

Zoonoses in Solid-Organ and Hematopoietic Stem Cell Transplant Recipients

Camille N. Kotton

Transplant and Immunocompromised Host Section, Infectious Diseases Division, Massachusetts General Hospital, Boston, Massachusetts

Numerous reports exist of the transmission of zoonoses to humans during and after solid-organ and hematopoietic stem cell transplantation. Donor-derived infections of numerous etiologies, including West Nile virus infection, Chagas disease, toxoplasmosis, rabies, lymphocytic choriomeningitis virus infection, and infection due to *Brucella* species have been reported. Most zoonoses occur as a primary infection after transplantation, and immunocompromised patients are more likely to experience significant morbidity and mortality from these infections. Risks of zoonotic infection in the posttransplantation period could be reduced by patient education. Increased recognition of the risks of zoonoses, as well as the advent of molecular biology-based testing, will potentially augment diagnostic aptitude. Documented zoonotic infection as it affects transplantation will be the primary focus of this review.

Zoonotic illnesses represent a significant risk to patients undergoing solid-organ transplantation (SOT) and hematopoietic stem cell transplantation (HSCT). Numerous reports exist of the transmission of zoonotic infection at the time of transplantation, either with the allograft or with blood products, as well as in the posttransplantation period, via the usual methods of transmission. The studies of zoonoses and transplantation-associated infectious diseases are evolving fields that are receiving increased recognition. Of the 1407 organisms that have been identified as human pathogens, 58% are zoonotic and are twice as likely as other pathogens to be in the “emerging” category [1]. The population of immunocompromised hosts is also increasing; as the annual number of transplantations that are performed increases, transplant recipients are living longer [2], more-powerful immunosuppressive agents are being administered, and tools for the management of chronic graft-versus-host disease are improving.

Because numerous reviews of the effects of zoonoses on the general human population have been written, this review will primarily focus on documented zoonotic infection involving

SOT and HSCT (excluding corneal and musculoskeletal grafting). Although much concern has been registered regarding the risk of zoonosis transmission with xenotransplantation (i.e., the transplantation of organs from animals to humans) [3], because this is not currently clinical practice, this will not be covered in this article. Cases described herein were found in reports in English-language journals via a search of the Medline database, using the search term “transplant” along with the name of the genus or syndrome. The defining criteria for zoonoses that are covered herein, as previously defined [4], include pathogens that have a nonhuman vertebrate reservoir, entail transmission from animals to humans, and have a recognized infectious disease syndrome in susceptible humans. Infections that do not involve a nonhuman vertebrate intermediary, such as malaria and dengue fever, will not be included. Transmission may occur directly (via contact with infectious animals or their secretions), via a nonvertebrate vector, or indirectly (via food, water, or a shared environment) [5]. Significant zoonoses are covered in the text and in table 1, and rarer or less-commonly reported zoonoses are also included. Live viral vaccines are also discussed. In general, the incidence of zoonotic illness is not known to be higher in transplant recipients, although the related morbidity and mortality may be higher among this population.

Population shifts resulting from immigration and travel are occurring throughout the world. Approximately 10% of the population of the United States was born in a foreign country [126], and more Americans than ever before are traveling in-

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Reprints or correspondence: Dr. Camille N. Kotton, Transplant and Immunocompromised Host Sect., Infectious Diseases Div., Massachusetts General Hospital, 55 Fruit St., Cox 5, Boston, MA 02114 (ckotton@partners.org).

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Table 1. Zoonotic pathogens in transplant recipients.

Pathogen	Primary mode of transmission	Transplant type [Reference]	Donor-derived infection reported	Major animal reservoir(s)
Bacteria				
<i>Anaplasma phagocytophilum</i> (HGE)	Tick bite	Kidney [6, 7] and pancreas [8]	No	Deer, mice
<i>Bartonella henselae</i>	Direct contact	Heart [9] and kidney [10–13]	No	Cats
<i>Borrelia burgdorferi</i>	Tick bite	Kidney [14], heart [15], and HSC [16]	No	Wildlife, rodents, deer, dogs, cattle, horses
<i>Bordetella bronchiseptica</i>	Inhalation	Lung [17], heart [18], and HSC [19, 20]	No	Dogs
<i>Brucella</i> species	Direct contact, ingestion, inhalation	Kidney [21, 22] and HSC [23]	Yes [23]	Goats, sheep, cattle, swine, horses, dogs
<i>Campylobacter</i> species	Ingestion	Numerous	No	Numerous
<i>Coxiella burnetii</i>	Inhalation, tick bite	Liver [24] and HSC [25]	No	Cats, dogs, cattle, sheep, goats
<i>Ehrlichia chaffeensis</i> (HME)	Tick bite	Liver [26–28], kidney [29, 30], and lung [31]	No	Deer
<i>Ehrlichia ewingii</i>	Tick bite	Kidney [32]	No	Dogs
<i>Erysipelothrix rhusiopathiae</i>	Direct contact	Kidney [21]	No	Fish
<i>Francisella tularensis</i>	Direct contact, ingestion, tick bite, inhalation	HSC [33, 34] and kidney [35]	No	Rabbits, rodents, wildlife
<i>Leptospira interrogans</i>	Direct contact, ingestion of urine	Kidney [36]	No	Rodents
<i>Listeria monocytogenes</i>	Ingestion	SO [37–40] and HSC [41, 42]	No	Farm animals
<i>Mycobacterium bovis</i>	Ingestion	Kidney [43]	No	Cattle
<i>Mycobacterium microti</i>	Inhalation	Kidney [44]	No	Rodents
<i>Mycobacterium marinum</i>	Direct contact	Kidney [45, 46], lung [47], and pancreas-kidney [48]	No	Fish
<i>Plesiomonas shigelloides</i>	Ingestion	HSC [49]	No	Fish
<i>Rhodococcus equi</i>	Inhalation	Numerous, including pancreas [50], pancreas-kidney [51], heart [52], liver [53], and lung [54]	No	Horses, swine
<i>Rickettsia</i> species	Tick bite	Heart [55] and liver [56]	No	Wildlife, rabbits, goats, sheep, dogs
<i>Salmonella</i> species	Ingestion	Numerous	No	Numerous
<i>Vibrio</i> species	Ingestion	SOs [57–60] and HSC [61]	No	Fish
<i>Yersinia</i> species	Ingestion	Kidney [62, 63]	No	Numerous
Fungus				
<i>Cryptococcus neoformans</i>	Inhalation	Numerous SOs [64–66]	No	Birds
Dermatophytes ^a	Direct contact	Kidney [67, 68], liver [69], and heart-lung [70]	No	Numerous
<i>Sporothrix schenckii</i>	Direct contact	Kidney [71]	No	Cats
Virus				
Hepatitis E virus	Ingestion	Kidney [72, 73]	No	Swine, wild boar, deer
Lymphocytic choriomeningitis virus	Direct contact, inhalation	Multiorgan [74]	Yes [74]	Rodents
Parapoxvirus ^b	Direct contact	Kidney [75, 76] and HSC [77]	No	Cattle

(continued)

Table 1. (Continued.)

Pathogen	Primary mode of transmission	Transplant type [Reference]	Donor-derived infection reported	Major animal reservoir(s)
Rabies	Exposure to infectious saliva	Multiorgan [78, 79]	Yes [78, 79]	Mammals
SARS virus	Inhalation	Liver [80], kidney [81], and HSC [82]	No	Civets, bats
West Nile virus	Mosquito bite	SOs [83, 84]	Yes [83]	Birds
Parasite				
<i>Babesia</i> species	Tick bite	Heart [85] and kidney [86–88]	No	Rodents, cattle
<i>Clonorchis sinensis</i>	Ingestion	BM (before transplantation) [89] and liver [90–92]	No	Fish
<i>Cryptosporidium parvum</i>	Ingestion	Liver [93, 94], kidney [95, 96], and HSC [97]	No	Numerous
<i>Echinococcus granulosus</i>	Ingestion	Liver [98–101] and heart [102]	No	Dogs, sheep
<i>Giardia lamblia</i>	Ingestion	Intestine [103] and HSC [104]	No	Numerous
<i>Leishmania</i> species	Insect bite	Numerous SOs [105–115] and HSC [116]	No	Rodents, dogs
Microsporidia	Ingestion	Kidney [117], kidney-pancreas [117], and BM [118, 119]	No	Numerous
<i>Taenia solium</i>	Ingestion	Kidney [120, 121]	No	Swine
<i>Toxoplasma gondii</i>	Ingestion	Numerous	Yes [122, 123]	Cats, cattle, goats, sheep
<i>Trypanosoma cruzi</i>	Insect bite	Kidney [124], liver [124, 125], and pancreas [124]	Yes [124, 125]	Rodents, wildlife, house pets

NOTE. BM, bone marrow; HGE, human granulocytic ehrlichiosis; HME, human monocytic ehrlichiosis; HSC, hematopoietic stem cell; SARS, severe acute respiratory syndrome; SO, solid organ.

^a E.g., *Microsporium canis* and *Trichophyton mentagrophytes*.

^b Causing orf (ecthyma contagiosum) or milker's nodules.

ternationally. These are both factors that may augment the risk of donor-derived infections, particularly the more latent ones (i.e., infections due to *Mycobacterium tuberculosis*, *Trypanosoma cruzi*, parasites, and others). The rise in the number of SOTs performed in developing countries (some of which are performed in patients who return to industrialized countries after transplantation) may create another reservoir for unusual infections.

Immunocompromised patients may have atypical presentations of infectious diseases, and the diagnosis may be elusive, especially for some of the less common zoonoses. Many zoonotic illnesses involve specialized diagnostic tests. Transplant recipients may be slow to evolve a serologic response, which may delay or deter diagnosis, especially if their overall level of immunosuppression is high. The use of molecular biology-based tests, when available, may augment our diagnostic capacity in this population, similar to the contribution from nucleic acid amplification testing of the blood supply for West Nile virus (WNV) [127]. If donor-derived infection is a possibility, donor samples (e.g., serum, tissues, blood vessels, and

autopsy specimens) should also be tested. Increasing recognition of the risks of zoonotic infection will potentially augment diagnostic aptitude.

Donor-derived infection. Zoonotic infection in the peri-transplantation period can be transmitted via the organ or stem cell allograft, as well as through the transfusion of blood products. Both acute and latent infections (such as Chagas disease or toxoplasmosis) may be transmitted via an allograft. Risk factors for zoonotic illness may be overlooked during the standard screening process. Subclinical or atypical illness, such as was observed in the cases of lymphocytic choriomeningitis transmission [74] and rabies transmission [78, 79], may result in imperfect screening and subsequent transmission of infection. WNV infection [83], Chagas disease [124, 125], and toxoplasmosis [122, 123] have also been transmitted during SOT. WNV and *Brucella* infections have been transmitted in HSCT allografts. After transplantation, the clinical syndromes associated with these infections can be protean and may even be mistaken for transplant rejection (e.g., hepatitis after liver transplantation). When there is concern regarding donor-derived

infection, both donor and recipient samples must be examined. Novel transmission should be promptly reported, because increased recognition may reduce subsequent infections.

Recipient-derived infection. Recipients in whom an infection is incubating at the time of transplantation and who subsequently experience profound immunosuppression in the peritransplantation period may develop severe infection. The majority of zoonoses are acquired after transplantation. Certain epidemiological risk factors increase the risk for acquisition of zoonoses, including occupational exposure (e.g., in veterinarians, pet store employees, farmers, slaughterhouse workers, landscapers, and forestry workers), pet ownership, hobbies (e.g., hunting), and travel. These exposures should be limited or possibly avoided, especially during the first 6 months after transplantation or other significant immunosuppression [128]. Perhaps more insidious are the risks that are more common and less obvious—that is, contaminated drinking water and food, walking in the woods or wading in the ocean, visiting a petting zoo, or exposure to any house pets.

PATHOGEN-SPECIFIC INFECTION

The following sections include both donor- and recipient-derived infections, with a focus on the characteristics of specific pathogens, by category.

Bacterial Infection

Enteric infection. Bacterial enteric pathogens are common etiologies of foodborne and waterborne illnesses, and may represent contamination from farm animals or other vertebrate animals; they may also be directly transmitted from animals to humans. Transplant recipients have much higher rates of bacteremias due to *Salmonellae* species—ranging as high as 70%, compared with 3%–4% in normal hosts—and a higher risk of a metastatic focus of infection [129, 130]; this is true for other enteric pathogens, as well. *Campylobacter* species are another group of common zoonotic enteric pathogens in patients who undergo SOT or HSCT. As in other immunocompromised hosts, transplant recipients may have trouble completely clearing a *Campylobacter jejuni* infection [131]. Asymptomatic, prolonged *Campylobacter* species bacteremia in the peri-HSCT period has been documented [132]. *Yersinia* species can also cause invasive disease with bacteremia in SOT [62, 63]. Noncholera *Vibrio* species can cause fulminant illness in transplant recipients, with gastroenteritis, bacteremia, or skin and soft-tissue infections [57–60]; environmental flooding increases the risk of illness due to *Vibrio* species, as was observed after Hurricane Katrina [133]. Recent increases in drug-resistant bacteria, sometimes related to the use of antibiotics in animal feed (such as with multidrug-resistant *Salmonella* species), are especially concerning.

Pulmonary infection. *Bordetella bronchiseptica*, the etio-

logic agent of “kennel cough” in dogs, has caused serious respiratory illness in patients who undergo pediatric lung transplantation [17], heart transplantation [18], and HSCT [19, 20]. Several of these case patients had pet dogs. The “kennel cough” live vaccine, which contains a mixture of parainfluenza virus and *B. bronchiseptica*, has the potential to cause human *B. bronchiseptica* infection [134]. *Rhodococcus equi* has been increasingly documented as a pulmonary pathogen in transplant recipients [135], as well as an agent of unusual infection, including cerebral infection in a heart transplant recipient [52], pericarditis in a kidney transplant recipient [136], and vertebral osteomyelitis in a liver transplant recipient [53]. Cases of tularemia (due to *Francisella tularensis* infection) after both HSCT and renal transplantation have been reported [33–35].

Systemic and other infections. There have been numerous reports of ehrlichiosis reported in transplant recipients, including human monocytic ehrlichiosis due to *Ehrlichia chaffeensis* infection in liver [26–28], kidney [29, 30], and lung [31] transplant recipients and several cases of human granulocytic ehrlichiosis due to *Anaplasma phagocytophilum* infection in kidney [6, 7] and pancreas [8] transplant recipients. *Rickettsia rickettsii* infection (Rocky Mountain spotted fever) has been described after heart transplantation [55], and *Rickettsia conorii* infection has been reported after liver transplantation [56]. *Borrelia burgdorferi* infection (Lyme disease) has been described in the literature in transplant recipients, including 1 kidney transplant recipient [14], 1 heart transplant recipient (with carditis) [15], and 1 allogeneic hematopoietic stem cell transplant recipient [16].

Bartonella henselae infection has been described after heart [9] and kidney transplantation [10–13], with variations in the manifestation of infection that include hemophagocytosis [137], closely associated acute allograft rejection [10], peliosis hepatis [138], peliosis hepatitis and hepatorenal syndrome [139], pulmonary nodules [140], and osteomyelitis [141]. *Brucella* species infection has been reported after kidney transplantation [21, 22] and as a donor-derived infection during HSCT [23], mostly in areas of endemicity. Live, attenuated *Brucella* animal vaccine has been linked to human disease and has the potential to cause disease in immunocompromised hosts [134]. *Listeria monocytogenes* infection has been well described in patients who undergo SOT and HSCT [37, 38, 41], including the rare clinical manifestations of tricuspid valve endocarditis with septic pulmonary emboli [39], epididymitis and orchitis [40], and skin infection with cerebritis and hemophagocytosis [42]. A small number of cases of human tuberculosis are due to *Mycobacterium bovis* infection, also known as zoonotic tuberculosis—an opportunistic infection in immunocompromised hosts [142]; *M. bovis* infection of the urinary tract has been documented after kidney transplantation [43].

Skin and soft-tissue infection. Numerous reports exist of *Mycobacterium marinum* infection in patients who undergo SOT, sometimes after a patient's exposure to fish [45–48]. *Erysipelothrix* species–related endocarditis that occurs after aquarium contact has been reported in a kidney transplant recipient [21]. *Capnocytophaga* species infections are usually caused by the organisms that are found in the oral flora of immunosuppressed hosts [143, 144] and not by the zoonotic species (i.e., *Capnocytophaga canimorsus* and *Capnocytophaga cynodegmi*, which have not been reported in transplant recipients); similarly, *Pasturella* species infections have not been reported in this population.

Fungal Infection

Cryptococcus species is the third most common cause of invasive fungal infection in organ transplant recipients after *Candida* species and *Aspergillus* species [64]. Birds and their droppings are the most commonly perceived risk. A 72-year-old kidney transplant recipient who owned a pet cockatoo developed cryptococcal meningitis that was believed to be acquired from the cockatoo, because isolates obtained from the patient and the bird had identical biochemical profiles, the same monoclonal antibody immunofluorescence patterns, and indistinguishable patterns on RFLP analysis and karyotyping [65]. Some authors suggest that immunocompromised hosts should not keep cockatoos, given their association with cryptococcosis [145].

Sporotrichosis due to *Sporothrix schenckii* infection can be connected to animal contact, especially contact with cats [146], and has caused severe, recurrent disease in a kidney transplant recipient [71]. Dermatophytes are common in both regular and exotic animals and can cause both superficial and invasive disease in humans [145, 147]. *Trichophyton mentagrophytes*, a zoonotic dermatophyte, was the most common superficial dermatophyte observed after kidney transplantation in 1 series [67]. The zoonotic dermatophyte *Microsporum canis* has caused invasive cutaneous infection after liver transplantation [69], relapsing tinea capitis after kidney transplantation [68], and dermatophytic granuloma with erythematous pustules and papules in a heart-lung transplant recipient [70].

Viral Infection

Viral zoonoses are numerous [4] and are common among the emerging zoonotic pathogens [148]; however, the vast majority have not been reported among transplant recipients. WNV infection is one of the more commonly reported viral zoonoses in transplant recipients. It may be donor derived, transfusion related, or normally acquired, and it carries a high morbidity and mortality. The risk of meningoencephalitis in a transplant recipient infected with WNV is estimated to be 40%—much higher than in normal hosts [84]. A recent case of WNV infection was confirmed in 3 of 4 recipients of organs transplanted

from a single donor; 2 recipients subsequently experienced neuroinvasive disease, 1 recipient developed asymptomatic WNV infection, and a fourth recipient was apparently not infected [83]. Numerous additional reports exist of transmission following SOT and HSCT.

Rabies is rarely observed after SOT. In a recent case in Arkansas and Texas, 1 donor transmitted lethal rabies infection to 5 recipients [78], and in another case in Germany, 3 patients who underwent SOT developed neurological symptoms and died [79]. Live rabies vaccine for use in wildlife has caused human disease and presents a potential risk to transplant recipients who come in direct contact with it [134].

Yellow fever presents a risk to transplant recipients who are traveling to or whose donors are from areas of endemicity for the disease, although instances of infection in this manner have not been documented. Use of the live attenuated vaccine should be avoided in immunocompromised hosts [149]. Although a few immunosuppressed travelers have tolerated the vaccine (e.g., those in the early stages of HIV infection or who have a distant history of hematological malignancy [150–152]), complications, including death, have been reported [153].

A recent report documented the spread of lymphocytic choriomeningitis from 2 asymptomatic organ donors to 8 organ transplant recipients, 7 of whom died [74]. The severe acute respiratory syndrome (SARS) virus caused significant disease in patients who underwent HSCT and recipients of liver and kidney transplants [80–82]. Infection with parapoxvirus, the agent responsible for orf (ecthyma contagiosum) and milker's nodules, has been observed after HSCT (in these cases, the infection was transmitted by cows) [77] and kidney transplantation [75, 76].

Parasitic Infection

Numerous zoonotic parasites have been shown to cause disease in transplant recipients. Bloodborne and organborne infection may be transmitted at the time of transplantation; enteric pathogens are less likely to be transmitted during the peritransplantation period, although this could potentially occur with intestine and liver transplantation. Depending on the location and circumstances, toxoplasmosis, babesiosis, Chagas disease, and leishmaniasis are among the more common parasite-related infections observed in transplant recipients.

Toxoplasma gondii infection can be caused by primary infection transmitted by an allograft, as well as by reactivation disease. *T. gondii* allograft transmission is classically associated with heart transplantation, in which case can persist as a latent infection in the myocardium, although it has been transmitted through transplantation of other organs. Among patients who undergo HSCT, toxoplasmosis occurs in 0.3%–7.6% of cases, with higher rates in countries where toxoplasmosis is more prevalent and among patients with graft-versus-host disease

[122]. The use of trimethoprim-sulfamethoxazole for post-SOT prophylaxis has decreased the risk of toxoplasmosis [154, 155]. In a recent review of 52 noncardiac SOT-related cases of toxoplasmosis, 86% of patients developed disease within 90 days of transplantation; of these patients, 42% had primary infection, 21% had reactivation or reinfection, and 37% had cases that could not be determined [123]. Classically, non-allograft-associated transmission was linked to the ingestion of either uncooked or undercooked meat containing viable tissue cysts or oocysts from the feces of infected cats; a report of an outbreak of toxoplasmosis associated with unfiltered municipal drinking water contaminated by felid waste [156] reiterates the importance of clean drinking water for transplant recipients.

Babesiosis has caused severe disease with hemophagocytosis and pancytopenia in asplenic renal transplant recipients [86, 87]. *Babesia* species have been transmitted through peritransplantation blood transfusions [85, 88]; in the United States, there has been a sharp increase in the number of transfusion-transmitted infections of *Babesia* species [157], suggesting a potential for increased infection in this generally heavily transfusion-dependent population.

Trypanosoma cruzi, the etiologic agent of Chagas disease, has been transmitted during SOT [124, 125], as well as during blood transfusions [158–160], and infection can also reactivate after transplantation [161]. Although most commonly associated with heart transplantation, other organs (including liver, kidney, and pancreas) may transmit *T. cruzi* as well [124, 125]. In a recent survey of 404 deceased organ donors in Southern California, where 25% of organ donors are of Hispanic ethnicity, 6 donors (1.5%) were found to be initially reactive by EIA, and 1 donor (0.25%) was found to have confirmatory *T. cruzi* antibodies, suggesting a beneficial role for the screening of transplant donors [162].

Leishmania species cause significant disease in immunocompromised hosts and could theoretically be transmitted via an allograft or a blood transfusion [163, 164]. Visceral disease (kala azar) is the most common manifestation after SOT, with 57 cases reported in the literature [105]. Patients who undergo HSCT appear to be rarely affected [116]. Cutaneous leishmaniasis has been reported in a handful of cases of SOT, some with concomitant visceral involvement [106–110]; mucosal disease has also been reported infrequently [111–115].

Enteric parasites are more likely to cause disease after transplantation. Microsporidia can cause infection in immunocompromised hosts; the most commonly reported is *Enterocytozoon bieneusi* infection following a transplantation [117]. Pulmonary infection has been described following allogeneic HSCT [118, 119], and disseminated disease was documented postmortem in a kidney-pancreas transplant recipient [117]. *Cryptosporidium parvum* infection is especially common in patients who undergo SOT in the developing world [95]. *Cryptosporidium*

species can also cause biliary disease and may play a role in some cases of otherwise unexplained cholangiopathies in liver [93] and kidney [96] transplant recipients. Disseminated *Cryptosporidium* species disease and related death have been described in liver [94] and stem cell transplant [97] recipients. Severe alveolar echinococcosis of the liver due to *Echinococcus* species has been successfully cured by liver transplantation [98–101], although there are risks of extrahepatic infection and potential echinococcal dissemination. Heart transplantation has been successfully performed in a patient who had hepatic echinococcosis [102].

RECOMMENDATIONS FOR PET OWNERS

Companion animals provide numerous benefits, along with some zoonotic risk. Discussions about pet ownership should optimally occur prior to transplantation. Pets may enhance health and well being, and many people would welcome advice and support to enable them to reconcile or manage pet ownership [165]. Guidance in pet choice can decrease zoonotic risk [128, 166]. In general, mature pets from reputable sources provide lower zoonotic risk. Fish are the pets least likely to be associated with illness (especially if aquarium cleaning by the transplant recipient is avoided). Animals to avoid as pets include reptiles (lizards, snakes, and turtles), baby chicks and ducklings, and exotic pets (chinchillas and monkeys); contact with stray and wild animals should also be avoided [128]. The individual risk of acquiring an infection from an animal is hard to calculate, and little work has been done in this field. In a survey of adult cats in Colorado, 13% were found to harbor zoonotic intestinal pathogens [167], and 41% of kittens in New York harbored a zoonotic agent [168].

Careful handwashing after any animal contact is imperative. Routine veterinarian care, with frequent stool examination for parasites, administration of routine vaccines, and evaluation when an animal is sick (especially with diarrhea), can reduce the risks of pet ownership to a transplant recipient. Immunocompromised hosts should avoid direct contact with any live viral vaccines that are administered to their pets and animals [134]. In addition, contact with animal excreta or saliva should be avoided. Good quality animal food should be given (not raw eggs or meat), and animals should not drink toilet bowl water. Humans should avoid flea and tick bites, as well as animal-related scratches and bites. Because small children are more likely to be bitten by pets and are less likely to practice good hand hygiene, pet ownership should potentially be deferred for very young transplant recipients. Pet therapy should potentially be avoided in hospitalized patients during the immediate posttransplantation period, when the patient is most immunosuppressed. The US Centers for Disease Control and Prevention's report on "Pets and Organ Transplant Patients" [169] provides both general and animal-specific guidelines.

CONCLUSIONS

Zoonotic infections are increasingly being recognized in transplant recipients, likely because of a greater number of transplantations, improved diagnostic testing in transplant recipients, and an augmented recognition of zoonoses. With increases in such factors as immigration, foreign travel, and exotic pet ownership, there may also be increased exposures to both donors and recipients. Risk of donor-derived infection may be reduced by improving the screening of donors, both through analysis of exposure history and through better molecular biology-based diagnostic testing. Given the current diversity and extent of animal contact, travel, occupational experience, and vector contact, a careful exposure history should be systematically ascertained in all transplant donors (when possible) and recipients. Education of transplant recipients before and after transplantation regarding zoonotic risks may further decrease zoonotic infection in this population.

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