative urinary albumin-to-creatinine ratio and the implications of preoperative microalbuminuria (urinary albumin-to-creatinine ratio 30 mg/gm or greater) were investigated. The relationships between perioperative urinary albumin-to-creatinine ratio and recovery of renal function and implantation biopsy histology were also analyzed.

**Results:** Mean ± SD preoperative urinary albumin-to-creatinine ratio was 7.1±12.7 mg/gm. The urinary albumin-to-creatinine ratio was increased after 1 day (24.7±18.9 mg/gm, $p < 0.001$) and stabilized after 1 month (10.3±10.7 mg/gm, $p < 0.001$). Preoperative microalbuminuria was not associated with perioperative estimated glomerular filtration rate during a followup period of 6 months but was associated with histological abnormalities. Donors with a higher urinary albumin-to-creatinine ratio before donation, even in the normal range, consistently had an increased postoperative urinary albumin-to-creatinine ratio. A ROC curve analysis showed that age, preoperative estimated glomerular filtration rate and 1-month postoperative urinary albumin-to-creatinine ratio were highly predictive of delayed recovery of renal function (AUC 0.884, $p < 0.001$). The 1-month postoperative urinary albumin-to-creatinine ratio was associated with delayed recovery of renal function (OR 1.05 for each 0.1 mg/gm increase, $p = 0.021$).

**Conclusions:** Donors with higher preoperative urinary albumin-to-creatinine ratio levels require close observation because there is a greater possibility of microalbuminuria developing after donation even if the ratio is within the normal range. A higher urinary albumin-to-creatinine ratio was also associated with delayed recovery of renal function and histological abnormalities.

**Key Words:** kidney transplantation, albuminuria, delayed graft function, nephrectomy, living donors

KIDNEY pathology or damage due to acute injury can lead to proteinuria.[1] Proteinuria can be measured by 24-hour urine collection, which is difficult and cumbersome for the donor. Urinary microalbumin, a potential



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substitute for measuring proteinuria, is more convenient because it can be checked in spot urine samples.[2] Currently microalbuminuria is more frequently measured because precise and simple methods for estimating microalbumin in urine are available.[3] It is usually expressed as the urinary albumin-to-creatinine ratio to compensate for hydration status.[4] Normally UACR is less than 30 mg/gm.[5]

Numerous studies have been performed to investigate the implications of MA. McCullough et al concluded that albuminuria is a typical marker of CKD in young adults, whereas reduced estimated glomerular filtration rate is the most frequent abnormality in elderly people with CKD.[6] A study in a large Canadian cohort demonstrated that MA is a risk factor for end stage renal disease even in patients whose eGFR is relatively normal (greater than 60 ml/minute/1.73 $m^2$).[7] Moreover, a UACR value greater than 10 mg/gm is a predisposing factor for all cause mortality and cardiovascular mortality in the general population.[8]

A reduction in nephrons after donor nephrectomy causes compensatory hyperfiltration of the contralateral remnant kidney, which can cause proteinuria.[9] MA can also easily occur after kidney donation.[10] However, to our knowledge no information on the significance of preoperative MA in kidney donors is available, and few studies evaluating the association between perioperative urinary albumin excretion and recovery of donor renal function have been reported in the literature.[9] Therefore, this study was conducted to assess the relationship between MA and kidney donor renal function. Our hypotheses were 1) donors with MA before nephrectomy may have worse remnant renal function, 2) MA is associated with delayed recovery of renal function and 3) MA can reflect histological abnormalities in implantation biopsies.

## MATERIALS AND METHODS

A total of 259 donors who underwent living donor nephrectomy at our institution between August 2010 and January 2013 were enrolled in the study. Donor data were prospectively recorded and archived. All donor nephrectomies were performed by 2 surgeons using video-assisted mini-incision surgery as previously reported.[11–13] All donors underwent DTPA renal scans and computerized tomography angiography, and provided 24-hour urine samples. Donors with a 24-hour urine creatinine clearance less than 80 ml/minute/1.73 $m^2$ and an eGFR less than 80 ml/minute/1.73 $m^2$ on preoperative examinations were excluded from the matched living kidney transplantation in accordance with our institutional donor criteria. The institutional review board approved this study (4-2013-0421) and all subjects provided written, informed consent.

### Renal Function and Urinary Microalbumin

Urinary microalbumin and serum creatinine were determined for all donors preoperatively, on postoperative day 1, and 1, 3, and 6 months after the procedure. Renal function was estimated with the MDRD formula. Urinary microalbumin was measured by an immunoturbidimetric method using an AU680 automated chemistry analyzer (Beckman Coulter, Inc., Brea, California). Because hydration status or dilution of urine can affect the concentration of urinary microalbumin, UACR was used as a measure of MA.[4] The normal range of UACR is defined as less than 30 mg/gm.[5] Differences between donors with MA before the operation and normal donors were investigated. Because some studies have reported that UACR values greater than 10 mg/gm are clinically important, despite being in the normal range,[8,14] we classified the donors into those with an increased UACR (10 mg/gm or greater) and those with UACR less than 10 mg/gm. Delayed recovery of renal function was defined as MDRD-eGFR less than 60 ml/minute/1.73 $m^2$ at 6 months after kidney donation.[15]

### Implantation Biopsy

Histological examinations were performed only in donors who agreed to IB before donor nephrectomy. After transplantation of the procured allograft IB was performed in the operating theater immediately before reperfusion. All biopsy samples were examined by light microscopy, and additional immunofluorescence and electron microscopic analyses were performed if needed.[16] Histological abnormalities were scored according to the 2007 Banff classification.[17] Global glomerulosclerosis was defined as at least 5% of all observed glomeruli showing sclerotic changes. The median number of observed glomeruli was 17. Unexpected IgA nephropathies were also counted.

### Statistical Analysis

Continuous data are presented as means $\pm$ SD. Differences between the 2 groups were analyzed using Student's t-test for continuous variables and Fisher's exact test and Pearson's chi-square test for categorical variables. Paired samples were analyzed using the paired Student's t-test. Logistic regression analysis was performed to model the value of different parameters in predicting delayed recovery of kidney function. A value of $p < 0.05$ was considered statistically significant. Statistical analyses were performed using SPSS® version 20.0.

## RESULTS

### Donor Characteristics

Donor characteristics are summarized in the table. Mean age was 40.6 $\pm$ 11.6 years, 40.9% of donors (106) were male and most donor nephrectomies (82.2%) were performed on the left side. No donors had diabetes but 8 had hypertension that was well controlled with medication. Mean preoperative MDRD-eGFR was 96.5 $\pm$ 16.7 ml/minute/1.73 $m^2$. No donors had proteinuria in preoperative 24-hour urine samples.

*Donor characteristics*

| | |
|---|---|
| Mean ± SD pt age | 40.6 ± 11.6 |
| No. male (%) | 106 (40.9) |
| No. female (%) | 153 (59.1) |
| No. laterality (%): | |
| Lt | 213 (82.2) |
| Rt | 46 (17.8) |
| Mean ± SD body mass index | 23.2 ± 2.5 |
| Mean ± SD body surface area | 1.69 ± 0.18 |
| Mean ± SD preop creatinine (mg/dl) | 0.79 ± 0.16 |
| Mean ± SD preop MDRD eGFR (ml/min/1.73 $m^2$) | 96.5 ± 16.7 |
| Mean ± SD preop DTPA eGFR (ml/min/1.73 $m^2$) | 106.1 ± 20.4 |
| Mean ± SD preop cystatin C eGFR (ml/min/1.73 $m^2$) | 129.1 ± 28.6 |
| Mean ± SD preop UACR (mg/gm) | 7.1 ± 12.7 |

## Perioperative UACR and Renal Function

Before donor nephrectomy mean UACR was 7.1 ± 12.7 mg/gm. A correlation analysis showed that UACR was correlated with age ($r^2$=0.019, p=0.036), but not with gender, laterality, body mass index or preoperative eGFR. Eight donors (3.1%) had preoperative MA (UACR 30 mg/gm or greater). These donors exhibited a lower MDRD-eGFR compared with other donors (90.3 vs 96.7 ml/minute/1.73 $m^2$). However, this difference did not reach statistical significance (p=0.286). The MA group also had a lower MDRD-eGFR at 6 months after kidney donation (60.5 vs 65.8 ml/minute/1.73 $m^2$, p=0.245).

At postoperative day 1 UACR was increased to 24.7 ± 18.9 mg/gm. One month after donation UACR decreased to 10.3 ± 10.7 mg/gm (p <0.001), and remained stable at 3 months (10.0 ± 10.2 mg/gm, p=0.943) and 6 months (13.6 ± 20.1 mg/gm, p=0.496) after the operation (fig. 1). In correlation analyses preoperative UACR was correlated with UACR at 1 day ($r^2$=0.039, p <0.001), and 1 month ($r^2$=0.033, p=0.009), 3 months ($r^2$=0.027, p=0.045) and 6 months ($r^2$=0.210, p <0.001) postoperatively. Overall 40 donors (15.4%) with preoperative UACR 10 mg/gm or greater consistently had higher postoperative UACR values than the other donors (fig. 2). However, renal function changes were not different between the 2 groups throughout the observation period. After 6 months mean eGFR was almost equal (66.1 vs 65.4 ml/minute/1.73 $m^2$, respectively, p=0.787).

## Perioperative UACR and Delayed Recovery of Renal Function

Of the 259 donors 124 (47.9%) completed a 6-month followup. Mean MDRD-eGFR at 6 months was 65.6 ± 12.5 ml/minute/1.73 $m^2$ (range 40.8 to 101.3). Recovery of renal function was delayed in 44 of 124 donors (38.6%). Among the donors with preoperative MA only 5 completed followup, of whom 3 (60%) were assigned to the delayed renal function recovery group (p=0.242).

A comparison of the delayed renal function recovery group to the normal renal function recovery group revealed no differences in preoperative UACR (8.8 vs 6.5 mg/gm, p=0.245) or postoperative UACR (22.1 vs 23.2 mg/gm, p=0.765, fig. 3). However, at 1 month after donor nephrectomy the delayed renal function recovery group still had a higher UACR level than the normal renal function recovery group (13.4 vs 8.7 mg/gm, p=0.015). UACR in the delayed renal function recovery group trended higher again

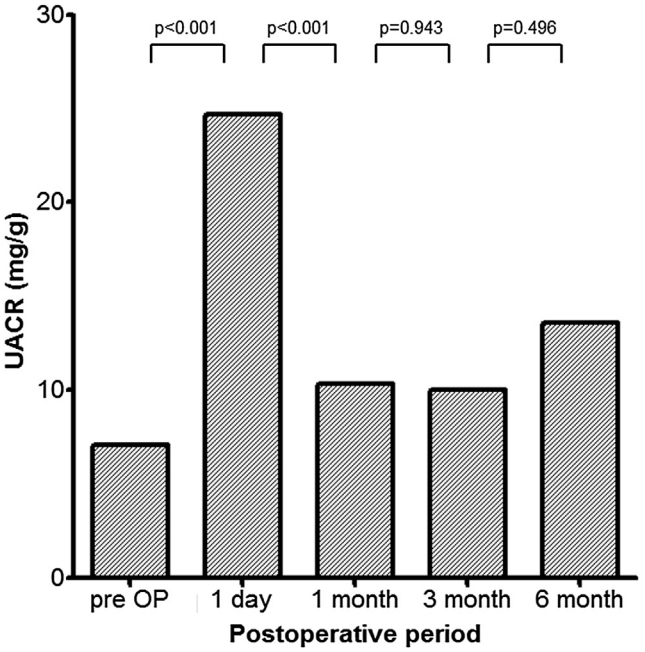


**Figure 1.** Overall change in UACR. UACR increases were greatest on postoperative day 1 and stabilized from 1 month postoperatively. *Pre OP,* preoperatively.

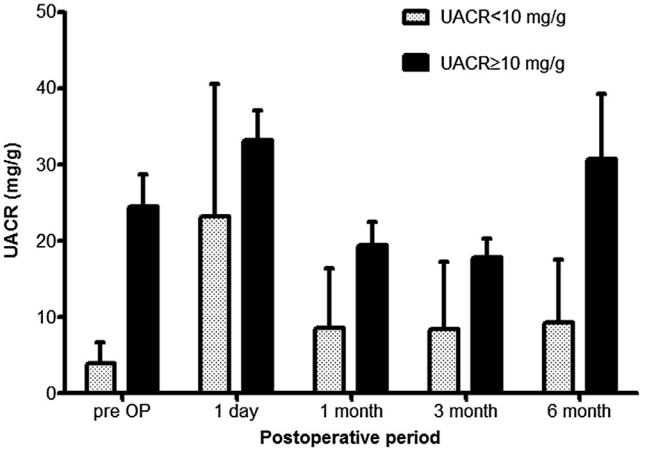


**Figure 2.** Postoperative UACR. Donors with preoperative UACR 10 mg/gm or greater had higher postoperative UACR throughout observation period than those with preoperative UACR less than 10 mg/gm. All differences between 2 groups were statistically significant (p <0.001). *Pre OP,* preoperatively.

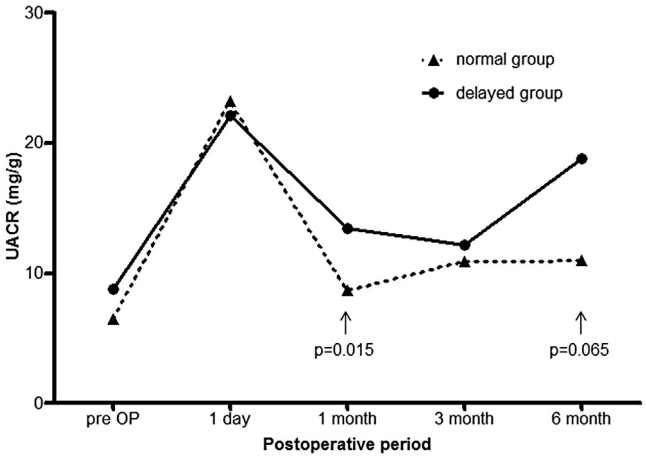


**Figure 3.** UACR of normal and delayed renal function recovery groups. Delayed renal function recovery group had higher UACR at 1 month after donor nephrectomy. *Pre OP,* preoperatively.

at 6 months after donation but this difference was not statistically significant (18.8 vs 11.0 mg/gm, p=0.065). One-month postoperative UACR was associated with delayed recovery of renal function (OR 1.05 for each 0.1 mg/gm increase, p=0.021). Multivariate logistic regression analysis revealed an association between older age (OR 1.07, p <0.001), lower preoperative MDRD-eGFR (OR 0.89 for each 0.1 ml/minute/1.73 $m^2$ increase, p <0.001) and higher UACR 1 month after kidney donation (OR 1.04 for each 0.1 mg/gm increase, p=0.016) and delayed recovery of renal function. A ROC curve analysis showed that age, preoperative MDRD-eGFR and 1-month postoperative UACR were highly predictive of delayed recovery of kidney function (AUC 0.884, p <0.001).

### Preoperative UACR and Implantation Biopsy

Of the 259 donors 127 (49%) agreed to a time-zero IB and of these donors 70 (55.1%) had histological abnormalities on IB. Among the 8 MA donors 6 provided IB. All biopsies of the MA group were histologically abnormal (p=0.024). One had IgA nephropathy, 1 had arteriosclerosis, 1 had focal global glomerulosclerosis (7.7% of all glomeruli showed sclerotic change), 1 had tubular atrophy and 2 had hyaline arteriosclerosis. Even among normal UACR donors those with a UACR 10 mg/gm or greater exhibited abnormal histology. Among donors with UACR 10 mg/gm or greater 84.2% (16 of 19) had an abnormal histology whereas 50% of donors with a UACR less than 10 mg/gm had an abnormal histology (p=0.006). Moreover increased UACR was associated with IgA nephropathy (15.8% vs 2.8%, p=0.043).

## DISCUSSION

Generally microalbumin in urine is considered to have come from glomerular filtration. In a normal kidney almost all microalbumin is absorbed or degraded in the proximal tubule. In pathological states glomeruli may become more permeable to albumin because of basement membrane abnormalities or malfunctions in endothelial cells or podocytes.[18] In addition, deteriorated reabsorption or degradation of proximal tubules may cause MA.[19]

Conventionally the normal range of UACR is considered to be less than 30 mg/gm.[5] However, the normal range is still under debate, with some arguing that this cutoff may be too high.[20] UACR greater than 10 mg/gm is associated with all cause and cardiovascular mortality.[8] Moreover, in their study of the metabolic syndrome Heo et al verified that MA and UACR values greater than 10.2 mg/gm are related to increased CKD prevalence.[14] Accordingly we sought to determine the significance not only of definitive MA (UACR 30 mg/gm or greater) but also that of the subclinical value of 10 mg/gm UACR.

The prevalence of MA or even proteinuria after donor nephrectomy has been previously demonstrated.[9] In results obtained in a French population Fourcade et al reported not only that donors may have MA but also that the concentration of microalbumin in urine increases at long-term followup.[21] In a meta-analysis Garg et al concluded that the pooled risk of MA after kidney donation is 3.9.[9] In the current study UACR increased immediately after the operation, which may have occurred because of the compensatory hyperfiltration of the contralateral remnant kidney. Kujal and Vernerova reported that glomerular hypertension is one of the main factors responsible for the development of renal injury in an experimental model of reduced nephron numbers.[22] They also reported on the accompanying proteinuria and deterioration of renal function.

We found that increased UACR stabilized 1 month after donor nephrectomy. In a previous study we demonstrated a functionally dynamic period of renal activity after donor nephrectomy that begins to stabilize at 1 month postoperatively.[23] These results are consistent with the suggestion that the contralateral remnant kidney adopts a hyperfiltration status within 1 month. However, donors with delayed recovery of renal function had relatively high levels of UACR until 1 month postoperatively. UACR levels also trended higher, albeit insignificantly, at 3 and 6 months in these individuals. One interpretation of these findings is that these donors are still in a functionally unstable condition at 1 month postoperatively and need more time to adapt to the reduction in nephron number.

Consequently donors with an increased UACR 1 month postoperatively need closer observation. To our knowledge, this is the first report to establish a relationship between perioperative UACR and recovery of renal function after donation.

In this study preoperative UACR was correlated with UACR on postoperative day 1, and at 1, 3 and 6 months. Moreover our comparison of donors with preoperative UACR above and below 10 mg/gm showed that those in the higher level group consistently had a higher level of UACR after donation. These findings suggest that a higher preoperative UACR is not an incidental finding but is rather a meaningful value, even if it is within the normal range. Increased preoperative UACR translates into a greater likelihood of MA or proteinuria developing after donor nephrectomy. We may assume that a higher preoperative UACR before donor nephrectomy reflects the possibility of preexisting renal impairment such as abnormal glomerular structure or deterioration of proximal tubular uptake. In addition, in a study of the risk factors for end stage renal disease after donation, Kido et al demonstrated that proteinuria was the only CKD progression factor acquired after donation.[24] This is made more meaningful by their observation that renal function stabilized for a long period after donation (mean 13.1 years) but suddenly started to decline in association with the appearance of risk factors for CKD such as proteinuria. Accordingly we might conclude that donors with a higher preoperative UACR should be followed regularly for years and closely monitored for renal function. To our knowledge there are no standards governing whether donors with increased urinary microalbumin secretion should be permitted to donate their kidneys.[25]

The proportion of donors with histological abnormalities was 55.1%, an unexpectedly considerable percentage. However, this ratio is similar to that reported in a study by Mancilla et al.[26] The clinical implication of IB in recipients is somewhat controversial.[27] A few studies have also investigated the association between IB and renal function of donors.[26,28,29] In the present study higher UACR levels were associated with histological abnormalities. However, Chauhan et al asserted that MA was not associated with a moderate to severe change in IB.[28] However, comparing the current results with the previous study is problematic because in that study the subjects were only categorized according to the presence of MA and only moderate to severe histological abnormalities were counted. Because Goecke et al contended that IB before donation is neither necessary nor useful, based on the absence of the development of clinical nephropathy in donors with histological abnormalities,[29] more long-term results are needed to demonstrate the clinical relationship between IB and renal function of kidney donors.

The current study has its limitations, including the fact that donors were only followed for approximately 6 months after donor nephrectomy and fewer than half of the study enrollees were followed. However, to our knowledge no studies have addressed the clinical implication of perioperative serial UACR. Our study is meaningful because it is the first to suggest the necessity of UACR examination before and after surgery in addition to routine laboratory tests such as serum creatinine and DTPA scans. Clearly additional research involving a larger number of donors and longer followup is required to definitively establish the significance of perioperative UACR.

## CONCLUSIONS

UACR becomes elevated during the period of renal function stabilization after donation and can provide information about delayed recovery of renal function. Donors with a preoperatively increased UACR require close observation, even if the level is within the normal range, because there may be a greater possibility of MA developing after donation, which is one of the predictive factors of CKD. Moreover higher UACR levels are associated with histological abnormalities. However, further studies with longer followup are needed to assess the association between UACR and long-term renal function in living kidney donors.

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