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## Original Article

# Risk factors for surgical site infection after kidney and pancreas transplantation

Yoichiro Natori MD[1,2], Salma Albahrani MD[1,2,3], Mohammed Alabdulla MD[1,2,4], James Vu BSc[1], Edwin Chow[1], Shahid Husain MD[1,2], Deepali Kumar MD[1,2], Atul Humar MD[1,2], Jeffrey Schiff MD[1], Mark Cattral MD[1,5] and Coleman Rotstein MD[1,2]

[1]Multi-Organ Transplant Program, University Health Network, University of Toronto, Toronto, Ontario, Canada, [2]Division of Infectious Diseases, University Health Network, University of Toronto, Toronto, Ontario, Canada, [3]Division of Infectious Diseases, King Fahad Military Complex, Dahran, Saudi Arabia, [4]Division of Infectious Diseases, King Saud Medical City, Saudi Arabia and [5]Department of Surgery, University Health Network, University of Toronto, Toronto, Ontario, Canada

**Abstract**

Objective: To evaluate the incidence of surgical site infection (SSI) in a cohort of pancreas transplant recipients and assess predisposing risk factors for SSI

Design: Retrospective cohort study

Setting: Single transplant center in Canada

Patients: Patients who underwent any simultaneous pancreas and kidney (SPK) or pancreas after kidney (PAK) transplant procedures between January 2000 and December 2015

Methods: In this retrospective cohort evaluation of SPK or PAK recipients, we assessed the incidence of SSI and risk factors associated with superficial, deep, and organ/space SSI. Multivariate logistic regression was used to identify independent risk factors for SSI in SPK and PAK recipients.

Results: In total, 445 adult transplant recipients were enrolled. The median age of these patients was 51 years (range, 19–71 years), and 64.9% were men. SSIs were documented in 108 patients (24.3%). Organ/space SSIs predominated (59 patients, 54.6%), followed by superficial SSIs (47 patients, 43.5%) and deep SSIs (3 patients, 2.8%). Factors predictive of SSIs in the multivariate analysis were cold pancreas ischemic time (odds ratio [OR], 1.002; $P = .019$) and SPK transplant (compared to PAK transplant recipients; OR, 2.38; $P = .038$). Patients with SSIs developed graft loss more frequently (OR, 16.99; $P < .001$).

Conclusions: Organ/space SSIs remain a serious and common complication after SPK and PAK. Prolonged cold ischemic time and SPK transplant were the risk factors predictive of SSIs. Appropriate perioperative prophylaxis in high-risk patients targeting the potential pathogens producing SSIs in kidney and/or pancreas transplant recipients and a reduction in cold ischemia may prove beneficial in reducing these SSIs.

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Kidney and pancreas transplantation is the preferred treatment modality to ameliorate renal failure and other comorbidities associated with type 1 diabetes mellitus. Approximately 75% of pancreas transplants are performed simultaneously with kidney transplants from the same deceased donor.[1] Pancreas transplantation has evolved over the past 20 years, with refinements in surgical technique, better organ preservation, and more potent immunosuppressive therapies, which have improved graft survival rates.[2] In particular, the evolution of immunosuppressive therapy has reduced rates of acute graft rejection but has enhanced the propensity to develop posttransplant infections.[3] Notably, simultaneous pancreas and kidney transplantation (SPK) has a higher rate of surgical complications than other solid organ transplants.[4] Postoperatively, surgical site infections (SSIs) and urinary tract infections are the most prevalent infections after kidney and pancreas transplantation.[5–7] An SSI often necessitates a repeat laparotomy to drain the intra-abdominal infection.[8]

Notably, few reports have assessed the infectious complications after SPK or pancreas transplantation after kidney (PAK) transplantation, even though these infectious complications may affect 7%–50% of patients who undergo this procedure.[4,9] Gram-negative pathogens predominate among the causative organisms, and 42% of these isolates prove to be extended-spectrum β-lactamase–producing organisms.[9] Vancomycin-resistant enterococci have recently emerged as important pathogens in intra-abdominal transplants.[10] The aim of this study was to assess the incidence of SSIs, causative pathogens, and risk factors for these SSIs in SPK and PAK recipients within the first 3 months after transplant.

**Author for correspondence:** Coleman Rotstein, MD, University Health Network, Toronto General Hospital, 200 Elizabeth Street, PMB 11-139, Toronto, Ontario M5G 2C4. E-mail: Coleman.Rotstein@uhn.ca

PREVIOUS PRESENTATION. These results were presented in part at ID Week 2017 in San Diego, California, on October 4–8, 2017 (abstract #2218).

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## Patients and methods

This retrospective cohort study included adult patients who underwent SPK or PAK at the Toronto General Hospital, University Heath Network, Toronto, Canada. The institutional research ethics board approved the study protocol. Our inclusion criteria were (1) any pancreas transplant (PT) recipient ≥18 years of age at the time of transplantation for the period 2000–2015 and (2) patients who survived >72 hours. We excluded patients with multivisceral intra-abdominal transplants including concurrent liver or bowel transplants. Electronic data were available only for those patients transplanted after 2000, although pancreas transplants themselves have been performed at our institute since 1995.

Perioperative prophylaxis for PT patients consisted of cefazolin 1 g every 8 hours intravenously for 3 days. In penicillin allergic patients, vancomycin intravenously for 3 days was substituted for cefazolin. The antimicrobial prophylaxis did not change over the study period. The surgical techniques for SPK and PAK were performed as previously described.[11] Also, maintenance immunosuppression was initiated immediately after induction therapy was administered. After hospital discharge, all transplant recipients were followed weekly for the first 4 weeks and then weekly or biweekly for the next 8 weeks as outpatients, depending on their condition and complications (ie, 3 months).

### Variables

We extracted the following information from patients' electronic medical records: recipient age, recipient gender, duration of surgical procedure, total ischemic time of the donated organ (kidney and/or pancreas), perioperative antimicrobial prophylaxis regimen, induction regimen, postoperative immunosuppressive regimen, operative surgeon, presence of SSI within 90 days of the surgical procedure,[12] causative pathogens implicated in the SSIs and their susceptibilities (if available), antimicrobial therapy within 3 days prior to the transplant and for 30 days post-transplant, and other documented infections. The cultures of the potential SSIs were obtained at the time of diagnosis or when diagnostic procedures were performed (ie, interventional radiology procedures or intraoperative cultures at the time of drainage of the organ/space SSI).

### Definitions

The SSIs were classified according to the Centers for Disease Control and Prevention (CDC) classification system.[12] These infections are divided into the following categories: (1) superficial incisional SSI involving only the skin or subcutaneous tissue of the incision; (2) deep incisional SSI involving the fascia and/or muscular layers in the primary incision (deep incision primary) in a patient who had an operation involving 1 or more incisions and an SSI identified in the secondary incision (deep incision secondary) in an operation with more than 1 incision; and (3) organ/space SSI involving any part of the body opened or manipulated during the procedure excluding the skin incision, fascia, or muscle layers.[12] Other anatomic sites were considered to be infected if there were clinical signs of infection (eg, purulence or fever). In addition, SSIs were determined based on clinical signs of purulence plus 1 of the following conditions: redness, edema, pain, and confirmation by the surgeon. Cultures were obtained from superficial, deep tissue, and organ/space sites to document SSIs if purulent material for culture was available. For superficial SSIs, the wound was documented as infected and was then cultured. For a deep incisional SSI, once documented, the wound was cultured. Finally, for organ/space SSIs, cultures of infected areas were obtained by interventional radiology-directed aspirate or at the time of surgical drainage in the operating room. All microbiological data were retrieved from the patients' electronic medical records with accompanying identification and susceptibilities of the microorganisms whenever possible.

### Statistical analysis

Categorical variables comparing patients who developed SSIs to those who did not were analyzed using $\chi^2$ tests, with the Pearson and Fisher exact tests when appropriate. For continuous variables, we conducted Mann-Whitney *U* tests. We analyzed risk factors for SSI in all SPK and PAK recipients by multivariate logistic regression analysis with stepwise backward elimination for those patients who developed SSIs compared to those who did not. For the multivariate model, we included all factors, with $P < 0.2$ in the univariate analysis. We also checked for interaction of the variables, and if there was significant interaction between variables, we chose only 1 variable to put into model. $P < .05$ was considered statistically significant. All statistical testing was performed using SPSS version 22 software (2013; IBM, Chicago, IL).

## Results

A total of 445 adult patients who underwent SPK or PAK at our center between January 2000 and December 2015 were enrolled: 305 SPK transplants in 295 patients (5 patients underwent 2 pancreas-kidney transplants, but only the first SPK was analyzed) and 150 PAK transplants. Consecutive patients who underwent pancreas transplantation during this period were included. No patients died within 72 hours of the transplant procedure. In addition, we recorded no deaths within 3 months after the transplants. The demographic characteristics of the enrolled patients are shown in Table 1. The median age of the recipients was 51 years (range, 19–71 years), and most patients were men (64.9%). The induction immunosuppressive therapy employed with the study patients are documented in Table 1. Virtually all patients received tacrolimus, mycophenolate, and a steroid as maintenance immunosuppressive therapy. All pancreas grafts were drained enterically.

**Table 1.** Demographic Data for All Pancreas Transplants

| Characteristic | Patients (N = 445) |
|---|---|
| Age, median y (range) | 51 (19–71) |
| Male/female, no. (%) | 289 (64.9)/156 (35.1) |
| SPK/PAK, no. (%)[a] | 305 (66.9)/150 (33.0) |
| Antithymocyte globulin induction, no. (%) | 361 (81.1) |
| Basiliximab induction, no. (%) | 66 (14.8) |
| Length of hospital stay, d (range) | 10 (3–96) |
| Cold pancreas ischemic time, min (range) | 482 (104–1,146) |

Note. SPK/PAK, simultaneous pancreas and kidney/pancreas after kidney transplant.
[a]305 SPK transplants in 295 patients (10 patients received 2 transplants); only 295 SPKs were analyzed. 150 PAK transplant procedures.

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Overall, 108 of 445 study participants had SSIs (24.3%). The annual incidence of SSIs is reflected in Figure 1. No statistical significant differences in infection rates were detected among the years of study, although the annual rate was based on relatively small numbers of infections. Moreover, 115 SSIs occurred among the 108 infected patients: 29 with superficial SSIs, 3 with deep incisional SSIs, and 83 with organ/space SSIs. Of 108 patients, 5 patients had 2 different organ/space infections, and 3 patients had concurrent superficial and organ/space infections. Furthermore, 21 SSIs were included using non-CDC criteria, and all were classified as superficial SSIs. We identified 45 mixed infections in 43 patients. The overall time from the transplant to the diagnosis of SSI was a median of 16 days (range, 1–75 days). For superficial infection, the time from transplantation to the diagnosis of the SSI was a median of 14 days (range, 2–50 days), 20 days (range, 14–25 days), and 18 days (range, 1–75 days). For deep infection, the time from transplantation to the diagnosis of the SSI was a median of 20 days (range, 14–25 days), and for organ/space infections, the elapsed time was a median of 18 days (range, 1–75 days).

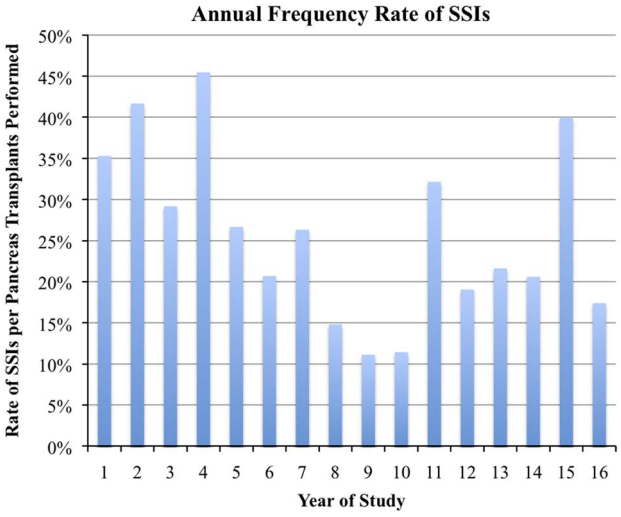


**Fig. 1.** Annual rate of SSIs per pancreas transplants performed.

### *Risk factors for SSIs in pancreas transplant patients*

First, we analyzed the risk factors for developing SSIs in all 445 patients who received a pancreas transplant. Compared with SPK patients, PAK patients were less likely to develop an SSI (odds ratio [OR], 0.45; 95% confidence interval [CI], 0.27–0.75; $P = .002$) as detected in the univariate analysis (Table 2). Older

**Table 2.** Risk Factors for All Pancreas Transplants

| Characteristic | No SSI (n = 337) | SSI (n = 108) | Univariate OR (CI) | *P* Value | Multivariate OR (CI) | *P* Value |
|---|---|---|---|---|---|---|
| **Recipient factor** | | | | | | |
| Age, median y (range) | 51 (19–71) | 51 (28–68) | 1.01 (0.99–1.04) | .31 | | |
| Female gender, no. (%) | 114 (33.8) | 42 (38.9) | 1.25 (0.80–1.95) | .34 | | |
| PAK, no. (%) | 127 (37.7) | 23 (21.3) | 0.45 (0.27–0.75) | .002 | 0.53 (0.29–0.96) | .038 |
| ATG§ induction, no. (%) | 283 (84.0) | 78 (72.2) | 0.50 (0.30–0.83) | .007 | … | … |
| Basilliximab induction, no. (%) | 42 (12.5) | 24 (22.2) | 2.01 (1.15–3.50) | .013 | … | … |
| Cold ischemic time, median min (range) | 470 (104–1,146) | 495 (229–1,025) | 1.002 (1.000–1.003) | .038 | 1.002 (1.000–1.004) | .019 |
| **Surgeon, no. (%)** | | | | | | |
| Surgeon 1 | 143 (42.4) | 36 (33.3) | | | | |
| Surgeon 2 | 48 (14.2) | 20 (18.5) | | | | |
| Surgeon 3 | 41 (12.2) | 18 (16.7) | | | | |
| Surgeon 4 | 23 (6.8) | 9 (8.3) | | | | |
| Surgeon 5 | 82 (24.3) | 25 (23.1) | | .38 | | |
| **Donor factor** | | | | | | |
| Donor gender (female), no. (%) | 117 (34.7) | 33 (30.6) | 0.83 (0.52–1.32) | .43 | | |
| Donor age, median y (range) | 25 (2–52) | 29.5 (2–52) | 1.027 (1.005–1.049) | .016 | 1.024 (0.99–1.05) | .060 |
| **Outcome** | | | | | | |
| Length of hospital stay, median d (range) | 10 (5–48) | 12 (3–96) | 1.11 (1.07–1.16) | <.001 | | |
| ICU stay, median d (range) | 3 (0–19) | 4 (0–32) | 1.09 (0.99–1.19) | .21 | | |
| Graft loss within 3 mo, no. (%) | 5 (1.5) | 22 (20.4) | 16.99 (6.25–46.15) | <.001 | | |

Note. SSI, surgical site infection; OR, odds ratio; CI, confidence interval; PAK, pancreas transplant after kidney transplant; ATG, antithymocyte globulin.

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**Table 3.** Risk Factors for Simultaneous Pancreas and Kidney Transplants

| Characteristic | No SSI (n = 210) | SSI (n = 85) | Univariate OR (CI) | *P* Value | Multivariate OR (CI) | *P* Value |
|---|---|---|---|---|---|---|
| **Recipient factor** | | | | | | |
| Age, median y (range) | 51 (19–71) | 51 (28–68) | 1.01 (0.98–1.05) | .35 | | |
| Female gender, no. (%) | 65 (31.0) | 33 (38.8) | 1.42 (0.84–2.39) | .19 | … | … |
| ATG induction, no. (%) | 161 (76.7) | 57 (67.1) | 0.62 (0.36–1.08) | .089 | … | … |
| Basilliximab induction, no. (%) | 37 (17.6) | 22 (25.9) | 1.63 (0.90–2.98) | .11 | … | … |
| Cold ischemic time (P), median min (range) | 451 (104–1,146) | 500 (229–1,019) | 1.002 (1.000–1.004) | .016 | 1.002 (1.000–1.004) | .014 |
| Cold ischemic time (K), median min (range) | 561 (175–1,226) | 660 (322–1,138) | 1.002 (1.000–1.004) | .023 | … | … |
| Peritoneal dialysis | 56 (26.7) | 21 (24.7) | 0.90 (0.51–1.61) | .73 | | |
| **Surgeon, no. (%)** | | | | | | |
| Surgeon 1 | 100 (47.6) | 29 (34.1) | | | | |
| Surgeon 2 | 23 (11.0) | 15 (17.6) | | | | |
| Surgeon 3 | 26 (12.4) | 14 (16.5) | | | | |
| Surgeon 4 | 14 (6.7) | 8 (9.4) | | | | |
| Surgeon 5 | 47 (22.4) | 19 (22.4) | | .20 | | |
| **Donor factor** | | | | | | |
| Donor gender (female), no. (%) | 71 (33.8) | 25 (29.4) | 0.82 (0.47–1.41) | .47 | | |
| Donor age, median y (range) | 27 (2–51) | 31 (2–52) | 1.01 (0.99–1.04) | .28 | | |
| **Outcome** | | | | | | |
| LOS, median d (range) | 10 (5–48) | 13 (3–96) | 1.10 (1.06–1.15) | <.001 | | |
| ICU stay, median d (range) | 4 (0–19) | 4 (0–32) | 1.05 (0.97–1.13) | 0.91 | | |
| Graft loss within 3 mo, no. (%) | 5 (2.4) | 16 (18.8) | 9.51 (3.36–26.91) | <.001 | | |

Note. SSI, surgical site infection; OR, odds ratio; CI, confidence interval; PAK, pancreas transplant after kidney transplant; ATG, antithymocyte globulin; LOS, length of hospital stay.

donor age was also risk factor with univariate analysis (OR, 1.027; 95% CI, 1.005–1.049; $P = .016$). Also, patients treated with antithymothyte globulin (ATG) induction were less likely to develop an SSI (OR, 0.50; 95% CI, 0.30–0.83; $P = .007$). Basiliximab use was a risk factor for developing an SSI (OR, 2.01; 95% CI, 1.15–3.50; $P = .013$) as was longer pancreas ischemic time (OR, 1.002; 95% CI, 1.000–1.003; $P = .038$). The surgeon who performed the operation had no bearing on the risk of developing an SSI. We did not include basiliximab use in the multivariate model because there was strong negative correlation between basilliximab and ATG ($r = -0.87$; $P < .001$). In the multivariate analysis, PAK patients were less likely to develop an SSI (OR, 0.53; 95% CI, 0.29–0.96; $P = .038$), whereas longer cold ischemic time was an independent risk factor related to the development of an SSI (OR, 1.002; 95% CI, 1.000–1.004; $P = .091$). Also, older donor age was associated with SSI in this cohort (OR, 1.024; 95% CI, 0.99–1.05; $P = .060$) (Table 2).

### *Risk factors for SSIs in simultaneous pancreas and kidney transplant patients*

We analyzed the risk factors developing an SSI in 295 SPK patients. Overall, 85 patients developed SSIs. In the univariate analysis, longer ischemic time was a risk factor for both pancreas transplantation (OR, 1.002; 95% CI, 1.000–1.004; $P = .016$) and kidney transplantation (OR, 1.002; 95% CI, 1.000–1.004; $P = .023$). Peritoneal dialysis prior to transplantation, pertinent only to SPK transplants, did not emerge as a significant factor associated with SSI in 21 of 85 patients (24.7%) who received peritoneal dialysis who developed SSIs compared to 56 of 210 patients (26.7%) who did not receive peritoneal dialysis ($P = .73$) (Table 3). We checked the correlation between the variables, and again, we detected a strong correlation between cold pancreas and kidney ischemia time ($r = 0.93$; $P < .001$). As a result, we analyzed gender, ATG use, and pancreas ischemia time in the multivariate model. In the multivariate model, only pancreas ischemic time was a statistically significant predictor for SSI (OR, 1.002; 95% CI, 1.000–1.004; $P = .014$).

### *Risk factors for SSIs in pancreas after kidney transplant patients*

We subsequently analyzed risk factors for SSI in 150 PT patients (Table 4). Only 23 of 150 patients (15.3%) developed SSIs, and due to small sample size, we were only able to perform a univariate analysis. No statistically significant variables predicted SSIs in this cohort; however, older donor age was associated with SSI in this cohort.

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### *Microorganisms producing SSIs*

Causative organisms are shown in Figure 2. Most SSIs were produced by polymicrobial infection (45 polymicrobial infections in 43 patients). In total, commensal flora (comprising a mixture of gram-positive as well as gram-negative organisms without a predominant organism type) followed by *Enterococcus* spp (not including vancomycin-resistant Enterococci), and *Candida* spp were most common. Notably, 85% of isolated organisms were resistant to cefazolin, which had been used as perioperative prophylaxis.

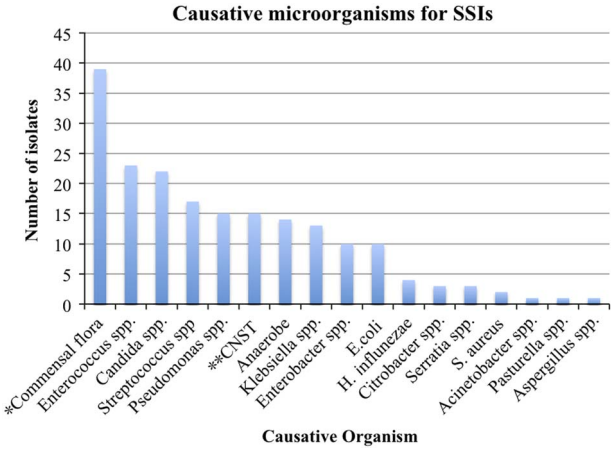


**Fig. 2.** Microorganisms causing SSIs in all pancreas transplants.

### *Outcome variables*

Compared with the PT group without SSI, the median length of hospital stay was significantly longer in the PT group with SSIs: 10 days (range, 5–48 days) vs 12 days (range, 3–96 days) ($P < .001$). Compared with the SPK group without SSI, the median length of hospital stay was significantly longer in the SPK group with SSIs: 10 days (range, 5–48 days) vs 13 days (range, 3–96 days) ($P < .001$). Compared with the PAK group without SSI, the median length of stay was not significantly longer in the PAK group with SSIs: 9 days (range, 5–21 days) vs 9 days (7–39 days) ($P = .019$). Similarly, versus transplant patients without SSI, graft loss at 3 months was more likely in patients with SSIs in the PT group (5 of 337 [1.5%] vs 22 of 108 [20.4%]; $P < .001$); in the SPK group (5 of 210 [2.4%] vs 16 of 85 [18.8%]; $P < .001$); and in the PAK group (0 of 127(0%) vs 6 of 23 [26.1%]; $P < .001$).

## Discussion

We conducted a retrospective cohort study to identify the incidence and risk factors for SSI in PT recipients at a single center.

**Table 4.** Risk Factors for Pancreas Transplantation After Kidney Transplantation

| Characteristic | No SSI (n = 127) | SSI (n = 23) | Univariate OR | *P* Value |
|---|---|---|---|---|
| **Recipient factor** | | | | |
| Age, median y (range) | 51 (34–68) | 50 (32–66) | 1.005 (0.95–1.06) | .78 |
| Gender (female), no. (%) | 49 (38.6) | 9 (39.1) | 1.02 (0.41–2.54) | .99 |
| ATG induction, no. (%) | 122 (96.1) | 21 (91.3) | 0.43 (0.08–2.37) | .29 |
| Basilliximab induction, no. (%) | 5 (3.9) | 2 (8.7) | 2.32 (0.42–12.77) | .29 |
| Cold ischemic time, median min (range) | 505 (251–969) | 494 (298–1,025) | 1.001 (0.998–1.005) | .69 |
| **Surgeon** | | | | |
| Surgeon 1 | 43 (33.9) | 7 (30.4) | | |
| Surgeon 2 | 25 (19.7) | 5 (21.7) | | |
| Surgeon 3 | 15 (11.8) | 4 (17.4) | | |
| Surgeon 4 | 9 (7.1) | 1 (4.3) | | |
| Surgeon 5 | 35 (27.6) | 6 (26.1) | | .94 |
| **Donor factor** | | | | |
| Donor gender (female), no. (%) | 46 (36.2) | 8 (34.8) | 0.94 (0.37–2.38) | .99 |
| Donor age, median y (range) | 20.5 (10–52) | 23.5 (15–50) | 1.04 (1.00–1.09) | .067 |
| **Outcome** | | | | |
| LOS, median d (range) | 9 (5–21) | 9 (7–39) | 1.13 (0.99–1.25) | .109 |
| ICU stay, median d (range) | 3 (0–10) | 4 (0–11) | 1.37 (1.10–1.70) | .019 |
| Graft loss within 3 mo, no. (%) | 0 (0) | 6 (26.1) | … | <.001 |

Note. SSI, surgical site infection; OR, odds ratio; CI, confidence interval; PAK, pancreas transplant after kidney transplant; ATG, antithymocyte globulin; LOS, length of hospital stay.

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Overall, 108 of the 445 patients in our pancreas transplant cohort (24.3%) had SSIs. Also, organ/space SSIs predominated among SSIs and were mainly caused by *Enterococcus* spp and *Candida* spp, which were not covered by the current prophylactic antimicrobial regimen. We found that SPK transplantation and longer pancreas ischemic time were significant predictors for SSIs. Finally, as might be expected, SSI was associated with longer hospital stays and poorer graft outcomes.

Several reports have addressed SSI among PT recipients. Previous studies showed the incidence of SSI in this patient population to be between 9% and 45%.[5,6,13–19] These data are comparable to our incidence of SSI (24.3%). Our study also showed that SSIs produced longer lengths of hospital stays. However, to the best of our knowledge, no studies have addressed the cost produced by this extended length of stay as a result of an SSI in recipients of kidney and/or pancreas transplants.

Risk factors associated with SSI in PT in previous studies have been delineated: reoperation, prolonged operative time, prolonged ischemic time (>4 hours), enteric drainage (rather than bladder drainage), posttransplant fistula, hand-sewn anastomoses (rather than stapled anastomoses), blood transfusions; donor age >55 years; acute tubular necrosis in the allograft, and graft rejection.[5,9,20,21]

As demonstrated in previous reports, surgical technique other than cold ischemic time may also have played a more significant role predisposing PT recipients to SSI in our study. Since 1997, enteric drainage has been used in all recipients. We moved away from portal venous to systemic venous drainage in 2001. In our institution, hand-sewn enteric anastomosis drainage of the transplanted pancreas transitioned to bladder drainage part way through our study period. Nevertheless, this alteration in surgical technique did not emerge as a significant risk factor for SSI in PT recipients. Also, peritoneal dialysis that could have induced peritoneal adhesions was not a factor in SPK transplants, as has been reported in a previous study.[22] Moreover, we explored whether individual surgeons could have played a role in predisposing recipients to infection, but they did not.

This study has several limitations. First, it was conducted retrospectively. As with all retrospective data analyses, data collection was hampered occasionally by missing data. Also, it may be difficult to differentiate between colonization and true infection. Cultures of wound discharge were performed once the diagnosis of SSI was made based on wound purulence. Not all wounds were cultured when the diagnosis was made though. No other criteria were used to differentiate infection from colonization. However, we believe that we captured all SSIs because the diagnosis of SSIs was made by treating physician and was categorized retrospectively by 2 transplant infectious disease specialists (Y.N. and C.R.). However, the diagnoses of SSIs might have been underestimated by the treating physician because an SSI diagnosis might be perceived to reflect badly on the surgeon performing the procedure. All medical records were reviewed carefully for all potential SSIs. Third, we collected data from 2000 to 2015. The transplant procedure techniques might have changed with experience over these 15 years, which may have impacted our estimate of SSI incidence. However, there was no overt significant change in the surgical technique, and thus, this did not appear to be the case. The study period had no bearing on the production of SSIs. In covering this time span and obtaining as much data as possible, we have conducted one of the largest studies to date regarding SSI in PT patients.

In conclusion, longer cold ischemic time was a statistically significant predictor of SSI. Improved surgical technique to reduce cold ischemic time should be undertaken to assess whether this will impact SSI development. The adequacy of the prophylactic regimen employed in our retrospective study as well as whether 3 days is the optimum duration of prophylaxis may be questioned. The propensity of patients to develop an SSI after PT may potentially be overcome with appropriate antimicrobial prophylaxis addressing the potential pathogens that cause SSI after PT.

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