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Risk Factors for Renal Allograft Compartment Syndrome

Jorge Ortiz[1], Afshin Parsikia[1], Mindy M. Horrow[2], Kamran Khanmoradi[1], Stalin Campos[1], Radi Zaki[1]

[1]*Department of Transplant Surgery, Einstein Medical Center, Philadelphia, Pennsylvania, USA*

[2]*Department of Radiology, Einstein Medical Center, Philadelphia, Pennsylvania, USA*

Renal allograft compartment syndrome (RACS) is graft dysfunction secondary to intracompartment hypertension. The purpose of this study was to identify risk factors for RACS. We reviewed 7 cases of established RACS and all intra-abdominal placements of the kidney in order to include potential RACS. We also studied early graft losses in order to rule out a missed RACS. We compared the allograft length and width, recipient height, weight, body mass index, aberrant vessels, site of incision, and side of kidney with the remainder of the cohort as potential predictors of RACS. Among 538 transplants, 40 met the criteria for actual RACS or potential RACS. We uncovered 7 cases of RACS. Only kidney length and width were statistically significant ($P = 0.041$ and 0.004, respectively). The width was associated with a higher odds ratio than was length (2.315 versus 1.61). Increased allograft length and width should be considered as a potential risk for RACS.

Key words: Kidney – Transplant – Compartment Syndrome

Intracompartment hypertension leading to tissue ischemia has been described in the extremities,[1,2] the abdomen,[3] after liver transplantation,[4] and after kidney transplantation.[5,6] The incidence of abdominal compartment syndrome ranges from 0.5% to 8%.[7] Risk factors include massive crystalloid infusion, multiple transfusions, hypothermia, acidosis, and body mass index (BMI) greater than 30.[3]

The renal allograft compartment syndrome (RACS) has only been described in case reports and small series.[5,6] The kidney is placed at risk when intracompartment hypertension in the confined retroperitoneal space leads to ischemia. The diagnosis is frequently entertained in the operating room or in the postanesthesia care unit when a previously well-functioning/appearing kidney ceases to create urine. Ultrasound reveals extremely

Corresponding author: Afshin Parsikia, Department of Transplant Surgery, Einstein Medical Center, 5501 Old York, Philadelphia, PA 19141.
Tel.: 2154567827; E-mail: parsikia@einstein.edu

diminished color Doppler flow. Spectral Doppler findings are variable and include either elevated systolic velocities and distal parvus tardus waveforms typical of a renal artery stenosis, or very low systolic velocities with normal waveforms. Color flow is well maintained in most cases of Acute Tubular Necrosis (ATN), and spectral Doppler waveforms are normal, though the resistive index may be elevated. The diagnosis is confirmed, and the graft may be salvaged upon urgent reexploration. The kidney may initially appear dusky and soft. When the intracompartment hypertension is relieved, the kidney will become pink and tumescent. The poor flow may be a direct result of compression of the organ, or the limited space may lead to kinking of the artery or vein. This phenomenon was first described in adults in 1996.[8] The incidence appears to be about 2%.[6] This may be an underrepresentation, because many primary nonfunctions, vascular thromboses, and other early graft losses may have been undiagnosed RACS. Additionally, the RACS may have been prevented by prophylactic measures such as intra-abdominal placement of the kidney or the use of a synthetic mesh hood.[5] We have attempted to determine the true incidence and risk factors for RACS by reviewing (1) our transplants with known diagnoses of RACS; (2) those patients who suffered early graft losses, in order to rule out undiagnosed RACS; (3) and all patients who underwent intra-abdominal placement of the kidney (or other wound manipulations) for the purpose of avoiding RACS.

Methods

In this study, which complied with the Health Insurance Portability and Accountability Act and was approved by the institutional review board, informed consent was waived. This study consisted of a retrospective review of our renal transplant database and hospital electronic records from December 2004 through November 2011. Dual kidney transplants, infant kidneys, and combined transplantations were excluded. In order to capture all potential RACS, we reviewed all early graft losses, including those with a diagnosis of compartment syndrome, vascular thrombosis, rejection, "unknown," and primary nonfunction. We also closely evaluated the operative dictations of all intra-abdominal placements of the kidney in order to include the *fear of compartment syndrome/vascular kink* cases. The combination of these patients and patients who subsequently developed actual compartment syndrome was called *patients at risk* of compartment syndrome (study group). In order to have a confirmed diagnosis of compartment syndrome, we focused on cases with available ultrasound studies within 4 hours of transplant. Images of all abnormal studies were reviewed by a senior radiologist. We compared the study group to the rest of the cohort without potential or actual development of RACS (control group).

Statistical analysis

We ran a logistic regression analysis to evaluate the allograft length, allograft width, recipient height, recipient weight, donor BMI aberrant vessels, site of incision, prior peritoneal dialysis, and left or right kidney as potential predictors of RACS. The length and width of the donor kidney was obtained from files from the organ procurement agency and from the immediate postoperative ultrasound report. All deceased donor right kidneys were transplanted with a vena cava extension. Subsequently, we ran a multivariate logistic regression with variables that were statistically significant in the univariate analysis. The calibration of the model was tested with a Hosmer-Lemeshow test. Additionally, we built a second model specifically for the 7 patients with actual compartment syndrome. All variables were tested for normality. Data are presented as mean $\pm$ SD. Group differences were calculated using the Student t test for continuous variables and the χ^2 test for ordinal variables. The 1-year graft survival between the 2 groups was compared using Kaplan-Meier methodology (Fig. 1).

Results

A total of 502 kidney transplants met the inclusion criteria. There were 4 retransplants. We found 7 transplants with surgically confirmed compartment syndrome. That is, 7 patients were reexplored after wound closure when the clinical and radiologic findings were consistent with RACS. In all cases, the kidneys' color improved and the graft became tumescent. Doppler ultrasonography also revealed improved flow and resistive indices. There were 33 transplants with the potential for compartment syndrome. We searched the data for the existence of any early graft loss that could have been due to RACS. We had a total of 12 primary nonfunctions (2.3%), 3 early rejections (0.59%), and 7 vascular thromboses (1.39%). Review of the inpatient charts, the operative notes, and the discussions with the

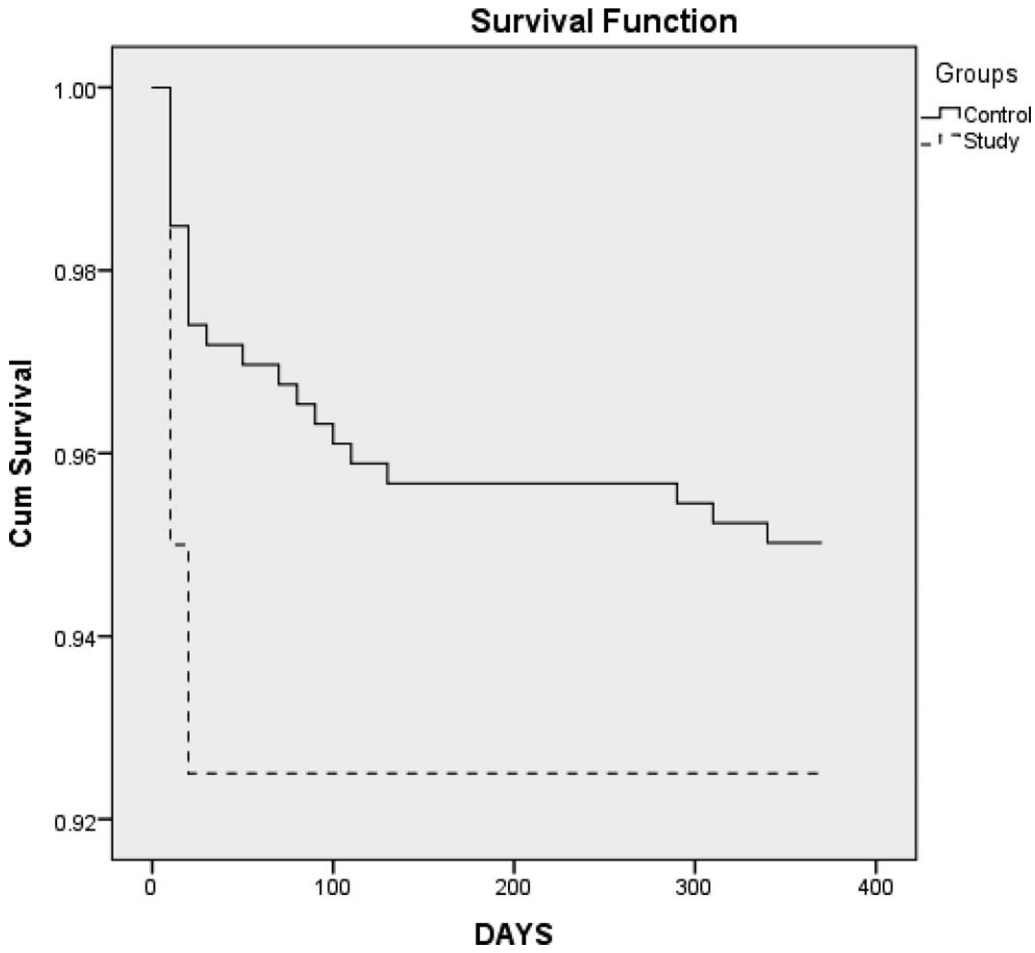

Fig. 1 Kaplan-Meier curve. One-year graft survival ($P = 0.471$).

surgeons of record revealed that none of the transplants was performed because of compartment syndrome. Therefore, there were a total 40 transplants ($33 + 7$) in the study group. The remainder of the cohort comprised the control group (n = 462). The demographic information is tabulated in Table 1. The overall rate of actual compartment syndrome in our cohort was 1.39% (7/502). The overall rate of patients at risk of developing compartment syndrome in our study was 7.96% (40/502). The difference in 1-year survival rates was not statistically significant between the 2 groups ($P = 0.471$; Fig. 1). Donor BMI was not statistically significant ($P = 0.372$) between the 2 groups. There was no statistically significant difference in delayed graft function rates between the 2 groups ($P = 0.161$).

We tested potential risk factors using a univariate logistic regression. The regression models used the following variables: allograft length, allograft width, recipient height, recipient weight, donor BMI, aberrant vessels, site of incision, prior peritoneal dialysis, and left or right kidney (Table 2). In the univariate analysis, the allograft size (length and width), recipient height, and right or left kidney were associated with a P value less than 0.2.

A multivariate logistic regression model was constructed with the eligible variables derived from our univariate analysis. In the multivariate analysis, the allograft length and width stayed significant ($P = 0.047$ and 0.006, respectively), and the other 2 covariates lost significance. This model was tested for colinearity by measuring the variance inflation factor. Colinearity was not observed. In our analysis, the allograft width was associated with a greater odds ratio than the allograft length: 1.367 [95% confidence interval (CI), 1.005–1.861] versus 1.926 (95% CI, 1.204–3.076). The model was well calibrated. The results of the multivariate logistic regression are demonstrated in Table 3.

Table 1 Demographic information

Variable	All (n = 502)	Study group (n = 40)	Control group (n = 462)	*P* value
Recipient age (SD), y	54.75 (12.25)	55.61 (13.01)	54.67 (12.1)	0.644
Recipient gender, male (%)	332 (66.1)	29 (72.5)	303 (65.6)	0.486
Recipient height (SD), cm	171.02 (10.30)	174.14 (8.6)	170.74 (10.40)	**0.046**
Recipient weight (SD), Kg	82.90 (20.22)	83.37 (20.66)	82.86 (20.2)	0.879
Recipient BMI (SD), kg/m^2	28.21 (5.86)	27.46 (6.45)	28.27 (5.81)	0.404
Single artery and vein (%)	399 (79.5)	29 (72.5)	370 (80.1)	0.306
Site of incision, right (%)	417 (83.2)	35 (87.5)	382 (82.9)	0.658
Right kidney (%)	224 (44.6)	22 (55)	202 (43.7)	0.187
Peritoneal dialysis	16 (3.2)	1 (2.5)	15 (3.2)	0.631
Kidney length (SD), cm	11.47 (1.29)	12.2 (1.19)	11.41 (1.28)	<**0.001**
Kidney width (SD), cm	5.54 (0.79)	6.02 (0.835)	5.49 (0.773)	<**0.001**

BMI, body mass index.

We repeated the logistic regression analysis again specifically for the 7 patients with actual compartment syndrome. Among the tested variables, only kidney width was statistically significant ($P = 0.022$), with odds ratio 3.242 (95% CI, 1.184–8.878). Kidney length did not reach significance in our second logistic regression analysis.

Discussion

RACS is an underrecognized cause of early allograft dysfunction that can be treated with early operative intervention and prevented by careful examination of the allograft and its position within the confined retroperitoneal space. Suggested risk factors include kidney-recipient size or donor-recipient size mismatch, a shallow male pelvis,[9] excessive administration of intravenous fluids, low preoperative hypoalbuminemia (in cirrhotics), and a noncompliant retroperitoneum secondary to peritoneal dialysis catheter-related infections.[10,11] Because our program is an aggressive, deceased-donor transplant service with a high incidence of delayed graft function, we routinely perform bedside ultrasound examinations in the postanesthesia care unit. Markedly decreased color Doppler flow in the kidney, with either high velocities in the main renal artery and parvus tardus waveforms more distally or high-resistance flow with low velocities in the main renal artery in the absence of extrinsic space occupying lesions, alerts us to the possibility of RACS.[9,12,13] Color flow is well maintained in almost all cases of ATN, and spectral Doppler waveforms are normal though the resistive index may be elevated.

Although RACS has been reported in the setting of bilateral, dual, retroperitoneal kidney transplantation in a patient with cirrhosis,[10] we decided to exclude dual transplants from our report in order to simplify the analysis. It is our policy to place both kidneys ipsilaterally in the retroperitoneum. We have never seen RACS in this scenario, but we have a low threshold to place the kidneys intra-abdominally in order to avoid RACS. Similarly, we excluded infant donor kidneys, both single and en bloc, from evaluation in order to provide a consistent analysis.

Our incidence of confirmed RACS was 1.4%. This is slightly lower than previously reported.[6] Fortunately, all 7 allografts were salvaged after RACS was diagnosed with screening ultrasound in the postanesthesia care room. We documented the utility of this policy in a recent publication.[13]

Our careful review of the patient records indicates that 8% of transplants were at risk for RACS. The only potential complication associated with intra-abdominal placement of the 33 kidneys was an incisional hernia. There were no torsed kidneys or bleeding episodes associated with posttransplant

Table 2 Univariate logistic regression for 40 patients at risk of compartment syndrome

Variable	*P* value	Odds ratio	95% CI
Kidney length	<**0.001**	1.61	1.250–2.094
Kidney width	<**0.001**	2.315	1.489–3.602
Single artery and vein kidney	0.257	0.656	0.316–1.361
Recipient height	**0.047**	1.034	1.0004–1.068
Recipient weight	0.879	1.001	0.985–1.017
Recipient BMI	0.403	0.976	0.921–1.034
Donor BMI	0.372	1.021	0.975–1.069
Peritoneal dialysis	0.797	0.7640.98	0.98–5.939
Site of incision	0.454	1.448	0.550–3.811
Which kidney	**0.172**	1.573	0.822–3.012

BMI, body mass index; CI, confidence interval.

Note: the numbers in bold were considered eligible for multivariate analysis.

Table 3 Multivariate logistic regression for 40 patients at risk of compartment syndrome

Variable	*P* value	Odds ratio	95% CI
Kidney length	**0.047**	1.367	1.005–1.861
Kidney width	**0.006**	1.924	1.204–3.076
Recipient height	0.085	1.032	0.996–1.070
Which kidney	0.063	1.971	0.964–4.031

CI, confidence interval.

biopsies. The use of a mesh hood fascial closure technique has also been shown to be a safe method to avoid RACS.[5]

Of all the variables studied, only the length and width of the kidney in potential RACS and the width of the kidney in actual RACS were risk factors. Therefore, we recommend that relatively large kidneys be considered for intraabdominal placement. Surprisingly, we found no other donor (including the use of right kidneys) or recipient variable that could reliably predict RACS.

The effect of large donor kidneys on recipient outcomes has been studied, with mixed results. Surrogates such as donor BMI or donor weight have been associated with delayed graft function and primary nonfunction.[14–17] In one study of the Organ Procurement and Transplantation Network database, donor BMI was an independent predictor of delayed graft function.[16] It is possible that some primary nonfunctions may have actually been undiagnosed RACS. Similarly, it is possible that intracompartment hypertension may be temporary in some cases, leading to delayed graft function. In our study, donor BMI was not an independent risk factor. Additionally, our delayed graft function rates were similar between the two groups.

Similar to the results of other published reports,[14–17] these large kidneys were not associated with worse allograft outcomes.

Weaknesses of this study include its retrospective nature and its relatively small sample size. Nevertheless, it is the first attempt to properly estimate the actual risk of compartment syndrome by evaluating RACS. We carefully reviewed the operative notes to determine whether an allograft was truly at risk of loss from RACS. It is conceivable that the dictations over- or underestimated this risk or were incomplete. Additionally, there is a possibility that there was a bias toward placing all large kidneys intra-abdominally, thereby decreasing the incidence of RACS. This would be unlikely because it is the preference of this program to avoid abdominal placement if at all possible. RACS as a cause for graft loss or the salvage of a graft suffering from ischemia due to RACS is not robustly collected in any large database. Therefore, we are reliant on single-center reports.

Conclusion

RACS is an underreported and underdiagnosed phenomenon that may lead to graft loss and delayed graft function. Preoperative risk factors have not been previously identified. We did not confirm causes speculated in other case series. Despite a robust attempt to analyze all possible prognosticators, the only factor associated with development of RACS was increased donor kidney size. Further studies are warranted in order to discern preoperative or intraoperative variables associated with this serious complication. In order to minimize potential graft loss, we recommend a low threshold for intra-abdominal placement of large allografts and immediate postoperative sonographic surveillance.

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