ORIGINAL ARTICLE

Surgical Site Infections After Liver Transplantation: Prospective Surveillance and Evaluation of 250 Transplant Recipients in Canada

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OBJECTIVE. To evaluate the incidence of surgical-site infections (SSIs) in a cohort of liver transplant recipients and to assess risk factors predisposing patients to these infections.

DESIGN. Prospective observational cohort study.

SETTING. Single transplant center in Canada.

PATIENTS. Patients who underwent liver transplantation between February 2011 and August 2014.

METHODS. Multivariate logistic regression was used to identify independent risk factors for SSIs in liver transplant patients.

RESULTS. We enrolled 250 liver transplant recipients. The recipients' median age at the time of transplantation was 56 years (range, 19–70 years), and 166 patients (66.4%) were male. Moreover, 47 SSIs were documented in 43 patients (17.2%). Organ-space, superficial, and deep SSIs were noted in 29, 7, and 3 patients, respectively. In addition, 2 patients developed superficial and organ-space SSIs, and another 2 patients were found to have deep as well as organ-space infections. In total, we identified 33 organ-space SSIs (70.2%), 9 superficial SSIs (19.1%), and 5 deep SSIs (10.6%). Factors predictive of SSIs by multivariate analysis were duct-to-duct anastomosis (odds ratio [OR], 3.88; 95% CI, 1.85–8.13; $P < .001$) and dialysis (OR, 3.57; 95% CI, 1.02–12.50; $P = .046$). Of the 66 organisms isolated in both deep and organ-space SSIs, 55 (83%) were resistant to cefazolin.

CONCLUSIONS. Organ-space SSIs are a common complication after liver transplantation. Duct-to-duct anastomosis and dialysis were independent risk factors associated with SSIs. Appropriate perioperative prophylaxis targeting patients with duct-to-duct anastomosis and dialysis while simultaneously providing optimum coverage for the potential pathogens causing SSIs is warranted.

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Liver transplantation is a lifesaving procedure for individuals with liver failure, and successful liver transplantation has dramatically changed the outlook for individuals with irreversible liver failure. However, surgical-site infections (SSIs) may cause considerable morbidity after this surgical procedure. In fact, SSIs occur more frequently in liver transplant recipients than in other types of organ transplantation.[1–4]

Surgical-site infections are among the most common and costly nosocomial infections, accounting for 20% of all hospital-acquired infections[5] and contributing to 7–11 days of extended hospital stay.[6–8] By contributing to extended length of stay and causing readmissions, SSIs produce excessive healthcare costs. However, SSI rates are often underestimated due to lack of commitment and resources for surveillance, which is labor-intensive work. These infections may be preventable and thus provide an opportunity for reduction in healthcare expenditures and improved quality of care.

According to the Centers for Disease Control (CDC) classification scheme,[9] surgical-site infections include superficial incisional, deep incisional, and organ-space infections.[9] Because the liver transplantation surgical procedure is considered a clean-contaminated type of surgery, perioperative prophylaxis is warranted.[10] Nevertheless, despite the use of perioperative antibiotic prophylaxis to prevent SSIs in liver transplantation, these infections continue to produce considerable morbidity. Indeed, post–liver-transplantation SSIs occur at a frequency of 6% to 43% depending on the site of involvement.[1,2,11–13] Based on these studies, there appears to be a lower SSI rate than for superficial incisional SSI versus deep incisional SSI or organ-space SSI. Contributing factors for these SSIs include patient-related factors such as obesity, diabetes mellitus, alcoholism, and poor nutrition, and sodium model for end-stage liver disease (MELD) score, as well as specific factors related to the procedure itself such as duration of surgery, number of red

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blood cell transfusions, type of anastomosis, abdominal infection prior to transplantation, retransplantation, cold ischemia time, prior colonization with resistant microorganisms, and pronounced immunosuppression.[1–4,11,14–17]

This prospective study was undertaken to evaluate the incidence of postoperative infections and SSIs in a cohort of liver transplant recipients, to identify the microorganisms implicated in these infections, and to assess the risk factors predisposing patients to these infections.

METHODS

Inclusion Criteria

This prospective observational study was conducted in adult patients at the Toronto General Hospital of the University Heath Network. The local research ethics board approved the study protocol. Patients provided consent for participation either prior to their liver transplant surgical procedure at a scheduled pre-transplant clinic visit or within 4–7 days after transplantation in the case of emergent transplant procedures.

Variables

Consecutive adult patients were approached for permission to access the following information: age, sex, underlying condition most responsible for liver transplantation, comorbid conditions (diabetes mellitus, bleeding diathesis, concomitant infections, etc), MELD score, type of donation (cadaveric or living donor), donor liver mass to recipient body size, type of anastomosis, number of red blood cell transfusions intraoperatively, duration of surgical procedure, total ischemic time of the donated organ (both warm and cold ischemic times), perioperative antimicrobial prophylaxis, induction and postoperative immunosuppressive regimens, presence of SSI types within 60 days of the surgical procedure,[9] encephalopathy, presence of ascites, vasopressor usage after transplant, presence of hepatoma, bile leak, amount of transfusion, cytomegalovirus (CMV) serostatus, retransplantation, and causative pathogens implicated in the SSIs and their susceptibilities. Encephalopathy was based on clinical documentation of encephalopathy as diagnosed by the treating physicians. Dialysis was defined as requiring dialysis before transplant. Mechanical ventilation was defined as requiring mechanical ventilation after transplant surgery in intensive care unit. Notably, SSIs were assessed prospectively by the treating physicians but were categorized retrospectively by 2 independent transplant infectious disease doctors (Y.N. and C.R.).

Definitions and Statistical Analysis

Surgical-site infections were classified according the CDC classification system as follows: (1) superficial incisional involving only the skin or subcutaneous tissue of the incision; (2) deep incisional 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 involving any part of the body opened or manipulated during the procedure excluding the skin incision, fascia, or muscle layers.[9] Other anatomic sites were considered to be infected if there were clinical signs of infection (eg, fever, pulmonary infiltrates, or purulence) with microbiological evidence of a pathogen present.

Subsequently, categorical variables in the patient population comparing those patients who developed SSIs to those who did not were evaluated using χ^2 tests, with Pearson and Fisher exact tests when appropriate. For continuous variables, we performed regression analysis. An assessment of potential risk factors for SSIs in liver transplant recipients was undertaken by multivariate logistic regression analysis with stepwise backward elimination incorporating risk factors for those patients who developed SSIs compared to those who did not. For the multivariate model, we included all factors with $P < .2$ in the univariate analysis. P values $< .05$ were considered statistically significant. All statistics were performed using SPSS version 22 software (IBM, Armonk, NY). Subsequent analyses using the aforementioned format were conducted for those patients who received only a single liver transplant.

RESULTS

A total of 250 adult patients who underwent liver transplantation at our center participated in the study between February 2011 and August 2014. During that period, 548 liver transplants were performed, but only 250 patients consented to participate in our study. The overall patient demographics are shown in Table 1. Notably, the overall median age of the recipients was 56 years (range, 19–70 years), and 66.4% were male. The median sodium MELD score was 21. Overall, 241 patients (96.4%) had only a liver transplant, while 8 patients (3.2%) had both liver and kidney transplants and 1 patient (0.4%) had liver and lung transplants simultaneously. Deceased donor liver transplants were performed in 180 patients (72.0%), and 70 patients (28.0%) had living donor grafts. In our hospital, cefazolin plus metronidazole are the standard perioperative prophylaxis; they were given in 189 of 250 patients (75.6%), with another 9 patients (3.6%) receiving cefazolin alone. Another 8% of patients received vancomycin plus metronidazole for purported cefazolin allergy, and the remainder received miscellaneous antibiotics. Fungal prophylaxis was not employed universally for our cohort. Fluconazole was administered as antifungal prophylaxis in only 4 of 250 patients (1.6%) within 7 days after transplant. We collected specimens for fungal culture when sampling for infection.

Table 1 also shows the underlying diagnoses leading to liver transplantation in the study cohort. Hepatitis C with alcoholic cirrhosis predominated (30.8%). The induction and maintenance immunosuppressive therapy used in the study patients is documented in Table 2.

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TABLE 1. Patient Demographics

Characteristic	No. (%) (N = 250)
Age, y, median (range)	56 (19–70)
Gender, female	84 (33.6)
BMI, kg/cm^2, median (range)	26.2 (14.5–47.4)
Sodium MELD score	21 (3–47)
Underlying disease	
Hepatoma	87 (34.8)
Hepatitis C	77 (30.8)
Alcoholic cirrhosis	44 (17.6)
Primary sclerosing cholangitis	34 (13.6)
Nonalcoholic steatohepatitis	30 (12.0)
Primary biliary cirrhosis	14 (5.6)
Hepatitis B	11 (4.4)
Comorbid diseases	
Ascites	131 (52.4)
Encephalopathy	72 (28.8)
Esophageal varices	98 (39.2)
Cholangitis	14 (5.6)
Diabetes mellitus	62 (24.8)
Dialysis	15 (6.0)
No. of transplants	
1	235 (94.0)
2	15 (6.0)
Transplant type	
Liver	241 (96.4)
Liver and Kidney	8 (3.2)
Liver and Lung	1 (0.4)
Donor status	
Deceased	180 (72.0)
Living, related	70 (28)
Cytomegalovirus serology	
Donor (+), recipient (+)	84 (33.6)
Donor (+), recipient (−)	29 (11.6)
Donor (−), recipient (+)	88 (35.2)
Donor (−), recipient (−)	49 (19.6)
Perioperative issues	
Duct-to-duct anastomosis	67 (26.8)
Transfusion, units, median (range)	3 (0–24)
Surgical time, h, median (range)	6.0 (1.1–14.1)
Mechanical ventilation	190 (76.0)

NOTE. BMI, body mass index; MELD, model for end-stage liver disease.

TABLE 2. Induction and Maintenance Immunosuppression

Type	No. (%)
Induction immunosuppression	
Antithymocyte globulin	15 (6.0)
Basiliximab	142 (56.8)
Mehylprednisolone (≥500 mg)	250 (100.0)
Maintenance immunosuppression	
Steroids (prednisone)	250 (100.0)
Mycophenolate mofetil or myfortic	197 (78.8)
Tacrolimus	189 (75.6)
Cyclosporine	70 (28.0)
Sirolimus	20 (8.0)
Azathioprine	7 (2.8)

The demographics of the 43 recipients who developed SSIs compared with those who did not are found in Table 3. In the univariate analysis, the following risk factors were statistically significant: induction immunosuppression use of antithymothyte globulin (ATG)/basiliximab (OR, 2.85; 95% CI, 1.30–6.24; $P = .005$), duct-to-duct anastomosis (OR, 4.26; 95% CI, 2.15–8.46; $P < .001$), bile leak (OR, 4.14; 95% CI, 1.07–16.12; $P = .05$), and previous transplant (OR, 3.57; 95% CI, 1.20–10.62; $P = .027$). Donor type, induction immunosuppression with ATG/basiliximab, duct-to-duct anastomosis type, bile leak, transfusion of ≥5 units, encephalopathy before transplant, dialysis before transplant, hepatoma, surgical time, and retransplant were entered into the multivariate model. Only duct-to-duct anastomosis (OR, 3.88; 95% CI, 1.85–8.13; $P < .001$) and dialysis before transplantation (OR, 3.57; 95% CI, 1.02–12.50; $P = .046$) were statistically significant risk factors associated with SSIs in liver transplantation according to our multivariate analysis. In addition, a trend was noted for an association between SSI and bile leak (OR, 4.03; 95% CI, 0.91–17.88; $P = .066$) and between SSI and induction immunosuppression with ATG/basiliximab (OR, 2.15; 95% CI, 0.92–5.05; $P = .078$).

Moreover, the 47 SSIs identified in 43 patients were comprised of superficial SSI in 7 patients, deep incisional SSI in 3 patients, and organ-space SSI in 29 patients. Superficial and organ-space SSIs were noted in 2 patients and deep incisional and organ-space SSIs were noted in 2 patients. Thus, among 47 SSIs, 33 (70.2%) were organ-space SSIs, 9 (19.1%) were superficial SSIs, and 5 (10.6%) were deep SSIs. Because organ-space SSIs were most common, further univariate and multivariate analyses of the factors associated with these infections were undertaken (Table 4). In the multivariate model of factors predictive of organ-space SSIs, duct-to-duct anastomosis (OR, 2.91; 95% CI, 1.28–6.58; $P = .011$), bile leak (OR, 4.85; 95% CI, 1.07–22.22; $P = .040$), induction immunosuppression with ATG/basiliximab (OR, 3.08; 95% CI, 1.11–8.55; $P = .030$), and retransplant (OR, 4.33; 95% CI 1.20–15.62) emerged as the key factors predisposing recipients to these SSIs. In a secondary analysis, we focused on those patients who only received 1 liver transplant (n = 241). We evaluated both total SSIs and organ-space SSIs (see Online Supplementary Tables). For total SSIs, the multivariate analysis revealed that dialysis and duct-to-duct anastomosis were significant factors. In contrast, for organ-space SSIs, induction regimen, duct-to-duct anastomosis, and retransplantation were significant factors. Of the 9 patients with multiple transplants, 2 patients had SSIs and both of these were due to organ-space SSIs.

The time from the transplant to SSI was a median of 9 days (range, 1–33 days). For superficial SSI, the time elapsed from transplantation to the diagnosis of the SSI was a median of 9 days (range, 5–24 days). For deep incisional SSI, the time elapsed from transplantation to the diagnosis of the SSI was a median of 9 days (range, 3–33 days). For organ-space SSI,

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TABLE 3. Risk Factors for Surgical-Site Infections (SSIs) Among Liver Transplant Recipients

Characteristic	SSIs (N = 43), No. (%)	Without SSIs (N = 207), No. (%)	Univariate *P* Value	Univariate OR (95% CI)	Multivariate *P* Value	Multivariate OR (95% CI)
Male gender	18 (41.9)	66 (31.9)	.21	0.65 (0.33–1.27)	…	…
Living donor	17 (39.5)	53 (25.6)	.064	1.90 (0.96–3.77)	…	…
Diabetes mellitus	9 (20.9)	53 (25.6)	.52	0.77 (0.35–1.71)	…	…
ATG/basiliximab	34 (79.1)	118 (57.0)	.005	2.85 (1.30–6.24)	.078	2.15 (0.92–5.05)
Duct-to-duct anastomosis	23 (53.5)	44 (21.3)	<.001	4.26 (2.15–8.46)	<.001	3.88 (1.85–8.13)
Bile leak	4(9.3)	5 (2.4)	.05	4.14 (1.07–16.12)	.066	4.03 (0.91–17.88)
Transfusion >5 units	9 (20.9)	65 (31.6)	.17	0.57 (0.26–1.27)	…	…
Mycophenolate mofetil or myfortic	35 (81.4)	162 (78.3)	.65	1.22 (0.53–2.80)	…	…
Encephalopathy	11 (25.6)	87 (42.0)	.044	0.47 (0.23–0.99)	.053	0.45 (0.20–1.01)
Dialysis	5 (11.6)	10 (4.8)	.12	2.59 (0.84–8.01)	.046	3.57 (1.02–12.50)
Mechanical ventilation	30 (69.8)	161 (77.8)	.26	0.66 (0.32–1.37)	…	…
Ascites	24 (55.8)	129 (62.3)	.43	0.76 (0.39–1.48)	…	…
Hepatoma	10 (23.3)	77 (37.2)	.08	0.51 (0.24–1.10)	…	…
Age, y, median (range)	55 (20–67)	56 (19–70)	.32	0.99 (0.96–1.01)	…	…
BMI, kg/cm^2, median (range)	27.0 (18–45)	25.8 (15–47)	.49	1.02 (0.96–1.09)	…	…
Retransplant	6 (14.0)	9 (4.3)	.027	3.57 (1.20–10.62)	…	…
CMV D+ /R–[a]	2 (4.7)	27 (13.0)	.19	0.33 (0.07–1.42)	…	…
MELD score, median (range)	21 (6–44)	21 (3–47)	.77	1.01 (0.97–1.04)	…	…
Surgical time, h, median (range)	6.3 (3.6–11.2)	5.9 (1.1–14.1)	.19	1.12 (0.95–1.32)	…	…
Cold ischemic time	…	…	.68	…	…	…
0–2 h	14 (32.6)	49 (23.7)	…	…	…	…
2–4 h	4 (9.3)	20 (9.7)	…	…	…	…
4–8 h	15 (34.9)	89 (43.0)	…	…	…	…
8–12 h	10 (23.3)	49 (23.7)	…	…	…	…

NOTE. OR, odds ratio; CI, confidence interval; ATG, antithymocyte globulin; BMI, body mass index; CMV, cytomegalovirus; D, donor; R, recipient; MELD, model for end-stage liver disease.

[a]CMV D+/R–, donor seropositive and recipient seronegative for cytomegalovirus.

the time elapsed from transplantation to the diagnosis of the SSI was a median of 8.5 days (range, 1–33 days). For superficial SSIs, methicillin-sensitive *Staphylococcus aureus* (known to be cefazolin susceptible) was the main cause of SSIs. In contrast, 55 of 66 organisms (83%) identified in both deep and organ-space SSIs were resistant to cefazolin (Figure 1).

DISCUSSION

This study is the first to assess SSIs after liver transplantation in Canada. We documented that 17.2% of liver transplants recipients in our cohort developed SSIs postoperatively, which is very similar to the rate reported by Viehman et al[18] (18%). Moreno et al[19] estimated the occurrence of SSIs after liver transplantation in Spain to be ~10%. In contrast, Yamamoto et al[20] evaluated SSIs in adult living donor liver transplants and found rates of 30.3% and 41.3% in 2 different time periods; in addition, they claimed that SSIs may be higher in living donor than cadaveric transplants due to increased surgical difficulty. However, a recent meta-analysis reported a pooled proportion of SSI infection rate of 11.8% in deceased donor transplants (95% CI, 5.4%–20.2%).[21] Our SSI rate was in line with these findings.

Organ-space SSIs occurred in 31 of 43 patients (72%) after liver transplantation in our study. The predominance of organ-space infections differs from the findings of earlier investigations of SSIs after liver transplantation. Older studies reported deep wound infections including intra-abdominal complications in 15%–19% of liver transplant recipients, with superficial SSIs accounting for 6%–8%.[22–24] More recent data claimed higher rates of SSI; Garcia-Prado et al[2] reported a rate of 33.7% among their cohort of liver transplants with a preponderance of organ-space SSIs (91% of all SSIs). Others have corroborated these findings and have reported rates of organ-space infections between 77.7% and 84.5% of all SSIs.[11,13] Our data regarding the predominance of organ-space SSIs after liver transplantation are certainly in keeping with these latter studies. Also, we showed that SSIs occurred at a median of 9 days after transplant, which is earlier than reported by Viehman et al.[18]

We sought contributing factors for the development of SSIs from the list of factors previously identified.[1–4,10,13–16] However, no correlates emerged from this list in our univariate and multivariate analyses. The risk factors predictive of SSIs in our study were duct-to-duct anastomosis as well as dialysis before transplantation. It is unclear why infections were more common in recipients with duct-to-duct anastomosis than with Roux-en-y reconstructions. Clinically evident bile leaks were noted in 9 patients in our cohort and were more common in patients undergoing Roux-en-y procedures

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TABLE 4. Risk Factors for Organ-Space Infections in Liver Transplant Recipients

Characteristic	Organ-Space SSIs (N = 33)	Without Organ-Space SSIs (N = 217)	Univariate *P* Value	Univariate OR (95% CI)	Multivariate *P* Value	Multivariate OR (95% CI)
Male gender	16 (48.5)	68 (31.3)	.052	2.06 (0.98–4.33)	…	…
Living donor	13 (39.4)	57 (26.3)	.12	1.83 (0.85–3.91)	…	…
Diabetes mellitus	7 (21.2)	55 (25.3)	.61	0.79 (0.33–1.93)	…	…
ATG/Basiliximab	27 (81.8)	125 (57.6)	.008	3.31 (1.31–8.35)	.030	3.08 (1.11–8.55)
Duct to duct anastomosis	18 (54.5)	49 (22.6)	<.001	4.11 (1.93–8.76)	.011	2.91 (1.28–6.58)
Bile leak	4 (12.1)	5 (2.3)	.005	5.85 (1.49–23.01)	.040	4.85 (1.07–22.22)
Transfusion >5 units	8 (24.2)	66 (30.6)	.54	0.73 (0.31–1.70)	…	…
Mycophenolate mofetil or myfortic	28 (84.8)	169 (77.9)	.36	1.59 (0.58–4.34)	…	…
Encephalopathy	8 (24.2)	90 (41.5)	.059	0.45 (0.20–1.05)	…	…
Dialysis	3 (9.1)	12 (5.5)	.42	1.71 (0.46–6.41)	…	…
Mechanical ventilation	22 (66.7)	169 (77.9)	.16	0.57 (0.26–1.25)	…	…
Ascites	20 (60.6)	133 (61.3)	.94	0.97 (0.46–2.06)	…	…
Hepatoma	6 (18.2)	81 (37.3)	.32	0.37 (0.15–0.94)	…	…
Retransplant	6 (18.2)	9 (4.1)	.007	5.14 (1.70–15.56)	.026	4.33 (1.20–15.62)
CMV D+/R–[a]	2 (6.1)	27 (12.4)	.39	0.45 (0.10–2.01)	…	…
Age, y, median (range)	55 (21–67)	56 (19–70)	.77	0.99 (0.96–1.03)	…	…
BMI, kg^2/cm (range)	26.9 (18–45)	25.9 (15–47)	.43	1.03 (0.96–1.10)	…	…
MELD score, median (range)	21 (6–44)	21 (3–47)	.81	1.01 (0.97–1.05)	…	…
Surgical time, h, median (range)	6.3 (3.6–11.2)	5.9 (1.1–14.1)	.095	1.17 (0.97–1.40)	…	…
Cold ischemic time						
0–2 h	11 (33.3)	52 (24.0)	…	…	…	…
2–4 h	3 (9.1)	21 (9.7)	…	…	…	…
4–8 h	11 (33.3)	93 (42.9)	…	…	…	…
8–12 h	8 (24.2)	51 (23.5)	.65	…	…	…

NOTE. OR, odds ratio; CI, confidence interval; ATG, antithymothyte globulin; CMV, cytomegalovirus; D, donor; R, recipient; BMI, body mass index; MELD, model for end-stage liver disease.

[a]CMV D+/R–, donor seropositive and recipient seronegative for cytomegalovirus.

(6 vs 3 patients who had duct-to-duct anastomosis), but infections occurred in 2 patients with each procedure, respectively. However, imperceptible bile leaks that were not clinically evident may have been associated with duct-to-duct anastomosis, thus promoting fluid accumulation intraabdominally that may have provided a growth medium and enhanced organism growth. Interestingly, Viehman et al[18] also found that SSIs after liver transplantation were associated with the duration of the operation ($P < .001$) and bile leakage ($P < .001$), emphasizing the role of fluid leakage possibly providing a growth medium. Dialysis before transplantation may merely be a marker for the severity of illness and a debilitated state, but it may also reflect the impairment of immune responsiveness that occurs with renal failure.[25]

When focusing on the organ-space SSIs, duct-to-duct anastomosis was again a prominent risk factor for these infections. Moreover, bile leak, a less frequent event in our study population, played a greater role in predisposing patients to infection similar to the study of Viehman et al.[18] Again, both duct-to-duct anastomosis and bile leak produce fluid accumulation in the abdomen, possibly providing a growth medium for organisms. Finally, induction immunosuppression with ATG/basiliximab also enhances the risk of these infections.[18]

The use of cefazolin as perioperative antimicrobial prophylaxis may have predisposed our patients to the development of cefazolin-resistant organisms. In fact, in our study, most microorganisms causing deep and organ-space SSIs were Enterobacteriaciae as well as Enterococcus spp. that were not susceptible to cefazolin. Viehman et al[18] documented that deep SSIs caused by multidrug-resistant bacteria are on the rise in the United States. In contrast, in our cohort, we found that SSIs occurred earlier (ie, ~9 days from the time of transplantation) than in the cohort of Viehman et al[18] (ie, 12.5 days for superficial and 13.5 days for deep SSI). Because of the earlier onset of infection in our study, implementing perioperative antibiotic prophylaxis addressing the most common pathogens causing SSIs and of sufficient duration may prove effective in reducing the incidence of SSIs. Assessment of effective perioperative antimicrobial prophylaxis in liver transplantation is warranted and should be derived through well-designed prospective study.

Our study has several limitations. First, some data were collected retrospectively, which led to missing information. Specifically, the operative data of warm ischemic times were collected in this way and resulted in some missing data. Second, we could not accurately report any extensions of length of stay attributable to SSIs because of concomitant morbidities that may

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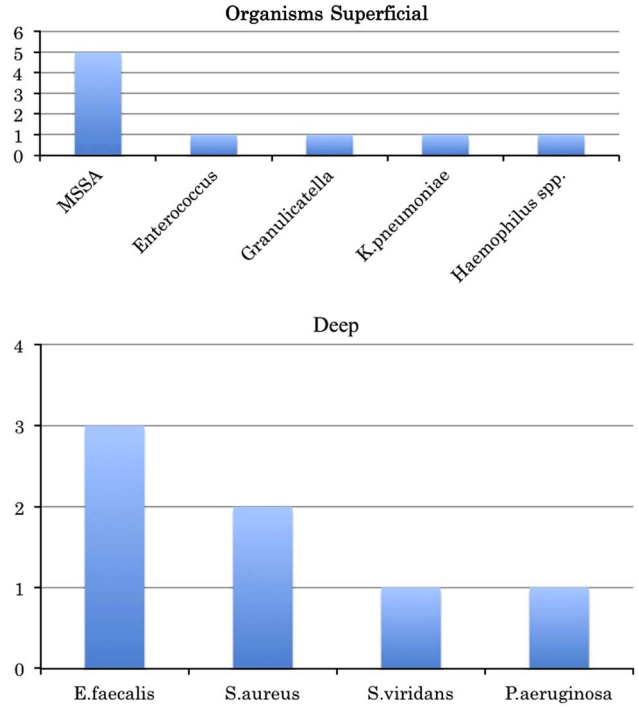

FIGURE 1. Microorganisms causing surgical-site infections.

also have influenced delays in the time of discharge, making it impossible to quantify the economic impact of these infections. Thus, it was difficult to gauge the cost impact of these infections. These patients had significant comorbid disease that impeded our efforts to isolate the SSI contribution to extended lengths of stay. Third, this study was conducted in a single transplant center, which may not be generalizable to other centers. Finally, we only enrolled ~250 of 548 of all liver transplant recipients (45.6%) during the study period. Notwithstanding these limitations, this is one of the larger studies evaluating SSIs and postoperative infections in liver transplant recipients prospectively.

In conclusion, organ-space SSIs are the most common type of postoperative SSI noted in liver transplant recipients. It appears that the predisposing factors of duct-to-duct anastomosis and dialysis before transplantation enhance the risk of SSIs. As perioperative antimicrobial prophylaxis is a key element of the prevention of postoperative infections, efforts to enhance perioperative prophylaxis coverage to address the pathogens implicated in these infections may be warranted.

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SUPPLEMENTARY MATERIAL

To view supplementary material for this article, please visit https://doi.org/10.1017/ice.2017.131

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