Review Paper

Dermatology

Dermatology 2017;233:358–365
DOI: 10.1159/000484405

Received: July 2, 2017
Accepted after revision: October 14, 2017
Published online: January 10, 2018

Skin Infections due to Bacteria in Solid Organ Transplant Recipients: A Review

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Keywords

Bacterial skin infections · Organ transplantation · *Staphylococcus* · *Streptococcus* · *Pseudomonas* · *Escherichia coli* · *Nocardia* · *Mycobacteria* · *Bartonella henselae*

Abstract

Though there is an abundance of information on cutaneous malignancies in transplant recipients, cutaneous infections in solid organ transplant recipients (SOTRs) are underrepresented in the dermatological literature. Our paper provides a comprehensive review of bacterial cutaneous infections within the solid organ transplant population. Cutaneous bacterial infections may lead to significant morbidity and even mortality in this immunosuppressed population. Thus, it is to the benefit of both dermatologists and other transplant care providers to better understand and recognize the features of cutaneous bacterial infections in SOTRs. This paper can aid providers in promptly identifying, diagnosing, and treating bacterial skin infections. This review discusses the diagnosis and treatment of the following bacterial species: *Staphylococcus*, *Streptococcus*, *Pseudomonas aeruginosa*, *Escherichia coli*, *Nocardia*, *Mycobacteria*, and *Bartonella henselae*.

Muneeb Ilyas and Nishita Maganty contributed equally to this work.

Introduction

Due to their being on immunosuppressive medications, solid organ transplant recipients (SOTRs) are afflicted with a wide array of cutaneous diseases. Despite the abundant literature on cutaneous malignancies in SOTRs, information on the diagnosis and management of bacterial skin infections is underrepresented. Bacterial infections can present with a variety of symptoms and may cause significant morbidity in SOTRs (Table 1). Therefore, it is beneficial for transplant care providers to recognize the spectrum and management of bacterial cutaneous infections.

Materials and Methods

For further details, see the online supplementary material (see www.karger.com/doi/10.1159/000484405) (Fig. 1).

Infection Types and Treatment

Cutaneous Staphylococcal Infection

Prior to transplantation, *Staphylococcus aureus* colonizes the anterior nares of 67% of transplant candidates [1, 2] compared to 50% of immunocompetent individuals

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Table 1. Sources of bacterial skin infections and their cutaneous manifestations in SOTRs

Bacterial species	Diagnoses
Staphylococcus	Folliculitis Impetigo contagiosa Bullous impetigo Ecthyma Necrotizing fasciitis Staphylococcal scalded skin syndrome MRSA infection
Streptococcus	Impetigo contagiosa Ecthyma Cellulitis
Pseudomonas aeruginosa	Necrotizing fasciitis Ecthyma gangrenosum
Escherichia coli	Necrotizing fasciitis
Nocardia	Nocardiosis
Mycobacteria	Nontuberculous mycobacterial infection Miliary tuberculosis Leprosy
Bartonella	Bacillary angiomatosis

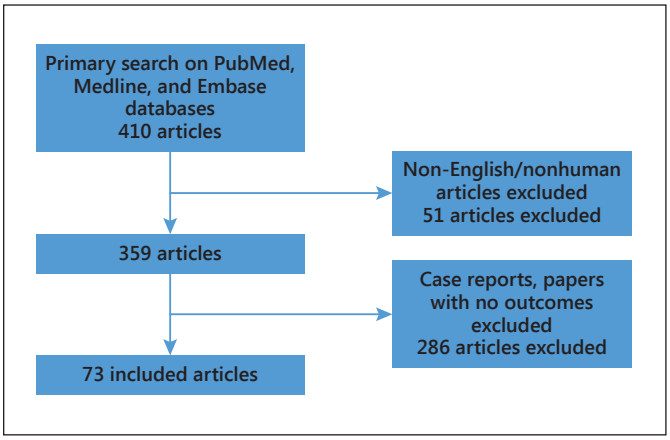

Fig. 1. Flowchart of the literature search.

[3]. In SOTRs, a majority of nasal carriers of *S. aureus* have methicillin-resistant *S. aureus* (MRSA), which if not properly eradicated can lead to bacteremia after transplantation. Folliculitis is an infection of the superficial hair follicle and the most prevalent cutaneous manifestation of *S. aureus* infection in the first 6 months following transplantation [4]. Hogewoning et al. [5] suggest that high-dose immunosuppression predisposes patients to folliculitis due to a diminished immune response. A study [6] found that folliculitis was significantly more prevalent in a group of Egyptian renal transplant recipients than an immunocompetent control group (10.3 vs. 1.7%, respectively). Euvrard et al. [7] found that 6.2% of pediatric SOTRs developed folliculitis. Methicillin-sensitive *S. aureus* (MSSA) folliculitis presents as follicular pustules and papules most commonly on the scalp and face [8]. MRSA folliculitis is more likely to occur on the trunk and scrotum when compared to MSSA [9]. Folliculitis is treated with topical antibiotics such as mupirocin or clindamycin and antibacterial washes [10]. If MSSA folliculitis is widespread, a 7- to 10-day course of oral dicloxacillin 500 mg q.i.d. or oral cephalexin 500 mg q.i.d. may be pursued [8]. If MRSA is cultured, a 7- to 14-day course of oral clindamycin 450 mg q.i.d. or oral doxycycline 100 mg b.i.d. can be used to resolve symptoms [9].

Impetigo contagiosa, or nonbullous impetigo, is a skin infection associated with species of *Staphylococcus* or *Streptococcus*. Patients present with thin-walled vesicles and pustules that later rupture. The residual exudate leaves a classic golden crust [11]. In contrast to impetigo contagiosa, bullous impetigo is associated strictly with staphylococcal infection, particularly strains that produce exfoliative toxin A, which targets the protein desmoglein 1 [12]. Bullous impetigo presents with vesicles that rapidly progress to sharply demarcated, serous or yellow fluid-filled superficial bullae. These bullae easily denude and leave behind a crusted erosion. Hogewoning et al. [5] found that 6.7% of renal transplant recipients developed impetigo of some form. Topical antibiotics, such as mupirocin ointment applied 3 times a day for 5 days, is the preferred treatment for limited impetigo [13], but widespread disease is best managed with systemic antibiotics [14]. Oral dicloxacillin 500 mg q.i.d. or oral cephalexin 500 mg q.i.d. for 7 days are the recommended treatment options for extensive impetigo [15]. If impetigo is suspected to be caused by MRSA, a 14-day course of oral clindamycin 450 mg q.i.d. or oral doxycycline 100 mg b.i.d. should be used [9].

Ecthyma is an ulcerative cutaneous infection commonly seen on the shins or dorsal feet. It initially presents as a painless macule that becomes painful within 24 h and develops into a hemorrhagic vesicle. These vesicles then rupture, leaving behind ulceration with a central black necrotic eschar. The most common causes of ecthyma in SOTRs are staphylococcal and streptococcal species. Ecthyma is treated with systemic antibiotics including oral

dicloxacillin 500 mg q.i.d. or oral cephalexin 500 mg q.i.d. for 7 days [15].

Staphylococcal species can also cause necrotizing fasciitis (NF). NF is a devastating cutaneous infection, especially so in the transplant population. NF spreads through the superficial fascia, subcutaneous fat, and deep fascia. It is most commonly seen in the extremities and abdominal wall [16]. NF is associated with a mortality rate between 25 and 30% and is diagnosed in 0.04 cases per 1,000 person-years within the general population [17]. Along with immunosuppression, other risk factors for NF include older age, diabetes, atherosclerosis, and drug or alcohol addiction [18]. Although commonly due to polymicrobial infection, staphylococcal species may be implicated [19]. Transplant recipients with NF are also much more likely to present with shock (50%) when compared to a control group (20.2%) [17]. Treatment of NF requires emergent surgical debridement along with broad-spectrum antimicrobial therapy with the addition of either intravenous vancomycin at 15–20 mg/kg/dose every 8–12 h or intravenous daptomycin 4 mg/kg once daily to cover for MRSA [20]. In severe cases, amputation may need to be considered to prevent mortality [18, 19].

S. aureus is capable of releasing epidermolytic toxins (ETA and ETB), which can cause a potentially fatal condition called staphylococcal scalded skin syndrome (SSSS) [21–23]. Clinically, SSSS develops as a scalantiform eruption with flaccid blister formation and a positive Nikolsky sign (Fig. 2a, b). It is seen particularly in the periorificial and flexural areas, although diffuse cutaneous involvement with large regions of epidermal shedding may be seen [24]. Other findings include fever, facial edema, fissures on the lips, and purulent conjunctivitis [25]. SSSS is normally seen in infants or young children and tends to self-resolve in these populations. However, in SOTRs, multisystem failure and electrolyte disturbance associated with SSSS can be potentially fatal [26]. The diagnosis is confirmed with biopsy or with a frozen section of a skin sample [26]. A Gram stain may be negative because the progression of the condition is due to toxin and not the presence of the bacteria itself [25]. Treatment is initiated with a parenteral penicillinase-resistant antistaphylococcal antibiotic such as intravenous flucloxacillin 500 mg per day, divided into 4 portions [27]. If the patient fails to improve following antistaphylococcal antibiotic therapy, MRSA should be considered as a possible source of infection and switching treatment to vancomycin may be considered [28]. Additionally, supportive skin care, intravenous fluids, and admission to the ICU or burn unit may be necessary depending on disease severity.

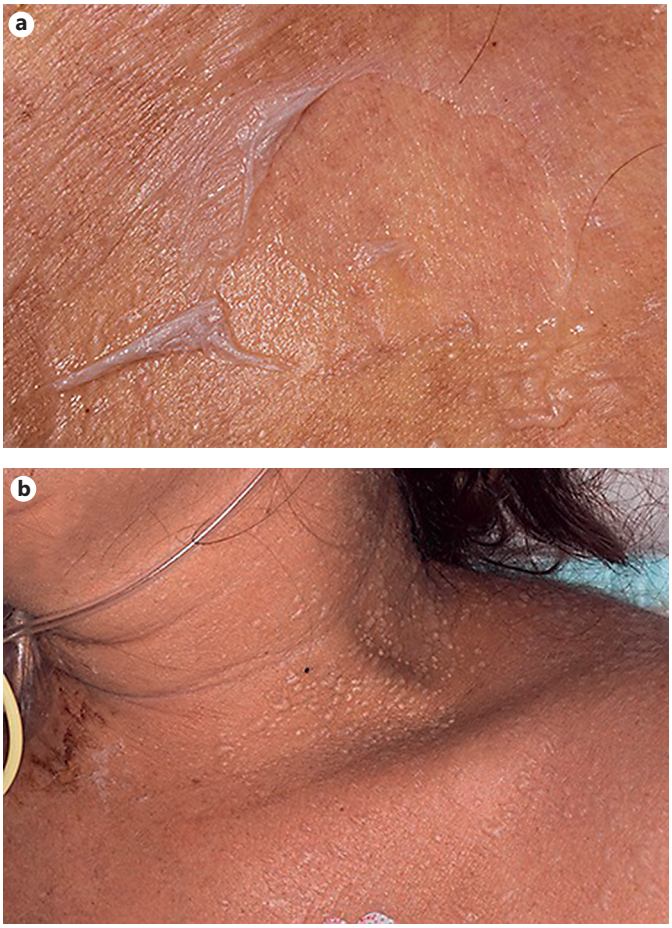

Fig. 2. a, **b** SSSS presenting with fragile bullae, desquamation, and a positive Nikolsky sign.

In recent years, there has been increasing evidence of community-acquired MRSA skin infections affecting posttransplant patients [29–31]. In 1 study [30], 3 of 11 (27%) patients with skin or wound infection after lung transplantation were infected with MRSA. Four of 11 (36%) were infected with MSSA, showing that *S. aureus* alone was responsible for 63% of skin or wound infections in lung transplant recipients [30]. Community-acquired MRSA infections are generally susceptible to an array of broad-spectrum antibiotics when compared to hospital-acquired MRSA [30]. A large study [31] on liver transplant recipients found that nasal colonization by *S. aureus* was associated with an increased likelihood of MRSA infection, and patients who developed MRSA infection had twice the risk of death. These findings suggest that SOTRs should be evaluated and treated for nasal MRSA colonization, as this may lower mortality rates in transplant recipients [31, 32]. Regardless of MRSA infection

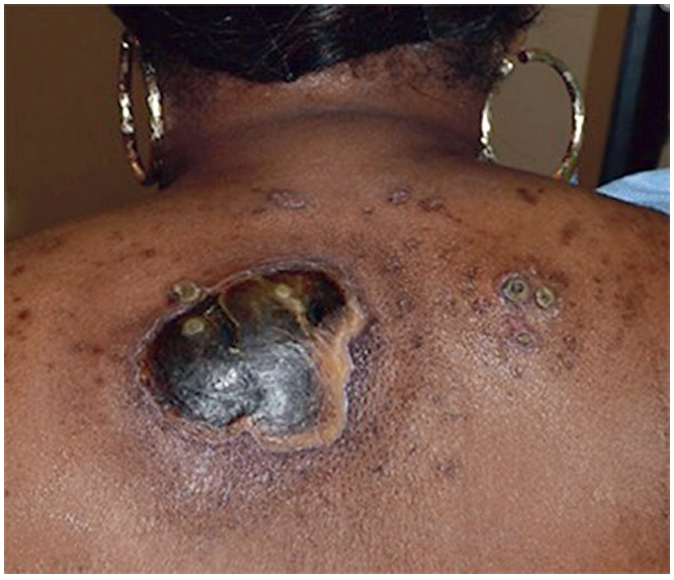

Fig. 3. Ecthyma gangrenosum presenting as a large ulceration with a black necrotic eschar. Reprinted with permission from Frey et al. [74]. Copyright 2014 by the Korean Society of Plastic and Reconstructive Surgeons.

type, sensitivity testing must be conducted owing to potentially large differences in antibiotic resistance between MRSA strains [29].

Cutaneous Streptococcal Infections

In SOTRs, streptococcal species are associated with causing a variety of skin infections, such as impetigo contagiosa [5], ecthyma, and NF [33]. *Streptococcus pyogenes* is the most common pathogen to cause monomicrobial NF [17], though *Streptococcus pneumoniae* has also caused disease in renal transplant recipients [34]. The management of these infections is similar whether infection is due to staphylococcal or streptococcal species.

Cutaneous Pseudomonal Infections

A severer variant, commonly regarded as a sign of underlying septicemia, is ecthyma gangrenosum (EG). The most common infection associated with EG is *Pseudomonas aeruginosa* [35]. EG lesions are described as gunmetal gray tense pustules that later evolve into round ulcerations with necrotic black eschars and surrounding erythema (Fig. 3) [35].

Nakai et al. [35] reported a case of EG without associated septicemia in a renal transplant recipient. Even without evidenced bacteremia, the patient showed progression of EG with numerous ulcerations and rapid spreading. This case was of particular interest given the presence of both *P. aeruginosa* and MRSA within the wound but the absence of both in the blood. It is imperative to make an early diagnosis and begin treatment promptly for suspected EG in the SOTRs [36]. Even without associated sepsis, immunosuppressed patients can rapidly develop life-threatening disease [35]. EG is treated using antipseudomonal monotherapy with intravenous ticarcillin-clavulanate 3.1 g/4 h (not currently available in the USA or Canada) or intravenous piperacillin-tazobactam 4.5 g/6 h [37]. Although controversial, dual therapy with an antipseudomonal penicillin and an aminoglycoside or other antipseudomonal is recommended in SOTRs [38].

In the renal transplant population, 1 study [39] found that the majority of NF infections (36.4%) were fungal, likely due to the iatrogenic immunosuppression. *P. aeruginosa* and *Escherichia coli* are other infectious causes linked with NF development in the renal transplant population [17]. There has also been a reported case of fatal NF due to carbapenem-resistant *Acinetobacter baumannii* in a transplant recipient [40].

Cutaneous Escherichia coli *Infections*

In SOTRs, 2 cases [39, 41] of NF due to *E. coli* have been described. NF due to *E. coli* is managed with surgical debridement along with dual therapy with ciprofloxacin and imipenem for antimicrobial therapy [39].

Cutaneous Nocardial Infections

Nocardial infection in the immunocompromised host can lead to localized infection or hematogenous spread, causing disseminated nocardiosis. Localized infection can include ulcers, abscess formation, granulomas, soft tissue infection, and lymphocutaneous infection. In SOTRs, disseminated disease with organ involvement has been described [42]. Disseminated nocardiosis may present with pustules, abscesses, or subcutaneous nodules (Fig. 4) [43]. For diagnosis, both microbiological and histological testing on biopsy specimens should be performed with attempts to culture *Nocardia*. Depending on infection severity, the immunosuppressive regimen may need to be modified, and combination therapy with imipenem along with either trimethoprim-sulfamethoxazole, amikacin, or linezolid should be started [44]. A study [45] found that SOTRs with nocardiasis treated for a median duration of 56 days had a 1-year cure rate of 88%.

Cutaneous Mycobacterial Infections

Cutaneous mycobacterial infection in SOTRs is most commonly due to nontuberculous mycobacteria (*Myco-*

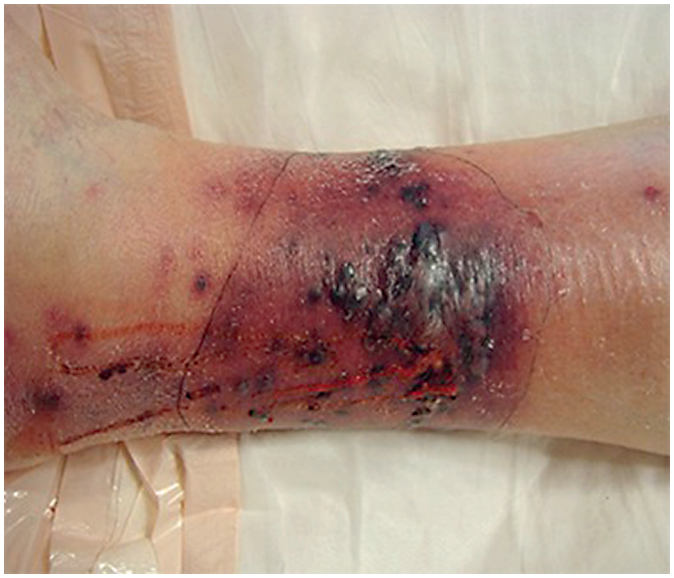

Fig. 4. Disseminated nocardiosis presenting as an indurated, erythematous plaque on the lower extremity. Reprinted with permission from Drone et al. [75]. Copyright 2014 by the *Indian Dermatology Online Journal*.

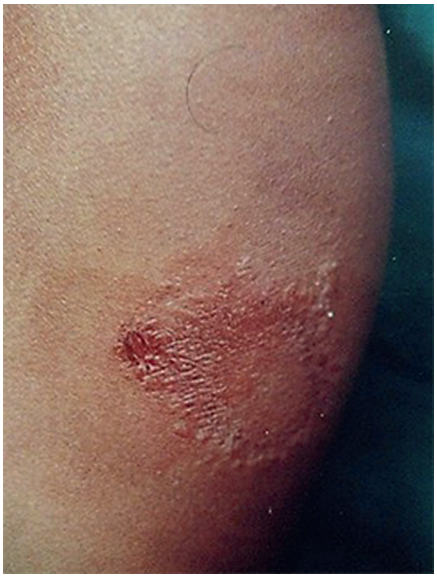

Fig. 5. Tuberculoid leprosy presenting as an annular, anesthetic plaque on the forearm. Reprinted with permission from Thakkar and Patel [76].

bacterium marinum, M. haemophilum, M. fortuitum, M. chelonae, M. abscessus and *M. ulcerans,* or *M. immunogenum*), although 2 cases of hematogenous dissemination of *M. tuberculosis* leading to cutaneous tuberculosis have been described [46].

Cutaneous manifestations of mycobacterial infections range from macular erythema to nonhealing ulcers, although erythematous nodules and papules are the most common presentations [46]. In kidney transplant recipients, cutaneous nontuberculous mycobacteria may present with painless, violaceous nodules that can ulcerate and manifest with a sporotrichoid appearance [45].

Hematogenous spread of mycobacterial infection could present as nodules or abscesses. Disseminated disease in transplant recipients is most commonly caused by *M. chelonae*, presenting with multiple red subcutaneous nodules or abscesses [47]. Additional nontuberculous mycobacteria associated with disseminated infection include *M. kansasii*, *M. haemophilum*, *M. fortuitum*, and others [48]. Other presentations include lupus vulgaris, which presents with small, sharply defined red papules with gelatinous consistency, or acute miliary tuberculosis, which presents with millet-sized red papules that may progress into ulcers and abscesses. Presentations with subcutaneous abscesses, cellulitis, erysipelas, or pseudotumors have also been described [46, 49, 50].

There are cases of *M. leprae* causing lepromatous leprosy in SOTRs [51–53]. Infection with *M. leprae* in the SOTR population presents with a variety of cutaneous manifestations (Fig. 5). Currently, there are 16 reported cases of lepromatous leprosy in SOTRs. One case [52] in a heart transplant recipient described the presence of a migratory papular red rash over the upper torso along with violaceous papules on the hands. A report on a renal transplant recipient describes development of a hypoesthetic, hypopigmented patch with thickening and tenderness of an adjacent nerve [51]. Another case describes a renal transplant recipient with erythematous papules and nodules over the face and earlobes, scars on the knee, and purulent discharge and impetiginous crust over the hands and feet [54]. Leprosy infections can range from milder tuberculoid to severe lepromatous infections. Immunosuppression in SOTRs is thought to contribute to disease severity as it reduces T-cell function [53]. All reported cases of lepromatous leprosy in SOTRs occurred in endemic areas or in patients originally from endemic regions [51–53, 55–57]. Currently, there are limited data on the effect of leprosy infection on allograft function or on the association between immunosuppression and leprosy infection.

When mycobacterial infection is being considered, a tissue specimen should be cultured for species identifica-

tion and sensitivity. Polymerase chain reaction (PCR) may be used for mycobacterial identification owing to the 12-week period needed to culture mycobacterial species. In the past, PCR only allowed for the differentiation between nontuberculous and tuberculous mycobacteria; However, recent advancements in PCR techniques allow clinicians to distinguish between nontuberculous mycobacterial species [58–61].

Treatment of cutaneous mycobacterial infection depends on the species and sensitivity data. Limited *M. marinum* may be managed with monotherapy using either clarithromycin, doxycycline, minocycline, or trimethoprim-sulfamethoxazole for 3 months. Severe *M. marinum* skin infection is managed using combination therapy with rifampin and ethambutol [47]. *M. ulcerans* is managed with rifampin and streptomycin treatment for 8 weeks along with surgical intervention [47]. Guidelines for treating nontuberculous mycobacteria are not clearly defined; hence, infectious disease consultation is advised in transplant recipients who develop mycobacterial infection. For primary prevention, transplant recipients should avoid fish tank water and fresh fish because exposure to these agents is a risk factor for infection with *M. marinum* and other nontuberculous mycobacterial species [49]. Acupuncture is associated with nontuberculous mycobacterial infections, and SOTRs should be appropriately cautioned [46, 62, 63].

Cutaneous Bartonella henselae *Infections*

Bacillary angiomatosis (BA) is a vasculoproliferative disorder caused by *Bartonella* species [64]. The most common bacterium associated with BA in SOTRs is *B. henselae* [64]. The classic presentation is with smooth, domed red or violaceous lesions that resemble hemangiomas. Lesions can be solitary or widespread and scattered, normally on the face and extremities. Development of cellulitis or of a large flesh-colored indurated mass is also a possible presentation. These lesions are very vascular and bleed with minor trauma. Additional findings include fever, lymphadenopathy, and vascular nodules within organs [64]. Exposure to the bacterium is most commonly through cats. About 25% of cases of BA in SOTRs were in the pediatric or adolescent population, despite only 3–4% of transplant recipients being in this age range [65–69]. BA can be associated with some devastating consequences. There have been documented occurrences of hemophagocytic syndrome and renal graft rejection, both due to BA in renal transplant recipients [65, 70].

BA may have an exceedingly similar presentation to that of Kaposi's sarcoma or pyogenic granuloma. A consequence of this similarity is the potential delay in diagnosis and subsequent treatment. A diagnosis can be made with electron microscopy showing clumped and solitary rods within the intercellular space. An alternative is using the Warthin-Starry stain, but this test may be difficult to interpret [64]. *Bartonella* species are difficult to identify in culture, and a negative result should not rule out disease [71]. Antibiotic therapy with doxycycline 100 mg b.i.d. for 3 months is recommended for BA in HIV patients [71]. Both ciprofloxacin and doxycycline have been used to resolve BA in SOTRs [72, 73].

Conclusion

SOTRs may present with a wide variety of cutaneous findings. Sources of skin pathology in SOTRs include infection, drug toxicity, and malignancy. Bacterial skin infections can vary in presentation, severity, and prognosis. In order to best reduce disease-associated morbidity and mortality, SOTR care providers must promptly identify, diagnose, and treat bacterial skin infections.

Key Message

A comprehensive review of bacterial cutaneous infections in the solid organ transplant population is presented.

Disclosure Statement

The authors have no conflicts of interest to disclose.

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