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Original Article

Correlation of intraoperative donor duodenal-segment swab cultures with the subsequent occurrence of surgical site infections in kidney and pancreas transplant recipients

Mohammed Alabdulla MD[1], Sanaa Alrehily MD[1], Yoichiro Natori MD[2], Kelsey Van[1], Mark Cattral MD[3], Trevor Reichman MD[3] and Coleman Rotstein MD[1]

[1]Multi-Organ Transplant Program and Division of Infectious Diseases, Department of Medicine and University Health Network, University of Toronto, Toronto, Ontario, [2]Division of Infectious Diseases, Department of Medicine, University of Miami, Miami, Florida and [3]Multi-Organ Transplant Program, University Health Network and Department of Surgery, University of Toronto, Toronto, Ontario, Canada

Abstract

Background: Pancreas transplantation is employed for the treatment of type I diabetes mellitus. It is postulated that surgical site infection (SSI), particularly organ-space infections, after pancreas transplantation may arise from microbial contamination arising from the donor duodenal segment. Therefore, some centers have adopted the practice of culturing the donor duodenal segment and subsequently administering antimicrobial therapy to the recipient directed at the microorganisms isolated to prevent SSI.

Methods: In this retrospective cohort study, we evaluated the correlation between positive donor duodenal-segment cultures and SSIs in the recipients. Data were recorded and analyzed to assess the correlation of the organisms isolated in the donor duodenal cultures with those producing SSI in the recipients.

Results: We evaluated 379 consecutive pancreas transplant recipients from January 2000 to December 2015. Donor duodenal swab cultures were performed at the time of pancreas transplantation, and 206 (54.3%) were positive. SSIs occurred in 51 of the 206 recipients (24.8%) with positive duodenal-segment cultures and in 41 of 173 individuals (23.7%) with negative cultures ($P = .81$; $r = 0.00$). Notably, deep and organ-space SSIs were observed in 27 of 206 of the positive duodenal culture groups (13.1%) versus 29 of 173 of the negative duodenal culture groups (16.8%; $P = 0.31$; $r = -0.059$). No differences were detected in the pathogens producing SSIs between the group with a positive duodenal swab versus the group with a negative swab. Microorganisms producing SSIs matched those found in the positive donor duodenal cultures in only 15 patients (7.8%).

Conclusion: Although positive cultures from the donor duodenal segment prompted the administration of antimicrobial therapy in the recipient directed against the pathogen isolated, this practice did not reduce SSIs compared with those transplant recipients with culture-negative duodenal swabs. In addition, the organisms isolated from the donor duodenal segment were not predictive of subsequent SSI.

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Pancreas transplantation is a widely accepted treatment option to improve long–term survival for type I diabetic patients.[1] The success of pancreas transplantation has improved over time through advances in surgical technique, improved antirejection medications, better organ preservation, and the effective use of antibiotics to prevent and treat infectious complications.[2] Despite reductions of complications in these patients, surgical site infection (SSI) remains a significant cause of morbidity and mortality.[2,3] We previously reported an incidence of 24.3% SSIs in simultaneous pancreas and kidney transplants (SPK) as well as pancreas after kidney transplantation (PAK), with organ-space infections predominating.[4] Others have demonstrated that infections may complicate 7%–50% of these procedures.[5,6] Factors implicated in predisposing pancreas transplant recipients to SSI were cold pancreas ischemic time and simultaneous pancreas and kidney transplantation.[4]

As a result of the whole pancreas–duodenal segment graft being removed from a deceased donor and transplanted into the recipient with connection of the venous system to an appropriate venous drainage system such as the iliac vein, vena cava, or superior mesenteric vein,[7] organ-space infections may arise due to contamination from the duodenal segment. In an effort to prevent this postoperative complication, a practice of culturing the transplanted duodenal segment was initiated to preemptively prevent SSIs due to the contaminated stump by administering antibiotics directed against the microorganisms isolated from positive duodenal-segment cultures.[7,8] However, the justification for this practice remains unclear.

Current data corroborating donor duodenal segment culture with subsequent recipient organ-space SSI are inconsistent.

Author for correspondence: Coleman Rotstein, E-mail: Coleman.Rotstein@uhn.ca
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Nevertheless, some data support that any positive culture from the donor duodenal segment may be a significant risk factor for intra-abdominal infection.[8] Such infections may produce significant morbidity and may reduce patient survival.[9] Efforts to curb this morbidity and mortality are warranted. However, the practice of performing donor duodenal swab cultures and then prescribing antimicrobial therapy for the recipient based on the culture results may be unwarranted and may produce unwanted side effects including infections due to *Clostridioides difficile* and/or multidrug-resistant bacteria. By identifying accurate parameters that predict the development of SSI, there is potential to direct resources and decrease incidence of SSIs.[10] Thus, in this study, we assessed whether positive intraoperative donor duodenal-segment swab cultures predict the occurrence of organ-space SSI in pancreas transplant recipients.

Patients and methods

We conducted a retrospective review of adult patients who underwent SPK or PAK between 2000 and 2015 at the Toronto General Hospital, University Heath Network, Toronto, Canada. We included pancreas transplant recipients aged ≥18 years at the time of transplantation for the study period who had a duodenal swab sent for culture. We excluded patients with multivisceral intra-abdominal, transplants including concurrent pancreas transplantation with liver or bowel transplants and those individuals who had undergone a repeat pancreas transplantation. The study period was selected because only data for those patients who received transplants after 2000 could be accessed electronically, even though pancreas transplants have been performed since 1995 at our institute. The study protocol was approved by the institutional research ethics board.

Pancreas transplant recipients were administered perioperative prophylaxis of cefazolin 1 g every 8 hours intravenously for 3 days, with the first dose being administered prior to surgery. For patients allergic to penicillin, intravenous vancomycin for 3 days replaced cefazolin. The antimicrobial prophylaxis did not change during the study period. Surgical technique for SPK and PAK conformed to the technique previously described.[11] At the time of surgical pancreas harvest, a swab was obtained for aerobic culture from the donor duodenal segment. Susceptibilities for all isolated microorganisms were performed according to routine microbiological practices. Postoperatively, antimicrobial therapy was directed against the specific microorganisms isolated in the duodenal swab culture for 7 days. Also, maintenance immunosuppression commenced immediately after induction immunosuppression therapy had been completed as previously described.[4] After hospital discharge, follow-up for all transplant recipients was performed weekly for the first 4 weeks and then weekly or biweekly over the next 8 weeks depending on the patient's condition and complications (3 months total).

Variables

The following information was collected 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, induction and postoperative immunosuppressive regimens, duodenal-segment swab sent for culture with the swab culture results recording all pathogens and antimicrobial susceptibilities, presence of SSI within 90 days of the surgical procedure,[12] causative pathogens producing the SSIs and their susceptibilities (if available), antimicrobial therapy administered within 3 days prior to the transplant and for 30 days posttransplant plus other documented infections within our 90-day time frame and their respective treatment. SSI cultures were obtained at the time of diagnosis or when diagnostic procedures were performed (ie, interventional radiological procedures or intraoperative cultures at the time of drainage of organ-space SSIs).

Definitions

SSIs were classified according to the Centers for Disease Control classification system.[12] These infections are categorized as follows: superficial incisional SSI involves only the skin or subcutaneous tissue of the incision; deep incisional SSI involves 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 a SSI identified in the secondary incision (deep incision secondary) in an operation with >1 incision; and organ-space SSI involves any part of the body opened or manipulated during the procedure excluding the skin incision, fascia, or muscle layers.[12] Infections in other anatomic sites were diagnosed based on clinical signs of infection (eg, purulence or fever noted). SSIs were deemed present based on clinical signs of purulence, plus redness, edema, or pain confirmed by the surgeon. Cultures were obtained from superficial, deep tissue, and organ-space sites to document SSIs if purulent material was available to be cultured. In superficial SSIs, the wound was documented as infected and then cultured. If a deep SSI was confirmed, the wound was cultured. Finally, for organ-space SSIs, cultures were obtained by an interventional radiology aspirate of material or at the time of operative surgical drainage. All microbiological data were retrieved from the patient's electronic medical record with organism identification and susceptibilities when feasible.

Statistical analysis

Categorical variables comparing those patients who had positive donor duodenal-segment cultures to those with negative donor duodenal-segment cultures were analyzed using the Pearson χ^2 test or the Fisher exact test, whichever was appropriate. For continuous variables, we conducted Mann-Whitney *U* tests. We then attempted to correlate positive donor duodenal-segment cultures with the the presence of deep and organ-space SSIs using a paired analysis comparing the organisms present in the donor duodenal segment and documented SSI cultures. In addition, the positive predictive value of the donor duodenal-segment culture for the development of SSI was calculated. A similar procedure was performed for those recipients with negative donor duodenal-segment cultures. In addition, we assessed whether the risk factors of SPK and PAK interacted with a positive donor duodenal swab to promote the development of organ-space SSI by multivariate logistic regression analysis. For the multivariate model, we included all factors with P values < .20 in the univariate analysis. P values < .05 were considered statistically significant. All statistical testing was performed using SPSS version 26 software (IBM, Chicago, IL).

Results

We retrospectively reviewed all 455 patients who underwent pancreas transplantation between January 2000 and December 2015. However, 76 patients were excluded from further analysis: (1) because duodenal swab cultures were not performed (n = 44) and (2) because the patient had repeat pancreas transplantation

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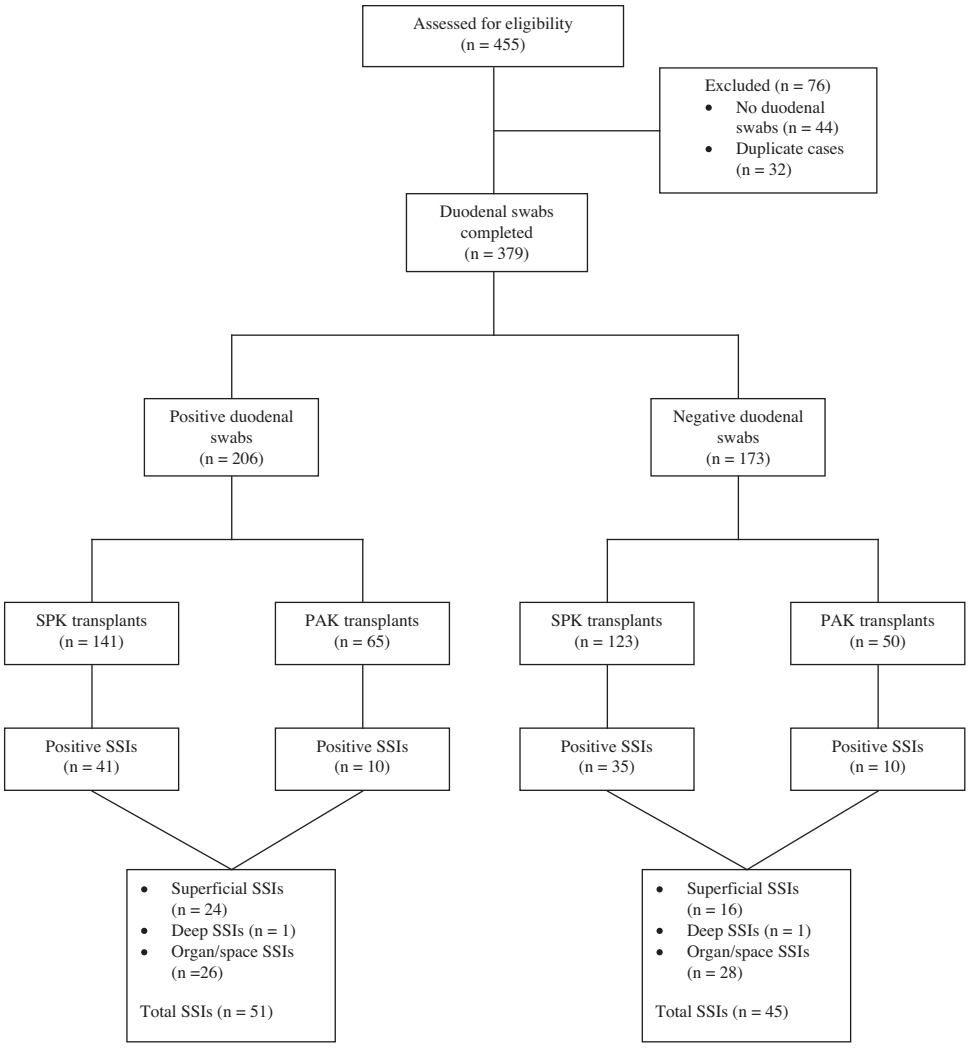

Fig. 1. Disposition of patients.

(n = 32). Thus, we focused on the 379 pancreas transplant patients for whom duodenal-segment swabs were performed (Fig. 1).

The demographic data for the 379 patients included in the study are presented in Table 1. Overall, 264 patients underwent SPK procedures and 115 patients underwent PAK procedures. In total, 206 patients (54.3%) had positive duodenal-segment cultures and 173 patients (45.7%) had negative cultures. The characteristics of patients with positive and negative duodenal swabs are compared in Table 1. No significant differences were noted between these groups except for a younger median age ($P = .021$), the administration of antimicrobial therapy both <48 hours before ($P = .024$) and >48 hours after the surgery ($P < .001$), and more dyslipidemia ($P = .016$) in those with positive duodenal cultures versus those with negative cultures. Antimicrobial therapy directed against the positive duodenal-segment swab culture pathogens was administered for 7 days.

As illustrated in Figure 1, 51 patients (24.8%) with positive duodenal cultures developed SSIs. These infections were observed in 41 of 141 SPK patients (29%) and 10 of 65 PAK patients (15.4%). Of 51 SSIs, 26 were organ-space SSIs (51%), 24 were superficial SSIs (47%), and 1 was a deep SSI (2%).

In contrast, 41 pateints (23.7%) developed SSIs among the 173 patients with negative duodenal-segment cultures. Similar to patients with positive duodenal swabs, 35 of the SSIs (28.5%) were diagnosed in SPK patients and 10 SSIs (20%) were diagnosed in the PAK patients. Of the SSIs in the group with negative duodenal cultures, 28 were organ-space SSIs (62.2%), 16 were superficial SSIs (35.6%), and 1 was a deep SSI (2.2%). The distribution of all of the SSIs is listed in Table 2. No significant differences were documented between the positive and negative duodenal culture groups. In particular, a comparison of both deep and organ-space SSIs in the positive versus the negative duodenal-segment culture groups revealed no significant difference (27 of 206 [13.1%] vs 29 of 173 [16.8%]; $P = 0.31$; $r = -0.059$). This finding indicates that the positive duodenal swab and subsequent antimicrobial therapy for the organisms isolated had little impact on the development of deep and organ-space SSIs.

Table 3 lists the microorganisms isolated in the positive duodenal cultures in the 206 patients who received pancreas transplants when the swab yielded growth. The most common microorganism isolated was *Candida* spp in 105 cultures (51%), followed by *Lactobacillus* spp in 31 cultures (15%). Yeast not otherwise specified occurred in 28 cultures (13.6%), and commensal flora occurred in 26 cultures (12.6%). Surprisingly, *Streptococcus* spp and *Enterococcus* spp were found in only 11 (5.3%) and 8 (3.4%) of the positive cultures, respectively.

The microorganisms producing SSIs were different from those isolated in the positive duodenal swabs. We specifically analyzed

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Table 1. Patient Demographics

Characteristic	All Patients (n=379)	Positive Duodenal Swabs (n=206)	Negative Duodenal Swabs (n=173)	*P* Value
Age, median y range)	50.2 (19–71)	49.5 (28–71)	51.5 (19–68)	.021
Sex, female, no. (%)	135 (35.6)	78 (37.9)	57 (33.0)	.32
PAK transplant type, no. (%)	115 (30.3)	65 (31.6)	50 (29.0)	.58
Antibiotics within 48 h of surgery, no. (%)	148 (39.1)	91 (44.2)	57 (33.0)	.024
Antibiotics after 48 h of surgery, no. (%)	220 (58.0)	159 (77.2)	61 (35.3)	<.001
Cefazolin preoperative prophylaxis no. (%)	265 (70.0)	149 (72.3)	116 (67.1)	.18
Comorbid disease				
Diabetes mellitus	352 (92.9)	180 (100.0)	172 (99.4)	1.00
Dyslipidemia	196 (51.7)	118 (57.3)	78 (45.1)	.016
Coronary artery disease	81 (21.4)	51 (24.8)	30 (17.3)	.063

Table 2. Type of SSI in Patients With Positive and Negative Duodenal Swabs

Characteristic	All Patients (n=379), No. (%)	Positive Duodenal Swabs (n=206), No. (%)	Negative Duodenal Swabs (n = 173), No. (%)	*P* Value
Positive for SSI	96 (25.3)	51 (24.8)	45 (26.0)	.98
Organ-space	54 (56.2)	26 (51.0)	28 (62.2)	.25
Superficial	40 (41.7)	24 (47.1)	16 (35.6)	.73
Deep	2 (2.1)	1 (2.0)	1 (2.2)	NA
Negative for SSI	283 (74.7)	155 (75.2)	128 (74.0)	.98

Note. SSI, surgical site infection; NA, not assessed because the cell numbers were too small.

Table 3. Microorganisms Isolated in Positive Donor Duodenal Swabs

Organism	Positive Duodenal Swabs (n=206), No. (%)
Candida spp	105 (51.0)
Lactobacillus spp	31 (15.0)
Yeast	28 (13.6)
Commensal flora	26 (12.6)
Streptococcus spp	11 (5.3)
Enterococcus spp	7 (3.4)
Enterobacter spp	8 (3.9)
Staphylococcus spp	7 (3.4)
Escherichia coli	6 (2.9)
Klebsiella spp	6 (2.9)
Bacteroides spp	3 (1.5)
Coliform	4 (1.9)
Serratia spp	4 (1.9)
Acinetobacter spp	3 (1.5)
Bifidobacterium spp	3 (1.5)
Gram-positive bacillus	3 (1.5)
Mixed fecal flora	2 (1.0)
Other species	9 (4.4)

the microorganisms responsible for superficial, deep, and organ-space infections. These SSIs types were further subdivided as those SSIs associated with positive or negative duodenal-segment cultures to assess the correlation of the positive duodenal cultures associated with the individual type of SSI (Table 4). For organ-space infection, the most frequent SSI, there was no difference in the frequency of the pathogens isolated between positive and negsative duodenal swab cultures ($P = .98$). Similarly, we assessed the microorganisms implicated in superficial SSIs among positive and negative duodenal cultures, and we did not detect any statistical difference between these 2 groups ($P = .57$). Because only 2 deep SSIs occurred in our study cohort (1 each, respectively, in the patients with and without positive duodenal cultures), we did not conducted an analysis of these infections. A paired analysis comparing the patients with positive donor duodenal cultures with the development of SSI may be seen in the supplementary data (Supplementary Table 1 online). As previously mentioned, 51 of the 206 patients (24.8%) with positive donor duodenal-segment cultures subsequently developed an SSI. Moreover, the microorganisms producing the SSIs matched those noted in the duodenal-segment culture in only 15 patients (7.8%). Thus, the positive predictive value of the donor duodenal culture was very low (16 of 206, 7.3%).

Discussion

SSIs pose a significant morbidity risk after pancreas transplantation. Indeed, we previously reported that SSIs complicated

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Table 4. Organisms Causing Surgical Site Infection (SSI)

Organism	Organ-Space SSI No. (%)			Superficial SSI No. (%)			Deep SSI No. (%)		
	Total (n=54)	Positive DS (n=26)	Negative DS (n=28)	Total (n=40)	Positive DS (n=24)	Negative DS (n=16)	Total (n=2)	Positive DS (n=1)	Negative DS (n=1)
Enterococcus spp	18 (33.3)	7 (27.0)	11 (39.3)	2 (5.0)	1 (4.2)	1 (6.3)	0	0	0
Candida spp	11 (20.4)	9 (34.6)	2 (7.1)	5 (12.5)	4 (16.7)	1 (6.3)	0	0	0
Coagulase-negative *Staphylococcus*	15 (27.8)	5 (19.2)	10 (35.7)	1 (2.5)	1 (4.2)	0 (0.0)	1 (50.0)	1 (100.0)	0
Commensal flora	13 (24.1)	8 (30.8)	5 (17.9)	22 (55.0)	10 (41.7)	12 (75.0)	0	0	0
Streptococcus spp	12 (22.2)	7 (27.0)	5 (17.9)	2 (5.0)	1 (4.2)	1 (6.3)	1 (50.0)	0	1 (100.0)
Klebsiella spp	10 (18.5)	3 (11.5)	7 (25.0)	1 (2.5)	1 (4.2)	1 (6.3)	1 (50.0)	0	1 (100.0)
Bacteroides spp	7 (13.0)	5 (19.2)	2 (7.1)	0	0	0	1 (50.0)	1 (100.0)	0
Escherichia coli	7 (13.0)	5 (19.2)	2 (7.1)	3 (7.5)	2 (8.3)	1 (6.3)	1 (50.0)	0	1 (100.0)
Pseudomonas aeruginosa	6 (11.1)	4 (15.4)	2 (7.1)	6 (15.0)	5 (20.8)	1 (6.3)	0	0	0
Enterobacter cloacae	5 (9.3)	3 (11.5)	2 (7.1)	4 (10.0)	4 (16.7)	0	0	0	0
Corynebacterium spp	5 (9.3)	1 (3.8)	4 (14.3)	0	0	0	0	0	0
Lactobacillus spp	4 (7.4)	2 (7.7)	2 (7.1)	1 (2.5)	1 (4.2)	0	0	0	0
Clostridium spp	2 (3.7)	0	2 (7.1)	0	0	0	0	0	0
Other species	18 (33.3)	11 (42.3)	7 (25.0)	7 (17.5)	3 (12.5)	4 (25.0)	1 (50.0)	1 (100.0)	0

Note: DS, duodenal swab.

24.3% overall of all pancreas transplant procedures: 29.2% of SPK transplant procedures and 15.3% of PAK transplant procedures. Moreover, organ-space SSIs accounted for 83 of the 115 SSIs (72.2%). As mentioned, one area of potential contamination predisposing patients to organ-space infections may be the pancreas–duodenal segment because it is removed from a deceased donor and transplanted into the recipient.[7,8] As a result, a common practice has emerged whereby the donor duodenal segment is cultured while performing the pancreas transplant procedure, and based on the culture results, pre-emptive treatment of the recipient for any pathogen isolated is initiated.[3,8] In our present study, we evaluated whether duodenal-segment swab cultures that demonstrate microorganisms can be correlated with subsequent SSI pathogens. If such a correlation exists, efforts directed at pre-emptive antimicrobial therapy for positive duodenal swab cultures could prevent or reduce the significant morbidity associated with SSIs.

Some investigators have attempted to establish the concordance of donor duodenal-segment cultures with subsequent pathogens causing SSIs. Woeste et al[8] demonstrated that swabs obtained from the donor's duodenum were positive in 31 of 140 cases (22.1%). In their study, 10 of the 19 patients (52.6%) undergoing relaparotomy for abdominal infection had positive duodenal swabs, but it remained unclear whether the pathogens identified from the donor duodenal-segment swab correlated with subsequent pathogens noted to cause abdominal infection at the time of relaparotomy because no matched analysis was performed for the swab culture microorganisms and the subsequent relaparotomy pathogens that caused infection.[8] In contrast, both Humar et al[13] and Troppmann et al[14] reported that positive duodenal swabs were not correlated with an increased risk of intraabdominal infections after pancreas transplantation. These investigators also failed to establish concordance of the donor duodenal-segment cultures with the cause of SSIs in pancreas transplantation.

In our study, we observed that 206 of 379 patients (54.3%) undergoing pancreas transplantation had positive donor duodenal-segment cultures. However, SSIs developed in 51 of these 206 patients (24.8%). In contrast, 41 of the 173 pancreas transplant recipients (23.7%) with negative duodenal-segment swabs also developed SSIs ($P = .81$). Thus, a positive donor duodenal-segment culture was not a predisposing factor for infection. This finding calls into question the ongoing practice of obtaining such cultures. In addition, the positive predictive value of the pathogens isolated from the positive donor duodenal swabs and those pathogens implicated in the SSIs was low particularly for those individuals with organ-space infections. Thus, one must question the utility of this practice.

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Our study has several limitations. First, there may be concern that we could have missed the initial pathogens producing SSI in the pancreas transplant recipients with positive duodenal-segment cultures that could have correlated with the pathogens causing SSI because our clinical practice was to initiate pre-emptive antimicrobial therapy for pathogens noted in the positive duodenal swab. We concede that this may have occurred, yet 24.8% of the recipients with positive duodenal cultures still developed SSIs, which is not much different than the proportion of recipients with negative duodenal-segment cultures (23.7%). Also, the development of SSI in pancreas transplantation may have occurred independent of a positive duodenal swab. Second, as with all retrospective studies, our study may have been hampered by missing data. However, we attempted to ensure very careful data collection, with special emphasis on the microbiological data. There may also be concerns about the generalizability of our rate of positive duodenal-segment swabs (54.3%) compared to 22.1% in the Woeste study. This finding may be explained by improved microbiological isolation techniques available during our study period.

In summary, we have demonstrated that donor duodenal swab cultures are not predictive of the development of SSIs in pancreas transplantation. It is essential to perform such quality assurance investigations to assess the utility of such practices in organ transplantation.

Supplementary material. To view supplementary material for this article, please visit https://doi.org/10.1017/ice.2020.262

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