Impact of the Mayo Adhesive Probability Score on Donor and Recipient Outcomes After Living-donor Kidney Transplantation: A Retrospective, Single-center Study of 782 Transplants

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INTRODUCTION

The Mayo Adhesive Probability (MAP) score was created to predict the presence of adherent perinephric fat (APF), which has been identified as a risk factor for surgical difficulty and longer operative time in patients undergoing partial nephrectomy.1 The MAP score ranges from 0 to 5, based on the posterior renal fat thickness and perinephric stranding; these aspects can be assessed using computed tomography or MRI.1 It has been reported that risk factors for high MAP score are old age, male sex, diabetes, hypertension, and alcoholism.2 Smoking is also considered a risk factor for perinephric fat stranding alone.2 The mechanism underlying APF formation is presumably related to the systemic chronic inflammatory state caused by the aforementioned risk factors.3

Not only in partial nephrectomy, a higher MAP score has been associated with longer operative time in hand-assisted laparoscopic donor nephrectomy (HALDN) for living-donor kidney transplantation (LDKT).4 Moreover, the APF risk and MAP score are both important influences on operative complexity, as well as postoperative kidney function. Lamacchia et al5 reported that a greater thickness of perinephric fat was
associated with lower eGFR in patients with type 2 diabetes. Cockerill et al.\(^6\) also reported that a MAP score ≥1 in the nondonated kidney was associated with lower donor eGFR after nephrectomy. Thus, we hypothesized that the donor APF and a high MAP score might contribute to poor outcomes in LDKT recipients. If the MAP score affects recipient outcome, it could be an important new donor criterion. To the best of our knowledge, associations between donor MAP score and LDKT recipient outcomes have not been reported; it remains unclear whether the MAP score is suitable for use in donor selection. This study was performed to examine the impacts of donor MAP scores on LDKT donor and recipient outcomes.

**PATIENTS AND METHODS**

This retrospective study included 782 transplants involving consecutive LDKT between February 2008 and October 2019 at Kyushu University Hospital. The following data were collected: donor characteristics (age, sex, and body mass index [BMI]; prevalences of hypertension, hyperlipidemia, diabetes, and history of cardiovascular disease [CVD]; sidedness of donor’s kidney; and surgical procedure), recipient characteristics (age, sex, and previous kidney transplant), number of HLA mismatches, ABO incompatibility, use of rituximab, donor-specific antibody status, and time on dialysis. Hypertension, hyperlipidemia, and diabetes were all defined on the basis of current medication use. History of CVD was defined as a history of myocardial infarction requiring percutaneous or surgical revascularization; atherosclerosis obliterans requiring limb amputation and revascularization; or stroke. The study was performed in accordance with the guidelines of the Declaration of Helsinki, and the study protocol was approved by the Kyushu University Institutional Review Board for Clinical Research (IRB-No 24-54). This study was registered in the University Hospital Medical Information Network Clinical Trials Registry System (UMIN000008475). The review board waived the requirement for patient consent due to the retrospective nature of the study.

The MAP scores of the donated kidneys were calculated using preoperative computed tomography images. As described by Davidiuk et al.\(^7\), the MAP score was calculated as the sum of the posterior renal fat and perinephric stranding scores. The posterior renal fat was measured as the length (in centimeters) of a straight line from the renal capsule to the posterior abdominal wall at the level of the renal vein of the donor kidney: <1 cm = 0 points, 1.1–1.9 cm = 1 point, and ≥2.0 cm = 2 points. Perinephric stranding was defined as soft tissue attenuation of the fat surrounding the kidney. If present, this was scored according to severity: no stranding = 0 points, thin mild stranding = 2 points, diffuse stranding = 3 points). The posterior renal fat and perinephric stranding scores were added to obtain a MAP score of 0–5.\(^7\)

The donor nephrectomy procedures differed over time, such that donors underwent hand-assisted retroperitoneoscopic donor nephrectomy (HARDN) from February 2008 to September 2015, HALDN from October 2015 to June 2019, and pure-retroperitoneoscopic donor nephrectomy (PRDN) from May 2019 to October 2019. These procedures were performed as previously described.\(^8\)

The immunosuppressive protocol was as follows: 20mg basiliximab was administered on the d of the operation and on postoperative d (POD) 4. For ABO-compatible recipients, oral immunosuppression agents (tacrolimus, mycophenolate mofetil or everolimus, and methylprednisolone) were initiated on preoperative d 7; ABO-incompatible transplant recipients began oral immunosuppressants on preoperative d 14, then received rituximab and plasma exchange before transplantation.

Kidney graft loss was defined as the time when dialysis was reintroduced or retransplantation was performed. Slow graft function was defined as serum creatinine >3 mg/dL on POD 5 but no need for dialysis; delayed graft function (DGF) was defined as the need for postoperative dialysis during the first 7 d postoperatively. Complications during hospitalization for surgery were considered surgical complications. In recipients, surgical complications were classified using the Clavien–Dindo system,\(^9\) while complications in donors were classified using a modification of the Clavien–Dindo system,\(^10\) that is specific for living-donor nephrectomy. Recipient surgical complications were analyzed in this study if their Clavien-Dindo grade was greater than III and could be attributed to donor factors. In accordance with hospital protocol, kidney biopsy was performed at the time of operation (0-h biopsy), as well as at 3 mo and 1 y postoperatively; it was also performed whenever clinical rejection was suspected.

**Statistical Analysis**

The normality of distributions for continuous variables were assessed using the Shapiro–Wilk test. Results are presented as the mean±SD for normally distributed variables, median (interquartile range) for variables that were not normally distributed, and count (percentage) for categorical variables. For normally distributed continuous variables, mean bivariate differences between 2 groups were assessed using Student’s t-test; mean univariate differences in ordinal variables among groups were assessed using the Wilcoxon rank-sum test. For continuous variables that were not normally distributed, median differences were compared using the Wilcoxon rank-sum test. Categorical variables were compared using the \(\chi^2\) test. Graft and patient survival were both calculated using the Kaplan–Meier method; differences between curves were evaluated using the log-rank test. The recipient eGFR within each group from POD 1 to 7 was compared using repeated-measures ANOVA. Bonferroni correction was used to reduce type I error because of multiple comparisons among time points. Multivariate analysis was performed using effect leverage plots to identify the effects of donor MAP score on recipient outcomes after adjustment for patient background. All statistical analyses were performed using JMP software (version 14, SAS Institute Inc, Cary, NC). A 2-sided P value of <0.05 was considered statistically significant.

**RESULTS**

No donor in this study required renal replacement therapy during the follow-up period, and no donor in this study began dialysis during the follow-up period. Analysis of donor MAP scores revealed scores of 0, 1, 2, 3, 4, and 5 for 451, 36, 95, 96, 82, and 22 donors, respectively (Table S1, SDC, http://links.lww.com/TXD/A343). The number of donors with a MAP score of 1 to 5 was smaller than the number of donors with a MAP score of 0. There was no statistically significant difference in death-censored graft survival among patients according to MAP score (Figure S1, SDC, http://links.lww.
com/TXD/A343; *P* = 0.433). Similarly, there was no strong evidence of linear relationships between MAP score and operative time, estimated blood loss during donor nephrectomy, incidence of perioperative donor and recipient complications, or recipient eGFR at POD 7 (Table S1, SDC, http://links.lww.com/CTXD/A343). Notably, several previous reports concerning the relationships of the MAP score with donor nephrectomy outcomes have categorized the MAP score as 0 or >0 because of the small numbers of donors with a MAP score of 1 to 5, which influences the statistical strength.4,5 Therefore, we divided all transplants into 2 groups: those with donor MAP score of 0 (ie, MAP*0* group) and those with donor MAP score of 1–5 (ie, MAP*1–5* group). The numbers of patients in the MAP*0* and MAP*1–5* groups were 451 and 331, respectively. Patients’ clinical characteristics are shown in Table 1. The mean age, male-to-female ratio, and BMI were higher among donors in the MAP*1–5* group than among donors in the MAP*0* group (all *P* < 0.001). The prevalences of hypertension (*P* < 0.001), hyperlipidemia (*P* = 0.013), and diabetes (*P* < 0.001) were significantly higher among donors in the MAP*1–5* group than among donors in the MAP*0* group. The histories of CVD, which were myocardial infarction, stroke, and no atherosclerosis obliterans, were not significantly different between groups. While mean age was also higher among recipients in the MAP*1–5* group than among recipients in the MAP*0* group (*P* = 0.046), the male-to-female ratio was lower among recipients in the MAP*0* group than among recipients in the MAP*1–5* group (*P* < 0.001). Other clinical characteristics were comparable between the 2 groups.

The donor and recipient outcomes are shown in Table 2. The operative time and estimated blood loss were both significantly greater in the MAP*1–5* group than in the MAP*0* group (*P* = 0.034 and <0.001, respectively). The percentage of glomerular sclerosis (%GS) of 0-h donated kidney biopsy was significantly greater in the MAP*1–5* group than in the MAP*0* group. Although no significant differences were found in the incidences of slow graft function or DGF between the MAP*0* and MAP*1–5* groups, the recipient eGFRs at POD 7 and at 5 y after LDKT were significantly higher in the MAP*0* group than in the MAP*1–5* group. The incidence of biopsy-proven acute rejection within 1 y after transplantation did not significantly differ between groups. Donor complications according to the classification by Köck et al13 are shown in Table 3; there were no statistically significant differences in the incidences and details of complications between the 2 groups.

The death-censored cumulative graft survival rates in the MAP*0* and MAP*1–5* groups were 98.7% and 98.5% at 1 y and 95.6% and 94.2% at 5 y, respectively (Figure 1); these rates did not significantly differ between groups (*P* = 0.769). The respective patient survival rates in the MAP*0* and MAP*1–5* groups were 99.6% and 99.1% at 1 y, while they were 96.2% and 97.7% at 5 y (Figure 2); these rates also did not significantly differ between groups (*P* = 0.476). The recipient mean eGFR from POD 1 to 7 was significantly greater in the MAP*0* group than in the MAP*1–5* group (*P* = 0.007, Figure 3). The recipient mean eGFRs from 1 to 5 y after transplantation are shown in Figure 4. Although the mean eGFR was lower in the MAP*0* group than in the MAP*1–5* group at all points, the eGFR reduction was comparable between groups. Multivariate analysis revealed that a MAP score of 0 did not affect recipient eGFR at POD 7 (*P* = 0.514, Table 4). The factors affecting recipient eGFR at POD 7 were donor age, recipient age, and female sex (*P* < 0.001, <0.001, and = 0.004, respectively).

### DISCUSSION

The key findings in this study were that donor MAP score >0 was associated with longer operative time and increased blood loss during donor nephrectomy but not with donor and recipient outcomes (eg, surgical complications and graft and recipient survival).

In this study, donors in the MAP*1–5* group had higher BMI and included a greater proportion of men, compared with donors in the MAP*0* group. Previous studies showed that old age, male sex, and high BMI were associated with APF and higher MAP score.2,7,11,12 In addition, we found that recipients in the MAP*1–5* group were older and included a greater proportion of women. This finding might have been influenced by the spousal relationship between the donor and recipient in 36.2% of transplants included in our study. The greater number of male donors might have been associated with the greater number of female recipients in the MAP*1–5* group. The prevalences of hypertension, hyperlipidemia, and diabetes were significantly higher among donors in the MAP*1–5* group than among donors in the MAP*0* group. This was expected because donors in the MAP*1–5* group were significantly older, had higher BMI, and were more likely to be men.

The operative time and blood loss during donor nephrectomy were both greater in the MAP*1–5* group than in the MAP*0* group. The MAP score was originally devised to predict intraoperative APF, which can prolong operative time and increase estimated blood loss during minimally invasive partial nephrectomy.1,7 It has also been reported that MAP score >0 is associated with longer operative time during HALDN.

### TABLE 1.

Comparison of kidney transplant donors and recipients according to MAP score group

| Variable                      | MAP score 0 (n = 451) | MAP score 1–5 (n = 331) | *P*   |
|-------------------------------|-----------------------|-------------------------|-------|
| **Donors**                    |                       |                         |       |
| Age, mean ± SD, y             | 53.2 ± 11.9           | 58.7 ± 10.6             | <0.001|
| Women:men, n                  | 361:90                | 124:207                 | <0.001|
| Body mass index, mean ± SD, kg/m² | 21.9 ± 2.9            | 24.3 ± 3.1              | <0.001|
| Hypertension, n (%)           | 40 (8.9)              | 97 (29.3)               | <0.001|
| Hyperlipidemia, n (%)         | 32 (7.1)              | 41 (12.4)               | 0.013 |
| Diabetes, n (%)               | 4 (0.9)               | 16 (4.8)                | <0.001|
| History of CVD, n (%)         | 5 (1.1)               | 1 (0.3)                 | 0.176 |
| Right nephrectomy, n (%)      | 2 (0.4)               | 3 (0.9)                 | 0.426 |
| HLDN, HARDN, PRDN, n          | 139:290:22            | 74:247:10               | 0.008 |
| **Recipient**                 |                       |                         |       |
| Age, mean ± SD, y             | 43.4 ± 17.0           | 45.7 ± 14.6             | 0.046 |
| Women:men, n                  | 151:300               | 165:168                 | <0.001|
| Retransplantation, n (%)       | 15 (3.3)              | 14 (4.2)                | 0.511 |
| HLA mismatches, mean ± SD, n  | 3.0 ± 1.5             | 2.9 ± 1.5               | 0.116 |
| ABO incompatibility, n (%)    | 133 (29.5)            | 101 (30.5)              | 0.758 |
| Rituximab, n (%)              | 159 (35.3)            | 122 (36.9)              | 0.645 |
| Preoperative donor-specific antibody, n (%) | 50 (11.1)              | 48 (14.5)               | 0.156 |
| Dialysis period, mean ± SD, mo | 10.6 (4–45)          | 13.1 (10–52.4)          | 0.220 |
| Follow-up duration, mean ± SD, mo | 75.0 ± 38.8           | 79.2 ± 35.8             | 0.120 |

CVD, cerebrocardiovascular disease; HLDN, hand-assisted laparoscopic donor nephrectomy; HARDN, hand-assisted retroperitoneoscopic donor nephrectomy; IQR, interquartile range; MAP, Mayo Adhesive Probability; PRDN, pure-retroperitoneoscopic donor nephrectomy.
although this association was statistically significant only for male donors.\(^4\) Our results were consistent with the findings of previous studies. However, relationships between the MAP score and perioperative donor nephrectomy complications have not been reported. Heimbach et al\(^{13}\) reported that donors with BMI ≥ 35 kg/m\(^2\) and donors with BMI < 25 kg/m\(^2\) had similarly low rates of major surgical complications (eg, conversion to open surgery and reoperation). The current study showed that the MAP score does not influence the rate of donor surgical complications. We also compared the rates of surgical complications between groups according to surgical procedure (HARDN, HALDN, and PRDN), which showed comparable results among procedures. While the incidence of recipient complications of Clavien–Dindo grade III or higher attributable to donor factors was 13.6% (3 of 22 patients) among recipients with a donor MAP score of 5; this incidence was statistically significantly higher than the incidence observed among other recipients (Table S1, SDC, http://links.lww.com/TXD/A343). All 3 complications were related to vesicoureteral anastomosis: 1 involved leakage and the others involved stenosis. Considering these findings, a MAP score of 5 might be associated with graft ureteral ischemia. However, we have intraoperatively placed ureteral stents in all patients since October 2015 and have not encountered any complications of Clavien–Dindo grade III or higher related to vesicoureteral anastomosis in recipients with a donor MAP score of 5. Therefore, vesicoureteral anastomosis should be carefully monitored for transplants involving a donor MAP score of 5. Donors with a high MAP score tend to have high BMI; notably, donors with obesity have been reported to carry a risk of DGF.\(^{14,15}\) However, we found no difference in the incidence of DGF between groups in this study. Moreover, multivariate analysis showed that donor BMI did not influence recipient kidney function after LDKT.

In this study, the recipient eGFR at 1 wk after LDKT was lower in the MAP1–5 group than in the MAP0 group. This result appears to support our hypothesis that the presence of APF and a high MAP score might influence LDKT recipient outcomes.

### TABLE 2.

Outcomes for donors and recipients

| Variable | MAP score 0 (n = 451) | MAP score 1–5 (n = 331) | P |
|----------|-----------------------|------------------------|---|
| Donors   |                       |                        |   |
| Operative time, mean ± SD, min | 180 ± 52              | 189 ± 66               | 0.034 |
| Estimated blood loss, mean ± SD, mL | 104 ± 156             | 191 ± 229              | <0.001 |
| Warm ischemia time, mean ± SD, min | 3.9 ± 1.9             | 4.0 ± 2.3              | 0.446 |
| Total ischemia time, mean ± SD, min | 152 ± 63              | 142 ± 58               | 0.025 |
| Postoperative stay, mean ± SD, d | 7.7 ± 2.5             | 8.0 ± 3.6              | 0.151 |
| Surgical complications, n (%) | 11 (2.4)              | 6 (1.9)                | 0.549 |
| Glomeruli per section of 0-h biopsy, mean ± SD, n | 25.8 ± 16.1           | 28.4 ± 17.7            | 0.050 |
| (n = 408) | (n = 263)             |                        |   |
| Glomerular sclerosis of 0-h biopsy, mean ± SD, % | 11.4 ± 13.4           | 14.5 ± 14.4            | 0.004 |
| (n = 408) | (n = 263)             |                        |   |
| Recipients |                       |                        |   |
| CD grade III or higher surgical complications, n (%)\(^a\) | 10 (2.2)              | 15 (4.5)               | 0.071 |
| Slow graft function, n (%) | 23 (5.1)              | 23 (7.0)               | 0.359 |
| Delayed graft function, n (%) | 13 (2.9)              | 14 (4.2)               | 0.223 |
| eGFR at POD 7, mean ± SD, mL/min/1.73 m\(^2\) | 61.2 ± 36.2           | 53.8 ± 35.1            | 0.004 |
| eGFR at 5 y after transplantation, mean ± SD, mL/min/1.73 m\(^2\) | 53.1 ± 18.4 (n = 231) | 46.6 ± 16.5 (n = 184) | <0.001 |
| Biopsy-proven acute rejection within 1 y after transplantation, n (%) | 62 (13.8)             | 40 (12.1)              | 0.494 |

\(^a\)Recipient surgical complications were counted only when they could possibly be attributed to donor factors, excluding delayed graft function.

CD, Clavien–Dindo classification; eGFR, estimated glomerular filtration rate; MAP, Mayo Adhesive Probability; POD, postoperative d.

### TABLE 3.

Donor complications according to the classification by Kocak et al\(^{10}\)

| Variable, n (%) | MAP score 0 (n = 451) | MAP score 1–5 (n = 331) | P |
|----------------|-----------------------|------------------------|---|
| 1. Nonlife-threatening complications (total) | 7 (1.6)              | 2 (0.6)                | 0.202 |
| Ileus resolving spontaneously | 3 (0.7)              | 2 (0.6)                | 0.916 |
| Surgical site infection | 3 (0.7)              | 0 (0)                  | 0.069 |
| Pressure ulcer | 1 (0.2)              | 0 (0)                  | 0.294 |
| 2. No residual disability |                       |                        |   |
| 2a. Requires use of only medication (total) | 3 (0.7)              | 2 (0.6)                | 0.916 |
| Acute pancreatitis | 1 (0.2)              | 0 (0)                  | 0.294 |
| Blood loss >500 mL | 2 (0.4)              | 1 (0.3)                | 0.749 |
| Pneumonia | 0 (0)              | 1 (0.3)                | 0.190 |
| 2b. Requires additional therapeutic intervention (total) | 1 (0.2)              | 3 (0.9)                | 0.183 |
| Small-bowel obstruction requiring operative procedures | 1 (0.2)              | 2 (0.6)                | 0.395 |
| Colectomy secondary to colon injury | 0 (0)              | 1 (0.1)                | 0.190 |
| 3. Residual disability | 0 (0)              | 0 (0)                  | – |
| 4. Renal failure or death | 0 (0)              | 0 (0)                  | – |
| Total | 11 (2.4)              | 6 (1.8)                | 0.549 |

MAP, Mayo Adhesive Probability.
Based on a previous study in which the presence of APF and a high MAP score influenced kidney function in LDKT recipients with type 2 diabetes, the eGFR reductions within 5 y after LDKT were similar in both groups. In addition, the recipient mortality and incidence of acute rejection within 1 y after LDKT did not significantly differ between the 2 groups.
These results reveal that a high donor MAP score was associated with low initial renal function but not with midterm renal transplantation outcomes (ie, within 5 y after LDKT). Moreover, multivariate analysis showed that the MAP score itself did not influence eGFR at 1 wk after LDKT. Multivariate analysis showed that older donor age, older recipient age, and male recipient sex were associated with low postoperative recipient eGFR. To explain this result, we carefully considered the association between the MAP score and the %GS of 0-h donated kidney biopsy. Escofet et al\textsuperscript{16} reported that high %GS of deceased donors at the time of transplantation was associated with both recipient postoperative kidney function and
TABLE 4.
Factors affecting recipient eGFR at postoperative d 7

| Variable             | \( \beta \)  | SE  | t    | P      |
|----------------------|--------------|-----|------|--------|
| MAP score 0          | 1.029        | 1.462 | 0.70 | 0.482  |
| Donor age, y         | −0.970       | 0.108 | −8.97| <0.001 |
| Female donor         | 0.612        | 1.425 | 0.43 | 0.669  |
| Donor BMI, kg/m²     | 0.106        | 0.397 | 0.27 | 0.790  |
| Recipient age, y     | −0.400       | 0.075 | −5.32| <0.001 |
| Female recipient     | 3.721        | 1.268 | 2.93 | 0.003  |

BMI, body mass index; eGFR, estimated glomerular filtration rate; MAP, Mayo Adhesive Probability.

graft survival. Therefore, glomerular sclerosis might have contributed to the difference in postoperative eGFR between the 2 groups in our study. However, Pokorná et al
d reported that %GS was associated with graft function in a simple regression analysis, although it did not remain significantly associated with graft function when donor age was considered. Thus, we conclude that %GS is associated with graft function but is greatly affected by donor age.

In this study, the prevalences of hypertension, hyperlipidemia, and diabetes were significantly higher among donors in the MAP1–5 group than among donors in the MAP0 group. Because these comorbidities are known as risk factors of renal failure, we performed multivariate analysis using donor and recipient ages; recipient sex; and donor hypertension, hyperlipidemia, and diabetes status as analysis variables. Notably, these comorbidities were not associated with a significant risk of lower eGFR at 1 wk after transplantation (Table S2, SDC, http://links.lww.com/TXD/A343). These results suggest that these comorbidities (eg, hypertension, hyperlipidemia, and diabetes) do not greatly affect renal function after LDKT if they are well controlled.

Cockerill et al6 reported that the nondonated kidney MAP score influenced postoperative donor eGFR, and Holscher et al18 reported that postoperative donor kidney function can predict graft survival after LDKT. Therefore, we investigated the relationship between donor MAP score and graft survival, but we found no difference in graft survival rate between the 2 groups in this study. We also compared graft survival rates between the 2 groups according to the operation performed (HARDN, HALDN, or PRDN) and found no significant differences among surgical procedures; additionally, there were no significant differences in graft survival rates according to donor MAP score when continuous score values were used. In the study by Holscher et al, the association between donor nephrectomy eGFR and recipient death-censored graft loss became weaker with increasing donor BMI. Thus, they suggested that postoperative eGFR is related to the capacity for hypertrophy of the remaining kidney, but donors with obesity have already developed obesity-related glomerulomegaly and might have a reduced capacity for hypertrophy in response to donation.14 Considering that donors with a high MAP score tended to have higher BMI, our finding that graft survival rates did not differ between the MAP0 and MAP1–5 groups is consistent with the explanation by Holscher et al. However, lower postoperative eGFR has been associated with a higher rate of graft failure.13 Therefore, the long-term graft survival rate might be lower in the MAP0 group than in the MAP1–5 group if the follow-up period was extended beyond the interval analyzed in the current study.

Although this study showed significant differences in postoperative %GS and eGFR between the 2 groups, there was no significant difference in %GS according to MAP score when continuous score values were used; moreover, the relationship between eGFR at POD 7 and continuous MAP score values did not demonstrate a clear positive correlation. We speculate that the MAP score is influenced by many factors such as age and sex; notably, %GS and eGFR after LDKT were more affected by age and sex than by the MAP score itself. Considering these results, it is inappropriate to apply the MAP score alone as a donor criterion for LDKT.

This study had several limitations. First, because it was retrospective study and the MAP score was associated with many factors such as donor age and sex, it was difficult to avoid bias when evaluating the effects of the MAP score on postoperative kidney function. Second, because the data were collected over >11 y, surgeons and surgical procedures changed during the study period; these changes could have influenced the study results. However, to the best of our knowledge, this study is the first report concerning the relationship between donor MAP score and recipient outcome; the findings suggest that the donor nephrectomy safety and long-term recipient outcomes are independent of the donor MAP score.

CONCLUSIONS

The results of this study suggest that the MAP score is useful for predicting donor nephrectomy difficulty; however, it does not influence donor and recipient surgical complications or graft survival. Thus, the MAP score should not affect donor selection.

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