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Simultaneous pancreas and kidney (SPK) retransplantation in prior SPK recipients

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Abstract

Introduction—We have performed 113 renal and 28 isolated pancreas retransplants in our cohort of more than 1200 prior simultaneous pancreas and kidney (SPK) recipients. On the basis of these experiences, we began performing repeat SPK in prior SPK recipients (n = 9).

Methods—This retrospective review summarizes our experience with repeat SPK transplantation in prior SPK recipients. Mean age at retransplant was 39 yr; mean interval to retransplant was 7.8 yr. Thirty-three percent were pre-dialysis. Eighty-nine percent of patients underwent transplant nephrectomy (five during the repeat SPK and three prior to it), and 78% underwent transplant pancreatectomy (four during the repeat SPK and three prior to it). Enteric drainage was performed in all repeat SPKs.

Results—Median length of stay was 11 d. Perioperative complications included the following: renal artery thrombosis (1), pancreatic portal venous thrombosis (1), enteric leak (1), and hematoma (2). Overall pancreatic allograft survival was 78% at one yr and 67% at two yr. Overall renal allograft survival was 89% at one yr and 78% at two yr. Patient survival at one and three yr was 100%.

Conclusions—Survival of repeat SPK allografts is acceptable despite the increased technical and immunologic demands of retransplantation. Graftectomy prior to or at the time of retransplantation is often necessary.

Keywords

kidney transplantation; pancreas transplantation; retransplantation

Major advances in the surgical technique of pancreas transplantation (1–4) in the early 1980s, as well as the development of novel immunosuppressive agents, led to a marked increase in the number of pancreas transplants performed worldwide over the past 2½ decades. As the typical pancreatic allograft half-life in simultaneous pancreas and kidney (SPK) recipients is estimated to be 11 yr (5), surgeons are increasingly called on to evaluate

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patients for retransplantation. Retransplantation can pose a number of technical challenges, particularly in prior SPK transplant recipients as both iliac vessels have been previously accessed. The outcomes of isolated pancreatic and renal retransplantation in prior SPK recipients have been reported (4, 6–12). However, SPK retransplantation in prior SPK recipients has been only briefly noted in reports from our center and from the Minnesota group (4, 13). There have thus far been no reports describing the technical complexities and complications associated with SPK retransplantation in prior SPK recipients.

Since 1985, our center has performed more than 1200 SPK transplants, and, consequently, an increasing number of patients are eligible to undergo retransplantation. We have performed 113 isolated renal and 28 isolated pancreas retransplants in prior SPK recipients. On the basis of these experiences, we began performing repeat SPK transplantation in prior SPK recipients.

Patients and methods

A retrospective review of all adult SPK retransplantations in prior SPK recipients (n = 9) performed between December 1, 1985, and December 31, 2008, was conducted utilizing the University of Wisconsin prospectively collected transplant database. Operative details were obtained from operative reports where necessary. The study was conducted under Institutional Review Board approval.

The primary outcomes of interest were patient, renal allograft, and pancreatic allograft survival. Pancreatic allografts were determined to be functional prior to renal retransplantation if patients maintained normoglycemia without exogenous insulin therapy or retransplantation. Pancreatic allograft loss was defined as permanent (>6 months) return to exogenous insulin use, retransplantation, or death with a functioning graft. Renal allograft failure was defined as a return to dialysis, retransplantation, or death with a functioning graft.

The immunosuppressive regimen utilized in the retransplantation evolved over time as novel immunosuppressants were introduced. Induction therapy was utilized in all instances given the exposure to prior allograft antigens and presumed sensitized status. A variety of agents, including rabbit antithymocyte globulin (ATG) (thymoglobulin, Genzyme, n = 4), alemtuzumab (Campath-1H, ILEX, n = 3), and equine anti-thymocyte globulin (ATGAM, Upjohn, n = 2), were used. One patient also received rituximab (Rituxan; Genentech, San Francisco, CA, USA), and another underwent plasmapheresis with IVIG. The present induction regimen consists of basiliximab for lower-risk recipients and ATG for highly sensitized recipients.

All patients received dexamethasone or methylprednisolone at the time of retransplantation, and maintenance therapy consisted of either mycophenolate mofetil (CellCept; Roche, Basel, Switzerland) or mycophenolic acid (Myfortic; Novartis, Basel, Switzerland), tacrolimus (Prograf; Astellas, Tokyo, Japan), and low-dose prednisone. Our current maintenance immunosuppressive regimen consists of mycophenolic acid, tacrolimus, and low-dose prednisone. Prednisone was tapered during the transplant hospitalization to 30 mg/d. This dose was tapered further over the first post-operative months to a baseline of 5–10 mg/d.

In the majority of cases, cytomegalovirus (CMV) prophylaxis consisted of valganciclovir for CMV-negative recipients of CMV-positive donor organs and those given thymoglobulin induction and acyclovir for all other donor-recipient combinations for three months. Trimethoprim/sulfamethoxazole (160 mg/800 mg daily for one yr) was used for

Pneumocystis carinii prophylaxis. Mucosal candidiasis prophylaxis was with oral nystatin or clotrimazole tablets for three months.

Renal biopsies were performed in patients with a creatinine elevated at least 20% above baseline, and pancreas biopsies were performed in patients with unexplained amylase or lipase elevations and/or hyperglycemia. All biopsies were scored using hematoxylin and eosin staining per the Banff criteria (14, 15). C4d staining to diagnose antibody-mediated rejection was introduced in 2002 for renal allograft biopsies and in 2006 for pancreas allograft biopsies.

Mean follow-up was 6.2 yr (range: 2.5–12.3).

Results

Demographics

Recipient—Recipient demographics at the time of retransplantation are shown in Table 1. All recipients were Caucasian, and 78% were women. Mean age at the time of retransplantation was 39 yr; mean interval to retransplant was 7.8 yr (range: 2.6–12.5). Average BMI was 24 (range: 19–30). Thirty-three percent were pre-dialysis. Of those on dialysis prior to retransplantation, only one was on peritoneal dialysis. Average time on dialysis prior to retransplantation was 19 months (range: 2–48). All patients had pre-transplant hypertension. Mean panel reactive antibody (PRA) at retransplantation was 10% (range: 0–67). Mean number of HLA mismatches was four (range: 2–6).

Donor—Donor demographics for the SPK retransplantation are shown in Table 2. All donors were Caucasian, and 56% were male. Mean donor age was 28 yr, and mean donor BMI was 21 kg/m^2. Two donors donated after cardiac death (DCD). Mean cold ischemia time was 15 h for the pancreas and 16 h for the renal allograft. Organs were preserved in University of Wisconsin solution.

Cause of primary allograft failure

The most common cause of primary allograft failure leading to repeat SPK transplantation was immunologic loss of both allografts. Mean primary renal allograft survival was 6.2 yr (range: 0.1– 11.9 yr), and causes of renal allograft loss included chronic rejection (n = 6), acute rejection (n = 2), and infection (n = 1). Primary pancreatic allograft survival was slightly lower at 4.8 yr (range: 0.6– 10.3 yr), and losses occurred secondary to rejection (n = 5), chronic graft failure (n = 2), graft thrombosis (n = 1), and insulin resistance (n = 1).

Surgical techniques in retransplantation

The surgical techniques utilized prior to, and at the time of, SPK retransplantation are summarized in Table 3.

Enteric conversion—Bladder drainage was originally performed in six patients, four of whom had undergone enteric conversion prior to their SPK retransplantation. Enteric conversion was performed as described previously (16). Briefly, the duodenal segment was excised from the bladder with a 0.5-cm rim of bladder using electrocautery. The cystotomy was then closed in two layers using absorbable suture. As the bladder-drained pancreas transplantation was performed with the pancreas in a head-down position to the right iliac vessels, enteric drainage in the conversion operation was performed to the terminal ileum. The terminal ileum is in an anatomically favorable position for this anastomosis, and the side-to-side anastomosis was hand-sewn in two layers using an outer interrupted permanent suture and an inner running absorbable suture. Changes to the enteric conversion technique

evolved over time, and in one early patient in this series, the ileum was divided proximal to the anastomosis to defunctionalize the segment to which the pancreas was anastomosed. Enteric continuity was then re-established with an ileocolostomy to the transverse colon.

Transplant pancreatectomy—Seventy-eight percent of patients underwent transplant pancreatectomy prior to repeat SPK transplantation (four during the repeat SPK procedure and three prior to it). Indications for pancreatectomy prior to repeat SPK transplantation included recurrent urinary tract infections in a bladder-drained pancreas and an infarcted pancreatic allograft with anastomotic leakage. The most common reason for pancreatectomy was to provide adequate mobilization and space for a subsequent allograft. There were four instances in which this was performed during the repeat SPK procedure and one that was performed during a solitary kidney transplant after the original SPK operation. The latter patient later underwent repeat SPK transplantation.

Our standard technique of transplant pancreatectomy was to first isolate the pancreatic enteric anastomosis. This was typically performed by dividing the bowel proximal and distal to the duodenal segment anastomosis and re-establishing enteric continuity with a two-layered, hand-sewn end-to-end anastomosis. In select cases with favorable anatomy, the duodenal-enteric anastomosis was simply stapled across and imbricated with Lembert sutures, or the duodenal segment excised and the enterotomy closed transversely in two layers. This was performed only in instances where the bowel lumen was not narrowed. Vascular control was obtained by ligating the common iliac portion of the donor iliac Y-graft and clamping the portal anastomosis. The portal vein was then oversewn with a running non-absorbable suture after excising the allograft. In two cases of bladder-drained allografts, the pancreas graft was small, fibrotic, and asymptomatic. In these instances, the pancreas was not removed.

Transplant nephrectomy—Eighty-nine percent of patients underwent transplant nephrectomy (five during the repeat SPK procedure and three prior to it). In one instance, nephrectomy was performed prior to repeat SPK transplantation because of a failed, symptomatic renal allograft that had experienced severe rejection. In a fashion analogous to transplant pancreatectomy, nephrectomy was most commonly performed to enable adequate exposure and access for allograft implantation. In five patients, nephrectomy was performed to provide adequate exposure during the repeat SPK operation. Two patients underwent isolated renal retransplantation prior to repeat SPK transplantation. In these two patients, nephrectomy of the original SPK renal allograft was performed at the time of isolated renal retransplantation.

Transplant nephrectomy, when performed at the time of repeat SPK transplantation, was performed in an extracapsular fashion. The renal artery and vein were individually dissected and ligated high in the hilum. These vessels were assessed for suitability upon which to implant the second renal allograft. The renal vein of the primary allograft was used frequently for drainage of the repeat renal allograft given the difficulty in isolating a deep left iliac vein in a re-operative field. In one patient, a small, fibrotic, and asymptomatic renal allograft was left in place.

Repeat SPK transplantation—Retransplantation was performed via a midline incision. The portal vein of the pancreas was anastomosed to the distal inferior vena cava (IVC) in all cases, and in each instance, a suitable portion of the IVC was available despite the prior transplantation. An end-to-side anastomosis between the donor iliac Y-graft and the right common iliac artery was the preferred technique for obtaining inflow. In one instance, the right common iliac artery was completely calcified, and the right external iliac artery was used for inflow. Enteric drainage was performed in all repeat SPK transplants. In cases in

which the duodenal segment of the prior allograft was excised by dividing the jejunum above and below the allograft, a Roux limb was performed for enteric drainage (n = 2). Otherwise, enteric drainage was performed to an appropriate portion of jejunum roughly 30 cm distal to the ligament of Trietz. Early in our experience, the enteric drainage of the repeat pancreatic allograft was to the terminal ileum (n = 2).

The technique of renal reimplantation was more varied. The deeper location of the left iliac system made retransplantation technically challenging. Venous drainage was established with a number of techniques, including anastomosing the renal vein of the retransplant to the renal vein of the prior transplant (n = 3), the left common iliac vein (n = 3), the left external iliac vein (n = 2), and the gonadal vein (n = 1). In distinction to the prior renal transplant vein, the prior transplant artery was utilized to establish renal retransplant inflow in only one instance. Instead, a fresh anastomosis was typically made to the left common iliac artery (n = 6) or the left external iliac atery (n = 2). The ureter was implanted into the bladder using a standard Lich technique.

Additionally, several patients had undergone transplant nephrectomy and renal retransplantation (n = 2) in the interval between the original and the repeat SPK transplantation.

Concomitant procedures—Four patients required significant lysis of adhesions at retransplant, and two required concomitant hernia repair.

Perioperative complications

There were two episodes of graft loss from technical complications: one renal artery thrombosis and one pancreatic portal venous thrombosis. One patient had a leak from an enterotomy at the site of the initial transplant pancreatectomy (performed at the time of repeat SPK transplantation). Two patients were explored for hematoma evacuation. The average intraoperative blood transfusion was 2.2 units (range: 0–5), and the mean number of subsequent blood transfusions in the first 48 h post-operatively was 1.2 units (range: 0–5). Two patients experienced delayed graft function requiring hemodialysis during the transplant hospitalization. Median length of stay was 11 d (range: 6–32).

Rejection during the first year

Episodes of rejection occurred in 33% of pancreatic allografts and 44% of renal allografts within the first year. Episodes of simultaneous rejection of both organs occurred in two patients during the first year, and the remaining episodes involved isolated pancreatic (n = 1) or renal rejection (n = 2). Treatment for rejection in the first year after transplant was diverse, depending on clinician practice and pathologic severity. Treatments included steroids (n = 4), rituximab (n = 3), plasmapheresis (n = 2), and intravenous immune globulin (n = 2). Two patients had multiple rejection episodes in the first year, and the second episode was limited to the renal allograft in both instances. One of these two developed a third episode of renal allograft rejection during the first year.

Patient and graft survival

Overall pancreatic allograft survival was 78% at one yr and 67% at two yr. Causes of pancreatic allograft failure included graft thrombosis (n = 1) and unknown causes (n = 2). In our recent review of 1000 SPK transplants, one-yr pancreatic allograft survival was 88% and two-yr survival was 84% (13). Overall renal allograft survival in repeat SPK recipients was 89% at one yr and 78% at two yr as compared to 91% and 89% in primary SPK recipients. Causes of renal allograft loss included chronic rejection (n = 3), acute rejection (n = 1), and renal artery thrombosis (n = 1). Patient survival at one and three yr was 100%.

Retransplantation after repeat SPK

Three patients underwent a third renal transplant following the repeat SPK transplantation, and one patient received a third pancreatic allograft.

Discussion

Retransplantation in SPK recipients is becoming increasingly frequent at major centers. The technical demands of repeat SPK transplantation are not insignificant, and re-operating on previously dissected iliac vessels requires significant pre-operative planning and intraoperative flexibility.

We have enjoyed success with portal drainage to the distal IVC, and in all re-operative cases, the distal IVC was used for portal drainage. Typically, the IVC anastomosis was performed slightly more cephalad to create a fresh venous anastomosis. Although we have not found it necessary, should the distal IVC or right-sided iliac venous system be completely inaccessible, portal venous drainage can be employed or the pancreas could be positioned in a head-down position and drained on the left iliac system. The length of the donor iliac Y-graft provides significant flexibility for choosing an appropriate, disease-free, portion of iliac artery for implantation. In cases where the right iliac artery is severely diseased or otherwise unusable, inflow through the left iliac artery can be considered as well. Although a chronically rejected pancreatic allograft is often small and fibrotic, transplant pancreatectomy was performed in the vast majority of cases. In instances of patients who had undergone either enteric conversion or primary enteric drainage, transplant pancreatectomy generally was required for adequate mobilization of the bowel to access the iliac vessels, as well as for additional space in which to position the allograft. In asymptomatic patients with failed bladder-drained pancreatic allografts, the small, shrunken pancreas was left in place. In these two instances, the pancreas did not impede mobilization of the bowel or access to the distal IVC and proximal iliac artery.

Although our preferred technique for transplant nephrectomy involves intracapsular dissection with subsequent control of the renal hilum from within the capsule, this was not the technique utilized when performing transplant nephrectomy at the time of repeat transplantation. In this case, an extracapsular technique was used, with individual isolation of the renal artery and vein. This preserves the vessels as an option to use for implantation of the repeat renal transplant. In the left iliac system in particular, the vein is more difficult to isolate for anastomosis on repeat exploration, and transplant nephrectomy maximizes the options available for implantation. As in pancreas retransplantation, a significant amount of flexibility is required to determine the optimal site for implantation.

Despite these challenges, there were only two technical graft losses in the perioperative period, one from a renal artery thrombosis and one from a pancreatic venous thrombosis. This rate of overall technical graft loss (11%) is remarkably higher than that noted in primary SPK transplantation. In our recent series of 1000 consecutive primary SPK transplants, the overall rate of technical graft loss in the perioperative period was 3%. Six renal allografts and 19 pancreatic allografts were lost because of thrombosis. An additional five pancreatic allografts were lost because of anastomotic leakage. The increased rate of technical graft loss in repeat SPK transplantationis, perhaps, not unexpected given the degree of difficulty associated with the operation.

Delayed graft function, defined as a dialysis requirement in the first week following transplant, occurred in two patients. Each patient had a number of standard risk factors for delayed graft function (DGF). The first patient was sensitized (67%), received DCD donor organs with 20 h of cold ischemic time, and had a difficult intraoperative dissection

requiring 5 units of packed red blood cells. The second patient who experienced DGF was also sensitized (PRA 10%) and received standard criteria donor organs with a cold ischemic time of 20 h. The mean cold ischemic time for the cohort was 14.5 h. Unexpectedly, no patient who underwent simultaneous nephrectomy and/or pancreatectomy experienced DGF.

In addition to the clearly increased technical complexities of retransplantation, the immunologic barriers presented in retransplantation offer additional challenges. Although the majority of patients had a low or absent PRA at the time of retransplantation, all recipients were perceived to have a high antigenic exposure and were treated as high immunologic risk recipients. All patients received induction with depleting antibody therapy (alemtuzumab, n = 3; ATGAM n = 2; thymoglobulin, n = 4). The particular induction therapy was chosen by the individual surgeon based on personal practice or the departmental practice at the time of repeat SPK transplantation. All patients were placed on a maintenance regimen of steroids, tacrolimus, and mycophenolate, which is currently our standard regimen for primary SPK recipients as well.

The two patients who underwent renal transplantation between the original SPK and the repeat SPK underwent therapy aimed against the antibody response in addition to the standard induction and maintenance regimen. One of these patients (PRA 0%) received rituximab, and the other (PRA 17%) underwent plasmapheresis. An additional patient, whose original pancreatic allograft was lost secondary to a particularly vigorous rejection episode occurring shortly after the first year post-transplant, was treated with the standard therapy as well as rituximab and plasmapheresis/IVIG despite a PRA of 0%. C4d immunostaining and DSA testing were not widely available in our center during the study period but will likely provide illuminating data in future studies.

As might be expected, the patients who underwent renal transplant between the original and repeat SPK transplants each experienced multiple episodes of rejection in the first year. Otherwise, broad conclusions regarding the incidence and severity of rejection are difficult to determine given the low frequency of repeat SPK transplantation. For instance, three patients experienced no episodes of rejection in the study period, including the most sensitized patients. However, of the remaining six patients, four experienced acute cellular rejection in the first month post-transplant and one additional patient experienced rejection in the first year. The final patient did not experience rejection until nearly four yr post-transplant. The first rejection episode commonly affects both organs simultaneously (n = 3) but nearly always affects the renal allograft (n = 5). This may be reflective of the relative ease in diagnosis of rejection in the renal allograft. Although steroids were used to reverse rejection in nearly all cases (five of six), therapy against antibody-mediated rejection was also used in three patients (plasmapheresis in two cases, immunoglobulin in 3, and rituximab in 3). Thymoglobulin was used in one other instance. Four patients had a second episode of rejection, and the renal allograft was involved in all instances. The pancreatic allograft was affected in the second episode of rejection in only one patient.

Within the confines of this small study, there appears to be no correlation between graftectomy (either before or during retransplantation) and subsequent episodes of rejection. Likewise, DGF following retransplantation did not seem related to subsequent rejection. The frequency of rejection occurring in the first year following retransplantation was comparable with that seen in primary SPK transplantation at our center (pancreas allograft: 23%, renal allograft: 45%) (13). Nonetheless, as the majority of graft losses were immunologic in nature, this prior allograft exposure can be presumed to have an impact on graft survival. As nearly all of the failures of the original pancreas and renal allografts were immunologic, it is difficult to determine the effect this has on subsequent graft survival after repeat SPK transplantation.

There was no effort at either HLA matching the recipient or avoiding the HLA subtype of the original SPK donor, and the mean HLA mismatch was 4 (range: 3–6). There was no evident correlation between HLA mismatch and subsequent rejection episodes.

The immunologic effect of concomitant nephrectomy and/or pancreatectomy at the time of retransplantation is less clear. The small number of patients and the diversity of surgical interventions make any conclusions suspect. Nonetheless, it can be seen that the PRA at the time of repeat SPK was typically zero (n = 6). The most common course was to perform nephrectomy (n = 5) and pancreatectomy (n = 4) at the time of repeat transplantation. Interestingly, the patient with maximal PRA (67%) underwent original SPK transplantation earliest in our series and did not undergo graftectomy of either organ. However, the remaining two (moderately) sensitized patients with PRAs of 10% and 17% underwent nephrectomy 2–3 yr prior to repeat SPK transplantation, and the latter patient received an additional renal transplant, which subsequently failed prior to repeat SPK transplantation. The other comparable patient in the series had a PRA of 0% at the time of repeat SPK transplantation despite an intervening renal transplant. Although the PRA was determined at the time of retransplantation, it was not subsequently followed and therefore the effect of transplant graftectomy of prior organs at the time of retransplantation would be difficult to determine and the effect challenging to separate from that of retransplantation. Blood transfusions during the first operation did not have a noticeable effect on the PRA at retransplantation.

The absence of perioperative deaths suggests that repeat SPK transplantation is a reasonable option in well-selected patients. Furthermore, there is a component of recipient self-selection as a number of SPK recipients with failed allografts expired because of other causes prior to undergoing evaluation for retransplantation. This group is certainly highly selected, and our standard pre-operative assessment includes screening for coronary disease with coronary catheterization. Patients deemed unsuitable for SPK retransplantation are offered renal retransplant alone or are denied transplantation. Thirtytwo prior SPK recipients with failure of both allografts underwent renal retransplantation alone. When these recipients were compared with recipients undergoing repeat SPK transplantation, groups were similar in regard to gender, race, BMI, peak PRA, and time since original SPK. SPK recipients who underwent renal retransplant alone tended to be slightly older (34 ± 4.6 vs. 31 ± 3.6, $p = 0.08$). There were no significant differences between groups in regard to coronary or vascular disease rates prior to retransplantation.

Survival of both repeat allografts is acceptable despite the increased technical and immunologic demands of retransplantation. Nonetheless, significant center experience with SPK transplantation as well as intraoperative technical flexibility may be required to achieve such outcomes.

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Table 1

Recipient demographics

	Mean
Age (yr)	39.0
BMI (kg/m^2)	24.2
Time since SPK (yr)	7.8
Peak PRA (%)	10.4
Gender	n (%)
Male	2 (22)
Female	7 (78)
Race	
Caucasian	9 (100)
Pre-transplant dialysis	
None	3 (33)
Hemodialysis	5 (56)
Peritoneal dialysis	1 (11)

Table 2

Donor demographics

	Mean
Donor age	28.1
Gender	n (%)
Male	5 (56)
Female	4 (44)
Race	
Caucasian	9 (100)
Donor type	
Donation after brain death	7 (78)
Donation after cardiac death	2 (22)

Table 3

Summary of surgical interventions

Patient	Drainage of original SPK	Enteric conversion (indication)	Transplant nephrectomy	Transplant pancreatectomy	Pancreas vascular anastomosis	Pancreas drainage	Kidney vascular anastomosis	Technical complications
1	Bladder	No	At SPK	Prior to SPK	IVC, RCIA	Jejunum	Tx vein, LCIA	
2	Bladder	No	Prior to SPK	No	IVC, RCIA	Terminal ileum	LEIV, LEIA	
3	Bladder	Yes (UTIs)	No	No	IVC, RCIA	Terminal ileum	L gonadal, LCIA	
4	Bladder	Yes (UTIs)	At SPK	Prior to SPK	IVC, RCIA	Jejunum	Tx vein, Tx artery	Pancreas venous thrombosis
5	Bladder	Yes (UTIs)	At SPK	At SPK	IVC, RCIA	Jejunum	LCIV, LCIA	Perforated ileum at tx pancreatectomy site
6	Bladder	Yes (UTIs)	Prior to SPK	At SPK	IVC, REIA	Jejunum	LCIV, LCIA	Hematoma
7	Enteric	NA	At SPK	At SPK	IVC, RCIA	Roux	LEIV, LEIA	Renal artery thrombosis
8	Enteric	NA	At SPK	At SPK	IVC, RCIA	Roux	Tx vein, LCIA	
9	Enteric	NA	Prior to SPK	Prior to SPK	IVC, RCIA	Jejunum	LCIV, LCIA	Hematoma

NA, not applicable; IVC, inferior vena cava; CIA, common iliac artery; CIV, common iliac vein, EIA, external iliac artery; EIV, external iliac vein; Tx artery, primary renal allograft artery; Tx vein, primary renal allograft vein; UTI, urinary tract infection.