Organ transplantation extends the life of >25,000 patients with end-stage organ disease each year in the United States (1). Patient outcomes have improved over time with the advent of potent immunosuppressive agents, improvement in surgical techniques, and expanded use of antimicrobial prophylaxis (2). Despite these advances, infections remain a leading cause of morbidity and mortality among transplant recipients (2–4). One method of categorizing post transplant infections is based on the time of onset. Most infections during the first month post transplant represent typical nosocomial infections expected in any surgical patient; rarely, infections that were incubating in the recipient pre-transplant or donor-derived infections (infections transmitted with the allograft) may manifest themselves early post transplant (2). Opportunistic infections typically present between 1 and 6 months post transplant, but may present later with enhancement of immunosuppression in response to rejection or late discontinuation of antimicrobial prophylaxis (2). Most infections that occur after 6 months represent community-acquired infections or, rarely, opportunistic infections (2).

Postsurgical nosocomial infections, including pneumonia, bacteremia, and urinary tract infections (UTIs) and surgical-site infections (SSIs), have been studied extensively in the general surgical population and in several specialty surgical populations. The most common nosocomial infections in surgical patients are pneumonia (33%), UTI (18%), SSI (14%), and bloodstream infection (BSI) (13%) (5). Data on the epidemiology of postsurgical infectious complications and advice on reducing their incidence are largely derived from the American College of Surgeon’s National Surgical Quality Improvement Program (ACS-NSQIP), an initiative designed to promote the identification and
prevention of surgical complications (6). Unfortunately, NSQIP includes few (~10) transplant programs, and therefore, data are limited on post transplant surgical infectious complications in their reports. Epidemiology of early post transplant nosocomial infections must therefore be extrapolated from the literature, and much of the existing literature represents single-center experience and is retrospective in nature.

The goal of this review was to survey the literature on early post transplant (<30 days post transplant) nosocomial infections. PubMed was searched using the following search terms: “early nosocomial infection transplant,” “pneumonia transplant,” “urinary tract infection transplant,” “bacteremia transplant,” “Clostridium difficile transplant,” and “surgical site infection transplant.” Only English-language articles that described infections that occurred within the first 30 days after any solid organ transplant (SOT) were reviewed. Studies that included large cohorts and those with data derived from multivariate analysis were deemed of greater importance in making conclusions about the epidemiology of early post transplant nosocomial infections. A summary of these studies is presented in Table 1 (7–73).

Pneumonia

Pneumonia represents the most common nosocomial infection in all surgical patients (5). The US Centers for Disease Control and Prevention (CDC) defines 3 broad categories of pneumonia for the purposes of nosocomial surveillance: clinically defined pneumonia, pneumonia with specific laboratory findings, and pneumonia in immune-compromised patients. The threshold for diagnosis of this latter category is lowered in this more vulnerable population, but laboratory findings are still required (74). The American Thoracic Society and Infectious Diseases Society of America have distinguished nosocomial pneumonias by mechanism of acquisition: healthcare-associated pneumonia, for patients with extensive healthcare contact; hospital-acquired pneumonia (HAP), occurring within 48 h after hospital admission; and ventilator-associated pneumonia, a subgroup of HAP that develops >48 h after endotracheal intubation (75).

In the general patient population, HAP and ventilator-associated pneumonia are associated with mortality rates as high as 40% (76). Risk factors for HAP include aspiration, colonization of the aerodigestive tract with pathogenic organisms, chronic obstructive pulmonary disease, acute respiratory distress syndrome, head trauma, coma, tracheostomy, reintubation, supine positioning, patient transport, the presence of a nasogastric tube or intracranial pressure monitoring, enteric nutrition, prior antibiotic exposure, increased age, administration of antacids or histamine type-2 antagonists, and witnessed aspiration (12). The pathogens that most frequently cause HAP include Pseudomonas aeruginosa, Enterobacter species, Klebsiella pneumoniae, Acinetobacter species, and methicillin-resistant Staphylococcus aureus (MRSA) (12).

Pneumonia in the SOT population is most often HAP and affects patients early in the course of their recovery from transplant surgery. Nosocomial pneumonia (odds ratio [OR]: 18.9, 95% confidence interval [CI]: 1.8–193.8, \( P = 0.013 \)) and the need for mechanical ventilation (OR: 16.3, 95% CI: 3.3–80.3, \( P = 0.001 \)) have been identified as 2 primary risk factors for increased mortality (77). Although etiology of the infection varies, MRSA is a frequently isolated pathogen (78).

Pneumonia in liver transplantation

Early onset pneumonia in liver transplant recipients occurs at an incident rate of 13.7–25.4% (7–9, 79–81). In this period, Enterobacteriaceae, Haemophilus influenzae, and P. aeruginosa are the most common pathogens causing pneumonia (7, 8) Although studies from the 1990s suggested that 1–8% of liver transplant recipients developed nosocomial pulmonary aspergillosis, more recent data suggest aspergillosis is a rare early complication: 45% of Aspergillus infections occurring in the first 90 days after transplantation between 1998 and 2001, while 75% of Aspergillus infections occurred in that period between 1990 and 1995 (\( P = 0.026 \)) (80, 82–87). Risk factors for Aspergillus in the liver transplant population include re-transplantation (OR: 29.9; 95% CI: 2.10–425; \( P = 0.02 \)) and requirement for dialysis (OR: 24.5; 95% CI: 1.25–354; \( P = 0.03 \)) (10, 85).

Elevated international normalized ratio (>2.3) before liver transplantation, which is likely a marker of more advanced liver failure (OR: 4.95; 95% CI: 1.86–8.59, \( P = 0.0004 \)), and restrictive lung physiology measured by pre-transplant pulmonary function testing (OR: 3.14; 95% CI: 1.51–6.51; \( P = 0.002 \)) have been found to increase the risk of acquiring pneumonia (8). Surgical technique also influences the risk of pneumonia in liver transplant recipients, with the use of piggyback anastomosis determined on multivariate analysis to be protective (OR: 0.45; 95% CI: 0.21–0.94; \( P = 0.027 \)), compared with traditional caval anastomosis (9). Prolonged assisted mechanical ventilation (OR: 1.51; 95% CI: 1.02–2.24; \( P = 0.046 \)), pulmonary noninfectious abnormalities observed radiographically (atelectasis,
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<th>Infection type</th>
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<th>Risk factors – solid organ transplant (SOT) recipients</th>
<th>Risk factors – general population (Reference)</th>
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<tr>
<td>Respiratory tract</td>
<td>Liver (7–11)</td>
<td>• Enterobacteriaceae&lt;br&gt;• <em>Staphylococcus aureus</em>&lt;br&gt;• <em>Haemophilus influenzae</em>&lt;br&gt;• <em>Pseudomonas aeruginosa</em></td>
<td>Risk factors for infection:&lt;br&gt;• Restrictive lung physiology&lt;br&gt;• Noninfectious radiographic abnormalities&lt;br&gt;• Pulmonary edema&lt;br&gt;• Elevated INR&lt;br&gt;• Prolonged mechanical ventilation&lt;br&gt;<strong>Protective:</strong> Piggyback anastomosis</td>
<td>Risk factors for infection (12):&lt;br&gt;• Advanced age&lt;br&gt;• Chronic obstructive pulmonary disease&lt;br&gt;• Acute respiratory distress syndrome&lt;br&gt;• Head trauma&lt;br&gt;• Coma&lt;br&gt;• Aerodigestive tract colonization&lt;br&gt;• Aspiration&lt;br&gt;• Tracheostomy&lt;br&gt;• Reintubation&lt;br&gt;• Nasogastric tube&lt;br&gt;• Supine positioning&lt;br&gt;• Patient transport&lt;br&gt;• Intracranial pressure monitoring&lt;br&gt;• Enteric nutrition&lt;br&gt;• Prior antibiotic exposure&lt;br&gt;• Antacids/histamine type-2 antagonists&lt;br&gt;• Witnessed aspiration</td>
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<td>Risk factors for Aspergillus infection:&lt;br&gt;• Re-transplantation&lt;br&gt;• Post-transplant dialysis</td>
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<td>Risk factors for mortality:&lt;br&gt;• Mechanical ventilation&lt;br&gt;• Pulmonary edema&lt;br&gt;• Emergency living-donor liver transplantation&lt;br&gt;<strong>Protective:</strong> Piggyback anastomosis</td>
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<td>Kidney (13–15)</td>
<td>• <em>P. aeruginosa</em>&lt;br&gt;• <em>Streptococcus haemolyticus</em>&lt;br&gt;• <em>S. aureus</em>&lt;br&gt;• <em>H. influenzae</em>&lt;br&gt;• <em>Klebsiella pneumoniae</em>&lt;br&gt;• <em>Acinetobacter species</em></td>
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<td>Heart (16–18)</td>
<td>• <em>P. aeruginosa</em>&lt;br&gt;• <em>Acinetobacter baumannii</em>&lt;br&gt;• <em>Enterobacter cloacae</em>&lt;br&gt;• <em>S. aureus</em></td>
<td>Risk factors for mortality:&lt;br&gt;• Mechanical ventilation</td>
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<td>Lung (19–24)</td>
<td>• <em>P. aeruginosa</em>&lt;br&gt;• <em>S. aureus</em>&lt;br&gt;• <em>baumannii</em>&lt;br&gt;• <em>Klebsiella species</em>&lt;br&gt;• <em>Aspergillus species</em></td>
<td>Risk factors for infection:&lt;br&gt;• Pre-operative gram-negative rod colonization</td>
<td>Risk factors for Aspergillus infection:&lt;br&gt;• Daclizumab induction&lt;br&gt;• Advanced donor age&lt;br&gt;• Prolonged ischemia time</td>
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<td>Risk factors for mortality:&lt;br&gt;• Cystic fibrosis</td>
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<td>Infection type</td>
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<td>Urinary tract infection</td>
<td>Liver (25, 26)</td>
<td>• <em>Enterococcus</em> species&lt;br&gt;• <em>Escherichia coli</em>&lt;br&gt;• <em>E. cloacae</em>&lt;br&gt;• <em>A. baumannii</em></td>
<td>Risk factors for infection of pediatric recipients:&lt;br&gt;• Decreased recipient age&lt;br&gt;• Biliary atresia</td>
<td>Risk factors for infection (27):&lt;br&gt;• Female gender&lt;br&gt;• Advanced age&lt;br&gt;• Diabetes mellitus&lt;br&gt;• Serum creatinine &gt;2 mg/dL&lt;br&gt;• Prolonged Foley catheter placement</td>
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<td>Kidney (28-34)</td>
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<td>• <em>E. coli</em>&lt;br&gt;• <em>K. pneumoniae</em>&lt;br&gt;• <em>Enterococcus</em> species&lt;br&gt;• <em>Serratia marcescens</em>&lt;br&gt;• <em>E. cloacae</em></td>
<td>Risk factors for infection:&lt;br&gt;• Female gender&lt;br&gt;• Advanced recipient age&lt;br&gt;• Glomerulonephritis&lt;br&gt;• Ureteral stent&lt;br&gt;• Decreased donor&lt;br&gt;• Simultaneous double renal transplant&lt;br&gt;• Delayed graft function&lt;br&gt;• Prolonged Foley catheter placement&lt;br&gt;• Change in initial immunosuppression&lt;br&gt;• <em>Protective:</em> Length of hospitalization before infection&lt;br&gt;• <em>Protective:</em> TMP-SMX prophylaxis</td>
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<td>Surgical-site Infections</td>
<td>Liver (35–39)</td>
<td>• <em>Enterococcus</em> species&lt;br&gt;• <em>S. aureus</em>&lt;br&gt;• Coagulase-negative staphylococci&lt;br&gt;• <em>Candida</em> species&lt;br&gt;• <em>P. aeruginosa</em>&lt;br&gt;• <em>K. pneumoniae</em></td>
<td>Risk factors for infection:&lt;br&gt;• Decreased donor liver mass-to-recipient body mass ratio&lt;br&gt;• Antibiotic use 3 months before transplant&lt;br&gt;• Operative time beyond 3.5 h</td>
<td>Risk factors for infection (40, 41):&lt;br&gt;• Advanced age&lt;br&gt;• Diabetes mellitus&lt;br&gt;• Urinary incontinence&lt;br&gt;• Complete neurologic deficit&lt;br&gt;• The presence of &gt;3 comorbid diseases&lt;br&gt;• Tobacco use&lt;br&gt;• Poor nutritional status&lt;br&gt;• Revision surgery&lt;br&gt;• Use of nonsteroidal anti-inflammatory drugs&lt;br&gt;• Blood transfusion&lt;br&gt;• Prolonged operative time</td>
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<td>Kidney (42–45)</td>
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<td>• <em>Enterococcus</em> species&lt;br&gt;• Coagulase-negative staphylococci&lt;br&gt;• <em>E. coli</em>&lt;br&gt;• <em>S. aureus</em>&lt;br&gt;• <em>K. pneumoniae</em></td>
<td>Risk factors for infection:&lt;br&gt;• Elevated BMI&lt;br&gt;• Pre-transplant diabetes mellitus&lt;br&gt;• Pre-transplant chronic glomerulonephritis&lt;br&gt;• Delayed graft function&lt;br&gt;• Acute graft rejection&lt;br&gt;• Sirolimus maintenance (reference: maintenance regimen that replaced sirolimus with a calcineurin inhibitor)&lt;br&gt;• Reoperation following transplantation&lt;br&gt;• <em>Protective:</em> Living donor</td>
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<td>Heart (46-53)</td>
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<td>• Coagulase-negative streptococci • S. aureus • Candida species</td>
<td>Risk factors for infection: • Advanced recipient age • Elevated BMI • Previous heart surgery • Previous VAD placement • Inotropic support • Sirolimus maintenance (reference: mycophenolate mofetil) • Tacrolimus maintenance (reference: cyclosporine) • Increased cardiac bypass time</td>
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<td>Lung (54)</td>
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<td>• S. aureus • P. aeruginosa • Enterococcus faecium</td>
<td>Risk factors for infection: • Diabetes mellitus • Prior cardiothoracic surgery • Female donor • Ischemic time • Number of red blood cell units transfused</td>
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<td>Bloodstream Infections</td>
<td>Liver (55-62)</td>
<td>• Coagulase-negative staphylococci • S. aureus • Enterococcus species • A. baumannii • E. coli • P. aeruginosa</td>
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<td>Risk factors for infection (63): • Advanced age • Diabetes mellitus • Peripheral vascular disease • Congestive heart failure • Renal disease • Hepatic disease • Red blood cell transfusion • Parenteral nutrition • Immunosuppression</td>
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<td>Risk factors for candidemia: • Age &lt;1 year • Bile duct complication</td>
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<td>Risk factors for mortality: • S. aureus bacteremia</td>
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<td>Risk factors for candidemia mortality: • Diabetes mellitus • Non-albicans Candida infection (Reference: Candida albicans) • Diagnosis &gt;30 days after transplantation</td>
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Table 1 Continued

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<tr>
<th>Infection type</th>
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<tr>
<td>Kidney (64–66)</td>
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<td>• E. coli</td>
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<td>• K. pneumoniae</td>
<td>• Pre-transplant hemodialysis</td>
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<td>• Enterobacter aerogenes</td>
<td>• Deceased donor</td>
<td>• Chronic comorbid conditions (chronic kidney disease, HIV infection, inflammatory bowel disease)</td>
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<td>• Coagulase-negative staphylococci</td>
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<td>• S. aureus</td>
<td>• Local infection</td>
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<td>• Enterococcus species</td>
<td>• Post-transplant ureteric stent insertion</td>
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<td>• P. aeruginosa</td>
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<td>• S. aureus</td>
<td>• Viral infection</td>
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<td>• Staphylococcus epidermidis</td>
<td>• Prolonged ICU stay</td>
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<td>Clostridium difficile infection</td>
<td>All SOTs (68–71)</td>
<td>Risk factors for infection of liver transplant recipients:</td>
<td>Risk factors for infection (72, 73):</td>
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<td>• Liver or lung organ recipients have a higher rate than kidney transplant recipients</td>
<td>• High MELD score</td>
<td>• Advanced age</td>
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<td>• Presence of a systemic infection</td>
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INR, international normalized ratio; TMP-SMX, trimethoprim-sulfamethoxazole; HLA, human leukocyte antigen; BMI, body mass index; VAD, vascular assist device; APACHE, Acute Physiology and Chronic Health Evaluation; ICU, intensive care unit; HIV, human immunodeficiency virus; MELD, model for end-stage liver disease.
pleural effusion) (OR: 2.93; 95% CI: 1.56–5.50; \( P = 0.001 \)), and pulmonary edema (OR: 2.79; 95% CI: 1.78–4.37; \( P < 0.001 \)) are also associated with pneumonia (9).

Early nosocomial pneumonia in liver transplant patients has been associated with mortalities as high as 40% (9, 79). Although the impact is significant in the early post transplant period, with a higher in-hospital mortality observed in patients with nosocomial pneumonia than those without (10.6% vs. 1.8%, \( P < 0.005 \)), mortality is more similar between groups at 1 year (21% vs. 13%, \( P = 0.14 \)) (8). Multivariate analysis has determined mechanical ventilation (OR: 3.55, 95% CI: 2.27–5.56; \( P < 0.001 \)), and the presence of pulmonary edema (OR: 2.32; 95% CI: 1.71–3.16; \( P < 0.001 \)) to be associated with greater mortality. The use of piggyback anastomosis (OR: 0.45; 95% CI: 0.27–0.74; \( P = 0.001 \)) was found to be protective (9). This protective effect is most likely a result of shorter operating times, decreased need for blood transfusion, and decreased intensive care unit (ICU) and hospital length of stay associated with the piggyback technique, which preserves the recipient vena cava and avoids retrocaval dissection (88). Emergency living-donor liver transplant patients who develop pneumonia also have increased mortality compared with patients who undergo the procedure under non-emergent situations and develop pneumonia (11). Pneumonia resulted in greater durations of mechanical ventilation (9 vs. 2 days, \( P < 0.01 \)) in addition to longer ICU (21.9 ± 31.2 vs. 7.8 ± 4.5 days, \( P < 0.001 \)) and hospital (44.5 ± 29.5 vs. 29.9 ± 16.7 days, \( P < 0.001 \)) lengths of stay (7, 8).

**Pneumonia in kidney transplantation**

Incident rates of early nosocomial pneumonia in the kidney transplant recipient range from 4.5% to 16% (13, 28, 89). Most common bacterial etiologies of pneumonia in kidney transplant patients include *S. aureus*, *P. aeruginosa*, Acinetobacter species, and *H. influenzae* (13–15). One study found 38% of pneumonia cases in kidney recipients to be nosocomial, with 10% of the pneumonia cases occurring in the first month after transplantation (13). Nosocomial pneumonia in kidney transplant recipients has a mortality of 35% (13).

**Pneumonia in heart transplantation**

The incident rate of pneumonia in heart transplant recipients ranges from 20% to 35% (16, 17, 90, 91). *S. aureus, P. aeruginosa*, Acinetobacter baumannii, and *Enterobacter cloacae* are the most commonly isolated pathogens (17, 18). Nosocomial pneumonia is diagnosed a mean of 20 days after heart transplantation (16). Nosocomial pneumonia in heart transplant recipients is associated with a mortality of 26–55.6% (16, 90), increasing in patients who require ventilation (16).

**Pneumonia in lung transplantation**

With prevalence rates as high as 60%, lung transplant recipients are at the greatest risk of developing pulmonary infections during the first month post transplant (4, 19–21). In one study, 30.7% of lung transplant recipients, 9.8% of heart transplant recipients, and 40% of combined heart/lung transplant recipients developed nosocomial pneumonia (20). Bilateral lung recipients are at greater risk of nosocomial infection than single lung recipients (20). Lung volume reduction (OR: 2.6, 95% CI: 1.13–6.3, \( P = 0.05 \)) to adjust for recipient/donor organ size mismatch, and re-do transplantations (OR: 5.25, 95% CI: 1.41–26.8, \( P = 0.04 \)) were identified as risk factors for nosocomial infection in lung transplant recipients on multivariate analysis (20). Preoperative colonization with gram-negative rods was also identified as an independent risk factor by multivariate analysis for the development of pneumonia in lung transplant patients (OR: 3.69, CI 95% 1.19–11.37, \( P = 0.004 \)) (19, 20). The median time of onset of pneumonia in lung transplant recipients is 34 days (0–486 days) (21). The most common causative organisms include *P. aeruginosa*, *S. aureus*, and *Aspergillus* species (19–23).

Bacterial pneumonia, responsible for 56% percent of deaths in the first month after transplantation in one study, remains the greatest cause of mortality in the early postoperative period following lung transplantation (92). Lung recipients who develop pneumonia have significantly reduced 1-year survival (74% vs. 99%, \( P < 0.01 \)) (21). Recipients with cystic fibrosis (CF) are more likely to die following the development of nosocomial infections, including pneumonia (OR: 3.20, 95% CI: 1.27–7.92, \( P = 0.005 \)) (20).

Although infection in the early postoperative period is most frequently nosocomial, recipient-derived infection secondary to preoperative colonization is also seen in this time period (2). The majority of CF patients are colonized with *P. aeruginosa* by the time they reach adulthood (93), and up to 90% of these patients are colonized at the time of lung transplantation (20, 94). The paranasal sinuses have been identified as a reservoir for recolonization after bilateral lung transplantation in CF patients. This patient population experiences greater rates of *Pseudomonas* pneumonia.
following lung transplantation than recipients with other indications for transplant (60.3% vs. 38.2%, \( P < 0.01 \)) (95). CF patients colonized with *Burkholderia cepacia* complex at the time of transplant have a significantly lower median survival than patients who were not (0.19 vs. 7.48 years, \( P = 0.041 \)) (96).

Colonization of the lung allograft with *P. aeruginosa* also increases the risk of bronchiolitis obliterans syndrome (BOS) (97, 98). *De novo* colonization after transplantation was much more strongly associated with BOS than pre-transplant colonization that persisted through the perioperative period (97). Eradication of colonized *P. aeruginosa* through sinus surgery and daily nasal irrigation with saline has been found to reduce recolonization of the allograft and post transplant pneumonias, improve survival, and reduce the incidence of BOS (99, 100).

Colonization of lung transplant recipients with *Aspergillus* species has also been associated with increased rates of BOS, as well as greater mortality following BOS diagnosis, compared with patients who were not colonized with this fungus (101). However, these findings extend beyond the early post transplant period, with the median time of pre-BOS colonization occurring at 90 days post transplant (101).

Previously, fungal infections appeared primarily in the early postoperative period, with a peak incidence between 10 days and 2 months after transplantation (102). With improved antifungal prophylaxis, these infections now occur later, with the median time of onset of invasive candidiasis 66 days after transplantation, and the majority of invasive *Aspergillus* infections occurring beyond the first postoperative year (55). *Aspergillus* remains the most common fungal infection in transplant recipients (55). Aspergillosis rarely occurs in the early postoperative period, but 6% of recipients receive this diagnosis at some point in their postoperative course, which is significant when balanced with the 52% mortality associated with *Aspergillus* infection (103). Risk factors for *Aspergillus* infection include increased donor age (OR: 1.40 per decade, 95% CI: 1.10–1.80), ischemia time (OR: 1.17 per hour increase, 95% CI: 1.01–1.39), and use of daclizumab instead of lymphocyte-depleting antibodies (OR: 2.05, 95% CI: 1.14–3.75) (24).

**UTIs**

Diagnosis of UTI in adults requires the presence of at least 1 UTI-related symptom (dysuria, increased urinary frequency, or suprapubic tenderness) and a positive urine culture, or the presence of at least 2 UTI-related symptoms and a urinalysis that suggests infection (74). The incidence rate of UTI in adult surgical and medical ICUs in the general population is 1.3 infections per 1000 catheter-days (27). The incidence rises in pediatric ICUs to between 2.2 and 3.9 UTIs per 1000 catheter-days (27). The predominant causative pathogens in the ICU setting are enteric gram-negative bacteria, *Candida* (18%), *Enterococcus* species (10%), and *P. aeruginosa* (9%) (27). Risk factors include female gender, age >50 years, diabetes mellitus, serum creatinine >2 mg/dL, and prolonged Foley catheter placement (27). UTIs have a low direct mortality, but complications – predominately bacteremia – have a higher case fatality rate (32.8%) (27). The cost per UTI episode is estimated to be $600 without bacteremia and $2800 per episode with bacteremia (27).

**UTI in liver transplantation**

Between 7.4% and 14.1% of liver transplant recipients are diagnosed with a symptomatic UTI in the first month after transplantation (25, 81). The most common causative pathogens are gram-negative bacteria (62.4%), including *Escherichia coli*, *E. cloacae*, and *A. baumannii* (25). Eighty percent of enterococci are highly aminoglycoside resistant and 17.6% are vancomycin resistant.

UTIs appear to be more common in the pediatric liver-transplant recipient population, with up to 38.5% of patients developing this infection within the first 30 days after surgery (26). The primary causative pathogens in pediatric patients are similar to those in the adult group (26). Pediatric recipients with UTIs tend to be younger (2.75 years vs. 9.75 years, \( P < 0.05 \)), and are more likely to have biliary atresia than recipients without UTI (\( P < 0.05 \)) (26).

**UTI in kidney transplantation**

The most common infection of kidney transplant recipients is UTI. The existing literature variably differentiates between symptomatic and asymptomatic UTI, which limits the available data slightly. These infections require prompt intervention, as they are responsible for up to 60% of bacteremias in kidney transplant recipients (28). Early postoperative UTIs are thought to be largely preventable, as they are considered to be caused most frequently by contamination of the catheter at the time of insertion (104). The incidence of UTI in this population ranges from 35% to 79%, with 22.7–56.7% of patients developing UTIs within the first month (28–33, 105–108). In patients...
with UTIs, 48% are symptomatic, 40% are asymptomatic, and an additional 12% develop urosepsis (32). Median time of onset of the first UTI within the first 3 months after transplantation is 19 days (34). The median time to onset of infection after transplantation, in patients with indwelling catheters, is 5 days (28).

*E. coli, K. pneumoniae,* and *Enterococcus* species are the most common causative pathogens (32, 33). Over 87% of enterococci were highly resistant to either aminoglycoside or vancomycin (33). Highly resistant gram-negative rods, including those that produce extended-spectrum beta-lactamase (ESBL), account for 22–33% of infections in more contemporary studies (28, 32). An increase in frequency of gram-positive and fungal UTIs is seen in more current studies as well (33).

Several risk factors for nosocomial UTI have been identified, including duration of bladder catheterization, especially when >7 days, use of ureteral stents, delayed graft function, age (per decade increase beyond a median of 53), and changes in initial immunosuppression regimen (28, 34). Additional risk factors include glomerulonephritis (OR: 2.075, 95% CI: 1.001–4.302, \( P = 0.0497 \)) and simultaneous double renal transplant (OR: 4.011, 95% CI: 1.68–13.775, \( P = 0.0273 \)) (31). Female gender is the strongest risk factor for UTI (OR: 4.397, 95% CI: 2.307–8.379, \( P = 0.0001 \)) (29–31, 34). Receiving a kidney from a deceased donor may also increase the risk of early UTIs (OR: 3.64; 95% CI: 1.0–12.7; \( P = 0.043 \)) (28). Trimethoprim-sulfamethoxazole prophylaxis is associated with a reduced risk of acquiring a UTI (hazard ratio [HR]: 0.55, 95% CI: 0.34–0.89, \( P = 0.02 \)) (34). Likewise, early urinary catheter removal is associated with a reduced risk of development of UTIs (8% when removed within 36 h vs. 74% when removed later, on average 8.2 days postoperatively) (109).

Kidney recipients with UTIs more often lose their graft than recipients who do not develop UTIs (16.1% vs. 6.3%, \( P = 0.08 \)) (29). Untreated UTI, but not treated UTI, was a risk factor for biopsy-confirmed acute cellular rejection (HR: 2.80, 95% CI: 1.27–6.20, \( P = 0.01 \)) (34).

**SSIs**

SSIs are early postoperative infections affecting the region of the surgical incision. Standard CDC definitions further categorize SSIs as superficial, deep, and with organ involvement (74). Superficial infections demonstrate only skin and subcutaneous involvement, and deep infections affect deeper tissue layers; diagnosis of an organ/space infection requires tissue that is manipulated during the surgical procedure other than the skin, fascia, or muscle layers to be infected (74). Diagnosis of an SSI requires a positive culture from the surgical site, a purulent exudate from a surgical wound, or a surgical incision that requires reopening. The infection must occur within the first 30 postoperative days, or in the case of deep and organ/space infections, after the placement of implanted device, within 90 days after surgery (74).

Each year >500,000 SSIs occur, with a significant impact on patient morbidity, mortality, and healthcare cost (40). SSIs in the general surgical population are associated with longer ICU stays, 5 times the rate of hospital readmission, and twice the mortality (110). SSIs also result in a median excess cost of $40,559 per infected patient (111). In the general surgical population, increased age, diabetes, smoking history, obesity, and increased operative time have been found to increase the risk of SSI (40, 41). Although the risk of infections at the surgical site may be impossible to eliminate, improvements are feasible, with 40–60% of SSIs in the general surgical population considered to be preventable (112).

**SSI in liver transplantation**

Owing to the complexity of the procedure and the potential for contamination, liver transplantation is associated with one of the highest rates of SSIs (113). Among nontransplant hepatic surgery, the extent of resection is predictive of the incidence of SSI, ranging from 9.7% in segmental liver resection patients to 18.3% in trisegmentectomy patients. Independent risk factors for SSI in patients undergoing liver resection include preoperative open wound, hypernatremia, hypoalbuminemia, elevated serum bilirubin, dialysis, and longer operative time (114). Surgical technique has been found to influence the risk of acquiring an SSI in the liver transplant recipient population, with rates varying from 7% to 27%; much of this variability appears to be associated with the operative surgeon (115). While rates correlate with a specific surgeon, the rates are independent of surgical experience, defined by the number of liver transplant surgeries performed (115).

The primary pathogens that cause SSI in adult liver transplant patients are *Enterococcus* species, *Staphylococcus* species, *Candida* species, and gram-negative bacteria (35). Frequently, the bacteria are resistant to first-line antibiotics, including methicillin-resistant coagulase-negative staphylococci (CONS), MRSA, and ampicillin- and vancomycin-resistant enterococci (36). Epidemiological data show an incidence of SSI in adult liver transplant recipients ranging from 18% to 37%, with 65–91% of those infections involving the organ and its
direct surrounding (37, 38, 116). Among pediatric liver transplant patients, one study estimates the incidence of SSI at 32.5%, with 67.5% involving the organ or surrounding spaces (39). SSIs typically present with induration, erythema, tenderness, and drainage from the incision. Peritonitis is of particular concern and is frequently associated with bacteremia (117).

SSIs have a significant clinical and economic impact on patients. Data on the impact of SSI on liver recipient morality have been conflicting, with one study demonstrating a significant increase in combined risk of patient mortality or graft loss at 1 year post transplantation (relative risk [RR] = 3.06; 95% CI: 1.66–5.64; P < 0.001), but another study failing to demonstrate a difference in patient or graft survival (37, 38). Nonetheless, SSIs have consistently been demonstrated to result in a longer hospital and ICU lengths of stay: 27 vs. 21 days (P = 0.001) and 10 vs. 8 days (P < 0.001), respectively (38, 118). The increase in hospital stay, together with the requirement of increased medical intervention, translates to a greater cost of care with pediatric liver transplant recipients, who incur an average additional cost of $132,507 (95% CI: $12,908–$252,106) because of SSIs (39). A cohort of primarily adult recipients was found to incur similar charges, with SSIs costing an additional $131,276 (118).

Risk factors for the development of SSI in liver transplant recipients include a 1-h increase in operative time beyond the average operative time of 3.5 h (RR = 1.19; 95% CI: 1.03–1.37; P = 0.018), surgery lasting >7 h (OR: 14.88; 95% CI: 1.84–120.40; P = 0.011), antibiotic therapy in the 3 months preceding liver transplantation (OR: 2.02; CI 95% 1.00–4.07; P = 0.049), leak at the biliary anastomosis (OR: 114.64; 95% CI: 4.94–999.0; P = 0.005), female gender (OR: 7.77; 95% CI: 1.12–54.05; P = 0.038), human leukocyte antigen mismatches (OR: 6.23; 95% CI: 1.20–32.37 P = 0.03), elevated preoperative white blood cell count (OR: 1.28; 95% CI: 1.06–1.54; P = 0.009), and a donor liver mass-to-recipient body mass ratio of <0.01 (RR = 2.56; 95% CI: 1.17–5.62; P = 0.019) (37–39).

**SSI in kidney transplantation**

The incidence of SSIs in kidney transplant patients varies from 7.25% to 18.5% (42–44). These infections are most commonly superficial (58–73.1% superficial, 12.5–31.5% deep incisional, and 10.5–14.4% organ/space) (42–44). The most frequently isolated pathogens are gram-positive bacteria, staphylococci, and enterococci (42, 43), which are often resistant to first-line antimicrobials (70% of staphylococci was MRSA, and 46% of enterococci were resistant to either vancomycin or aminoglycoside) (43).

Pre-transplant diabetes mellitus, delayed graft function, a high body mass index, pre-transplant glomerulonephritis, acute graft rejection, and need for reoperation early post transplant have each been implicated as independent risk factors for the development of SSIs in kidney transplant recipients (42–44). A maintenance regimen containing sirolimus was associated with a higher SSI rate (OR: 3.21; CI 95% 1.26–8.21) compared with a group that was prescribed a mycophenolate mofetil regimen (45). Live donation may be associated with a lower risk of SSI, although the data are not consistent across studies. One study found a lower odds of developing an SSI with a living donor (OR: 0.642, CI 95% 0.424–0.972) (44), while another found no significant difference in risk of acquiring an SSI, although the investigators did observe an overall reduced risk of nosocomial infection (28).

**SSI in heart transplantation**

SSI following heart transplantation is associated with significant morbidity and mortality (119). In general, nontransplant cardiac surgery, the incidence SSI is typically <5%, ranging between 0.5% and 10% (46). Using CDC classifications, the proportions of SSIs by type are up to 8% superficial, 2% deep, and <2% of patients develop mediastinitis. Sternal wound infections in these patients are associated with mortality up to 35%, and sternal wound dehiscence has been documented between 3% and 8%, nearly all of which are caused by infection (46). SSIs in the heart transplant population commonly present with pain out of proportion to sternotomy and elevated white blood cell counts. Positive culture of temporary cardiac pacing wires was also found to be an effective means of diagnosing mediastinitis (120).

The incidence of surgical wound complications in heart transplant recipients has been measured to be much higher than in patients undergoing other types of heart surgery: as high as 40%, with most reports placing the range between 8% and 15%. The subtypes are 3.9–16% superficial and 2.4–35% deep, including mediastinitis. Sternal dehiscence occurs in the heart transplant population at an incident rate ranging from 12.5% to 25%, far exceeding the rate in the general heart surgery population (46–48, 90, 120).

The majority of SSIs in heart transplant patients, as in other cardiac surgery patients, are caused by gram-positive bacteria, particularly MRSA. However, higher incidences of fungal pathogens have been measured in heart transplant recipients compared with
general cardiac surgical patients (46). One study found the etiology and type of infection to vary with postoperative time. The earliest infections, appearing within the first week, were caused by *Candida* species, while those appearing >2 weeks after transplantation were primarily bacterial. Infections in the first 2 weeks were predominantly at the sternum, as opposed to in the mediastinum or on the skin (49).

A number of risk factors for post-heart transplant SSIs have been documented, including body mass index >30 kg/m² (*P* = 0.02), previous heart surgery (*P* = 0.03), previous ventricular assist device implantation (*P* = 0.006), inotropic support (*P* = 0.04), and increased age (OR: 1.08, 95% CI: 1.05–1.1) (47, 48). Immunosuppression regimens that include sirolimus, compared with mycophenolate mofetil controls, have been identified in several studies as a risk factor for SSI, particularly mediastinitis and deep incisional infections (50, 51). Tacrolimus has been associated with increased rates of infection in heart transplant recipients (52), and the presence of a ventricular assist device has also been established as a risk factor, particularly for mediastinitis (48, 50). Prolonged cardiopulmonary bypass time has also been associated with increased incidence of SSI in heart transplant recipients on multivariate analysis (OR: 1.011; 95% CI: 1.000–1.022) (51).

Heart transplant recipients who develop deep sternal wound infections have increased in-hospital mortality compared with patients without infection (31 vs. 8%; *P* = 0.03) (48). Following successful recovery, however, 5-year survival is similar for both groups (47, 48).

**SSI in lung transplantation**

The literature about SSIs in lung transplant recipients is particularly sparse. A single retrospective study describes the topic (54). Of the 586 recipients enrolled, 5% developed SSIs. Organized by type, 42% of the SSIs were empyema, 29% surgical wound infection, 16% mediastinitis, 6% sternal osteomyelitis, and 6% pericarditis. Bacteria were the predominant causative pathogen (82%), roughly equal proportions gram-negative and gram-positive, and fungi were responsible for 10%. The most common pathogens were *S. aureus*, *P. aeruginosa*, and *Enterococcus faecium*. Antibiotic resistance was common, with the majority of gram-negative pathogens exhibiting resistance to multiple classes of antibiotics, and 40% of staphylococcal isolates were MRSA. Ischemic time (OR: 1.005, 95% CI: 1.001–1.009, *P* = 0.04), number of red blood cell units (OR: 1.04, 95% CI: 1.02–1.06, *P* < 0.0001), female donor (OR: 2.40, 95% CI: 1.21–6.50, *P* = 0.02), diabetes (OR: 3.03, 95% CI: 1.32–6.98, *P* = 0.009), and prior cardiothoracic surgery (OR: 4.15, 95% CI: 1.79–9.62, *P* = 0.001) were found to be independent risk factors for SSI by multivariate analysis. The length of hospital stay was significantly increased in patients with SSIs (61 vs. 26, *P* < 0.001). Mortality at 6 months and 1 year was also greater in recipients with SSIs (P = 0.01 and *P* = 0.002, respectively). Early mortality of patients with SSIs was especially high: 16% at 30 days post transplant.

**BSIs**

BSIs have been associated with significant morbidity and mortality in both the general surgical population and in transplant recipients. Risk factors identified for BSI in the general patient population include red blood cell transfusion, parenteral nutrition, age, immunosuppression, and medical comorbidities, including diabetes, peripheral vascular disease, congestive heart failure, and renal and liver disease (63). Overall mortality following BSI diagnosis is approximately 35% (63). The reported overall incidence of BSI in the first 30 days after transplantation ranges from 7.4% to 14% (64, 121). The incidence by type of organ transplanted is 4.8% in kidney, 4.5% in simultaneous pancreas-kidney, and 12% in liver transplantation (64). Among transplant recipients, gram-positive bacteria, particularly *S. aureus* and CONS, are most frequently isolated, although gram-negatives are more frequent among kidney and liver transplant recipients (64, 122). Antibiotic resistance is common, with 54% of *E. coli* producing ESBL and 31% of *P. aeruginosa* being multidrug resistant in more contemporary studies (64).

Bacteremia in transplant patients has been found to be hospital-acquired in up to 72% of cases. Septic shock occurs more frequently in liver transplant recipients compared with renal transplant patients (23% vs. 6% of bacteremia). Independent risk factors for septic shock on multivariate analysis include age (OR: 2.02; 95% CI: 1.11–3.69; *P* = 0.021), nosocomial infection (OR: 2.18; 95% CI: 1.15–4.11; *P* = 0.01), and pulmonary source (OR: 4.36; 95% CI: 1.97–9.61; *P* = 0.001) (104). Mortality from BSIs has been reported to be as high as 54.5%, with the highest rates among lung and liver transplant recipients (121, 122).

**BSI in liver transplantation**

The incidence of BSI in liver transplant recipients ranges from 10% to 39% (56, 57, 64, 122), with lower incidences reported in the more recent literature. More
than half of all bacteremia in this population occurs within the first month (56, 81, 122).

The most common bacteria isolated include *S. aureus* and CONS (56–58); however gram-negative bacteria, including large proportions of ESBL-producing *E. coli* and multidrug-resistant *P. aeruginosa*, have also been reported (64). The overall incidence of *S. aureus* bacteremia declined from 12% in the 1990s (56) to 5% in the 2000s (122). The principle sources of the bacteremia in liver transplant recipients include intravascular catheters (23–31%), abdominal or biliary (7–33.6%), lung (6–24%), urinary tract (1.3–11%), and surgical wounds (1.3–10%) (56, 57, 64, 122).

The literature available on pediatric liver transplant recipients shows an epidemiology similar to the adult population. The incidence rate of BSI is 21.5% in the first 30 days after transplantation with the predominant organisms being CONS (30.6%) and *K. pneumoniae* (22.5%). The most common source of the bacteremia is intravascular catheters (38.9%), followed by intra-abdominal infection (11.1%) (59).

Epidemiology of candidiasis is changing over time. Historically, the incidence of candidemia in liver transplant recipients has been as high as 42%, and has occurred early in the first postoperative month (123, 124). More recent literature suggests an incidence of invasive fungal infection in liver transplant recipients between 12% and 17.7% (60, 125, 126), with most cases occurring during the first postoperative month (60, 61, 125). A recent study of 429 SOT recipients found a median day of onset of invasive candidiasis in liver transplant recipients of 151.5 days after transplantation, with only 45.8% of cases occurring in the first 100 days (55). Likewise, non-albicans *Candida* species have become the most common fungal pathogen in all transplant recipients, with a larger proportion of isolates (up to 57%) being fluconazole resistant (55, 60). *Candida* species now account for as much as 80% of invasive fungal infections in liver transplant recipients (55, 127).

Predictors of BSIs among adult liver transplant recipients include diabetes mellitus (OR: 6.9; 95% CI: 1.2–40.6; \( P = 0.03 \)) and serum albumin levels >3.0 mg/dL (OR: 0.14; 95% CI: 0.03–0.75; \( P = 0.02 \)) (57), while age ≤1 year (OR: 3.90; 95% CI: 1.83–15.26; \( P < 0.05 \)) and bile duct complications (OR: 6.2; 95% CI: 3.21–35.23; \( P < 0.05 \)) are risk factors for pediatric liver transplant recipients (59). Risk factors for candidemia among liver transplant recipients include use of fluoroquinolone prophylaxis for spontaneous bacterial peritonitis (OR: 8.3, 95% CI: 2.1–32.5, \( P = 0.0024 \)), re-transplantation (OR: 16.4, 95% CI: 2.8–94.7, \( P = 0.0018 \)), post transplant dialysis (OR: 7.6, 95% CI: 2.3–24.9, \( P = 0.0009 \)), colonization with *Candida* species before transplantation (OR: 7.8; 95% CI: 3.9–16.2; \( P < 0.001 \)), prolonged or repeat operation, re-transplantation, high transfusion requirement (i.e., transfusion of ≥40 units of cellular blood products including platelets, packed red blood cells, and auto transfusion), and choledocho-jejunostomy (60–62). Most experts have recommended the use of antifungal prophylaxis among patients with ≥2 risk factors for candidemia (62).

The 30-day mortality for BSI is high (21–28%), particularly if *S. aureus* is isolated (RR = 3.13; 95% CI: 1.3–7.5; \( P = 0.01 \)) (56, 57). Invasive candidiasis also has a significant impact on patient outcomes, with an in-hospital mortality of 36.1% for cases compared to 2.8% for controls (OR: 25.0, 95% CI: 6.2–100.5, \( P = 0.0002 \)) (61). In patients with invasive candidiasis, 85% of the deaths occurred within 30 days of infection (61). Non-albicans *Candida* infection was associated with a mortality 58.3%, compared with 22.7% for infection with *Candida albicans* (\( P = 0.04 \)) (61). Diabetes mellitus (OR: 10.93; 95% CI: 1.27–94.02; \( P = 0.02 \)) and diagnosis >30 days after transplantation (OR: 5.87; 95% CI: 1.41–24.40; \( P = 0.01 \)) are risk factors for mortality attributed to candidemia (55).

### BSI in kidney transplantation

BSI within the first 30 days after transplantation complicates the postoperative course of 3.5–4.6% renal transplant procedures (64, 65). Most BSIs occur late and are secondary to a UTI (66). The low rate of use of central venous lines in this patient population likely partially explains this low rate. In the early postoperative period, the most common sources of BSI were urinary tract (38–61%), catheter-related (8.7–18.4%), pneumonia (11.9%), gastrointestinal (11.4%), and SSSs (5.9%) (64, 66). Most early BSIs in kidney transplant recipients are caused by gram-negative bacteria, although *S. aureus* and CONS are more common when the source is a vascular catheter or abdominal infection (64–66).

Risk factors for early BSIs in kidney transplant recipients include acute rejection (46% vs. 20%; \( P < 0.05 \)), hemodialysis prior to transplantation (71% vs. 43%; \( P < 0.05 \)), having a local infection (46% vs. 12%; \( P < 0.05 \)), insertion of a ureteric stent after transplantation (OR: 3.60; 95% CI: 1.50–7.52; \( P = 0.002 \)), and being a recipient of a deceased-donor organ (OR: 3.16; 95% CI: 1.39–7.17; \( P = 0.001 \)) (65, 66). The 30-day mortality of all kidney transplant recipients who develop a BSI is high (14–24.3%) and is increased compared to patients without BSI (65, 66). Risk factors
for fatal BSI include an APACHE II score (128) $\geq$ 20 (OR: 6.39; 95% CI: 2.07–19.73; $P = 0.001$), the presence of shock at diagnosis (OR: 9.87; 95% CI: 2.95–32.94; $P = 0.001$), and respiratory failure (OR: 7.66; 95% CI: 2.56–22.87; $P = 0.001$) (66).

**BSI in heart transplantation**

The incident rate BSIs in heart transplant recipients is 15.8% (67). The origin is predominately the lower respiratory tract (23%), followed by the urinary tract (20%), and intravascular catheters (16%) (67). Causative pathogens are 55.3% gram-negative, primarily *E. coli* and *P. aeruginosa*, and 44.6% gram-positive, mostly *S. aureus* and *Staphylococcus epidermidis* (67).

Three independent risk factors for BSI in this population have been identified: hemodialysis (OR: 6.5; 95% CI: 3.2–13; $P < 0.001$), a prolonged ICU stay (OR: 5.6; 95% CI: 1.6–8.1; $P = 0.002$), and viral infection, typically cytomegalovirus (OR: 2.1; 95% CI: 1.1–4; $P = 0.01$) (67). The mortality of heart transplant recipients with a BSI is as high as 59.2%, with 12% directly due to BSI (a risk factor for mortality: OR: 1.8; 95% CI: 1.2–2.8) (67).

**Clostridium difficile colitis**

*C. difficile* is a gram-positive, spore-forming, anaerobic bacillus that can cause pseudomembranous colitis and represents the most common hospital-acquired infection (72). In 2009, *C. difficile* infection (CDI) was a listed diagnosis in 8.53 per 1000 discharges in the United States, including both adult and pediatric patients (72). Antibiotic exposure and use of proton pump inhibitors are associated with an increased risk of developing *C. difficile* infection (72).

The overall incidence of CDIs in SOT recipients in 2009 was 2.7%, which is significantly higher than in the general hospital population (0.82%). On multivariate analysis, liver transplant recipients (adjusted OR: [aOR]: 1.73; 95% CI: 1.48–2.02; $P < 0.001$) and lung transplant recipients (aOR: 1.60; 95% CI: 1.20–2.14, $P < 0.001$) had the greatest risk of CDI. SOT recipients with CDI have significantly longer hospitalizations (mean difference 9.6 days; 95% CI: 9.3–9.9; $P < 0.001$) and hospital charges (mean difference $69,647; 95% CI: $66,190–$73,104; $P < 0.001$) compared with SOT patients without CDI. CDI in SOT patients was also found to be independently associated with complications of the transplanted organ (aOR: 1.36; 95% CI: 1.28–1.44; $P < 0.001$) and colectomy (aOR: 3.10; 95% CI: 2.35–4.08; $P < 0.001$), although the rate of colectomy was lower among SOT patients than non-SOT patients (aOR: 0.69; 95% CI: 0.53–0.90; $P < 0.001$). Incidence of CDI by organ transplanted: 2.2% in kidney, 2.7% in heart, 2.7% in >1 organ, 3.7% in lung, and 3.8% in liver (68). The median time of onset of CDI is 18–31.5 days after transplantation, with half of patients initially developing diarrhea within the first month after transplantation (69, 70). CDI in SOT patients was identified as an independent risk factor for mortality (aOR: 2.48; 95% CI: 2.22–2.76, $P < 0.001$). Lung transplant recipients had the highest risk for mortality (aOR: 3.02; 95% CI: 2.71–3.36; $P < 0.001$). SOT recipients from the low-income quartile and African-American race were associated with the highest mortality.

Risk factors for CDI include prior use of antibiotics (previous use of first- and second-generation cephalosporins, particularly) (HR: 3.68; 95% CI: 1.8–7.52; $P < 0.001$), ganciclovir prophylactic use (HR: 3.09; 95% CI: 1.44–6.62; $P = 0.004$), and corticosteroid use before transplantation (HR: 2.95; 95% CI: 1.1–7.9; $P = 0.031$) (124). The increased incidence of CDI in lung, liver, and heart transplant recipients may be attributed to the hypogammaglobulinemia commonly seen in these transplant recipients, which may weaken their immune response (129). Severe hypogammaglobulinemia is independently associated with *C. difficile*-associated disease in heart transplant recipients (RR = 5.8; 95% CI: 1.05–32.1; $P = 0.04$) (71). Among liver transplant recipients, re-transplantation (P = 0.013), having a higher model for end-stage liver disease score ($P = 0.007$), bile leak ($P = 0.026$), significant intra-abdominal bleed ($P < 0.001$), a biliary complication ($P = 0.034$), and presence of systemic infection ($P < 0.001$) are all risk factors for CDI (70).

**Discussion**

An understanding of nosocomial infection is critical to the postoperative management of SOT recipients. This knowledge guides key clinical decisions, such as choice of anti-infective prophylaxis or empiric treatment, and in some cases, decisions for organ transplant listing (130). Research into the epidemiology, risk factors, and outcomes of early post transplant infections has made significant advances in the last decade; however, significant gaps in our knowledge remain. The most apparent deficit is the dearth of data on pediatric patients, and on lung and combined organ transplant recipients, some of the most vulnerable transplant patient populations. Importantly, a minority of the studies performed thus far have been prospective.
A call for the development of a national transplant quality improvement program modeled after ACS-NSQIP has been made (131). The impact of ACS-NSQIP on general surgical patient outcomes has been well documented. Notably, the Department of Veterans Affairs decreased their 30-day postoperative mortality from 3.16% down to 1.70% after enrollment in the program (6). National surveillance lends itself well to the study of transplant infectious disease. The paucity of data in many areas of this field is largely a result of the small number of procedures performed each year at any one center. A national cohort will provide the statistical power to adequately describe infections in these transplant populations. Infection surveillance is also important for assessing the changing epidemiology of early post transplant infections. Available data suggest that the frequency of early fungal infections after transplantation has changed (132). Such data will be useful in optimizing outcomes and prophylaxis as our patients, resistant flora, and medical practices change.

Given the documented impact similar data has had on reducing the incidence and impact of early nosocomial infections in general surgical populations and the significant limitation of similar data in the transplant population, it is critical that research be devoted to early nosocomial infections in transplant patients. This research will require collaboration by the key constituencies in the transplant community and external groups, including NSQIP, to collect robust data. As the practice of transplantation is ever changing, with new surgical techniques, novel immunosuppressive approaches, and the maturation of resistant pathogens affecting this high-risk population, such data need to be collected longitudinally. Funding, through federal agencies, such as the National Institutes of Health, the Agency for Healthcare Research and Quality, and agencies, such as the National Institutes of Health, should be made available to support this important research.

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