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Donor-Derived Infection: Epidemiology and Outcomes

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Abstract Over the past decade, the solid organ transplant community has focused increased attention on unexpected transmission of infectious pathogens from organ donor to recipient. While unexpected donor-derived infections are relatively uncommon, recent cases of transmission of human immunodeficiency virus (HIV) and hepatitis C to multiple recipients, as well as transmission of HIV from a living donor, have further increased interest in improving the safety of solid organ transplantation. This article will review the epidemiology and outcomes associated with unexpected donor-derived infection. Furthermore, the reporting and patient safety process will be discussed, as will preventative measures that can reduce the burden of donor-derived infection.

Keywords Solid organ transplant · Donor derived infection

Introduction

The process of solid organ transplantation inevitably results in the transmission of infection from donor to recipient. In some cases (e.g., cytomegalovirus, Epstein–Barr virus), infected organs are knowingly transplanted into seronegative recipients, recognizing that the donor-derived infection can be managed with monitoring or preventative strategies. Less commonly, infections are unexpectedly transmitted from donor to recipient. These transmissions may result from limitations of the donor-screening process (e.g., window period infection), unusual pathogens not routinely screened (e.g., Chagas disease), or routine pathogens not known to be present in the donor at the time of transplantation (e.g., donor blood cultures for *Candida spp*. that become positive after the transplant). Notification of the transplant center of positive donor cultures allows interventions that may prevent donor-derived disease from developing. In other circumstances, donor origin of an infectious complication in the recipient may not be apparent until too late in the course to intervene, or the pathogen may have no reliably effective treatment (e.g., rabies virus). This article will review the epidemiology and outcomes associated with donor-derived infection. Furthermore, preventative strategies and the process involved in reporting possible donor-derived infection will be discussed.

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Epidemiology

General

Systematic information regarding donor-derived infection comes largely from the Disease Transmission Advisory Committee (DTAC), an Organ Procurement and Transplantation Network/United Network for Organ Sharing (OPTN/UNOS) committee. DTAC receives reports of possible donor-derived infections or malignancies in the United States. Reports may come from one of two sources. Organ procurement organizations (OPOs) are required to report new data, such as culture, sensitivity, or donor-screening tests, that become available after procurement to the accepting transplant center. In addition, OPOs and transplant centers must report suspected donor-derived infection to the OPTN/UNOS Patient Safety System. This process ensures notification of all transplant centers that have received organs from that donor, so that appropriate steps are taken to reduce recipient risk (e.g., administration of antimicrobials to at-risk recipients). The

committee reviews reported cases and categorizes cases as proven, probable, possible, IWDT (intervention without documented transmission), unlikely, or excluded. During the period 2006–2011, the number of potential donor-derived transmission events increased from 60 in 2006 to 181 in 2011 [1, 2]. This likely reflects not an increase in the number of transmission events, but a change in reporting policy, coupled with improved recognition and reporting of such events. Overall, a very small proportion of deceased donors (<2 %) have a potential donor-derived event reported. The number of reports varied widely from different donor service areas (DSAs), ranging from no reports to 44 reports from a single DSA [2]. Again, this likely reflects varying recognition and reporting of possible donor transmission events. From 2005 to 2011, 162 recipients with confirmed donor-derived infection were reported to DTAC. Of these, viruses were the most common (32 %), followed by fungi (26 %), bacteria (21 %), parasites (14 %), and mycobacteria (7 %) (see Table 1) [2]. Unusual organisms (often associated with poor outcomes) included viral pathogens such as lymphocytic choriomeningitis virus (LCMV), parvovirus, rabies virus, West Nile virus, and rickettsial organisms. Clusters of transmission from amoebic organisms (*Balmuthia mandrilaris, Naegleria fowleri)* and other tropical diseases, such as schistosomiasis and trypanosomiasis, were reported as well [1–3, 4••].

Outcome

General

The overall DTAC experience suggests that recipients affected by confirmed donor-derived infections have a relatively high mortality, with 42/162 (26 %) with death attributable to the donor-derived infection [2]. The pathogens causing recipient death have included bacterial organisms, West Nile virus, *Coccidioides*, *Cryptococcus neoformans, Balamuthia mandrilla*, *Mycobacterium tuberculosis*, LCMV, and *Scopulariopsis*, among others [1, 3]. Nonetheless, donor-derived transmission results in mortality in a very small proportion of transplant recipients. From 2005 to 2011, only 42 of approximately 200,000 transplant recipients died from donor-derived infection [2, 5]. It is likely, however, that unreported donor-derived events occurred. Furthermore, morbidity from donor-derived infection can be considerable (e.g., treatment, graft loss, prolonged hospitalization, and monitoring, psychic harm). Finally, the current patient safety system and increased awareness of donor-derived infection likely reduce transmission by alerting clinicians and allowing preventative interventions.

Epidemiology and Outcome by Organism

Blood-Borne Pathogens

While many less common pathogens (e.g., human T-cell lymphotrophic virus 1 [HTLV-1]) may be transmitted through blood transfusion, preventing transmission of hepatitis B, hepatitis C, and human immunodeficiency virus (HIV) has been the primary focus for those involved in regulating and improving the safety of blood product transfusion. The solid organ transplant (SOT) community has placed a similar focus on preventing transmission of these viruses. Prior to the availability of screening assays for HIV infection, several case reports described donor-derived HIV transmission [6, 7]. Outcomes were generally poor, with the development of opportunistic infection or another acquired immunodeficiency syndrome (AIDS) defining illness occurring as early as 3–4 months after transplantation [6–9]. Following the institution of routine donor screening, cases of donor-derived HIV infection have become extremely rare; however, two high profile cases of HIV transmission occurred recently in the United States. In 2007, a serologically negative, nucleic acid test (NAT) positive increased risk donor transmitted HIV and hepatitis C to four recipients, likely representing a window period transmission [10, 11]. Two patients died, and two others suffered graft loss. In 2009, a living kidney donor had a negative serum HIV antibody test 79 days prior to

Table 1 Etiology and outcomes of confirmed donor-derived infections reported to DTAC, 2005–2011

Disease	Number of donor reports	Number of recipients with confirmed transmission	Number of attributable recipient deaths	% Mortality
Virus	169	52 (32 %)	13	25 %
Bacteria	112	34 (21 %)	9	26 %
Fungus	78	42 (26 %)	11	26 %
Mycobacteria	51	12 (7 %)	3	25 %
Parasitic	34	22 (14 %)	6	27 %
Total infections	446	162	42	26 %

Adapted from Ison MG, Donor-derived infections: DTAC and UNOS—Rapid progress in 7 Years. In 24th International Congress of the Transplantation Society, July 19, 2012, Berlin, Germany

donation but acquired HIV prior to surgery. The recipient developed refractory esophageal candidiasis and was diagnosed with proven donor-derived HIV infection [12•]. While such cases are rare, they have led to calls to require donor screening, including NAT, of both deceased and living donors close to the time of transplantation [13].

Occasional cases of donor-derived hepatitis B and hepatitis C have been reported [1, 4••, 14]. Causes include human error (failing to correctly respond to a positive test in a living donor), window period infection, and transmission associated with vessels. In one recent case, three of five recipients from a standard risk seronegative donor developed acute hepatitis B. Subsequent testing on archived donor specimens demonstrated low-level viral viremia; thus, the donor was likely in the window period [14]. It is important to note that sole hepatitis B core antibody liver (and very rarely, sole core antibody nonhepatic) donors may transmit hepatitis B even if the recipient is vaccinated. Surface antigen positive or viremic donors are at higher risk for transmission. Prophylactic strategies using antivirals and hepatitis B immunoglobulin can markedly reduce the risk of donor-derived hepatitis B [15]. In the case of hepatitis C, preventative strategies are unproven, and treatment is likely to be poorly tolerated in SOT recipients. Furthermore, immunosuppressed hepatitis C patients are more likely to develop cirrhosis and end-stage liver disease.

HTLV-1 is a retrovirus that leads to neurological disease (HTLV-1-associated myelopathy or T-cell leukemia) in a minority of infected individuals and is uncommon among American donors. One report described the fairly rapid development of HTLV-1-associated myelopathy in three seronegative recipients of an infected donor in Spain [16]. Prior to 2009, OPTN policy required testing all deceased donors for HTLV-1. Both practical considerations (discontinuation of the most commonly used approved assay) and a high false positive rate in a low seroprevalence population, leading to wastage of uninfected organs, led to elimination of the requirement for testing [17]. Consideration of screening (particularly of living donors where time is available for confirmatory testing) of donors from high-risk geographic areas (Caribbean, part of South America, West Africa, and parts of Asia) is reasonable.

Bacterial Pathogens

Isolation of routine bacterial pathogens (representing colonization or disease) from donors is common [18], and in most situations, administration of prophylactic antibiotics prevents the development of disease in the recipient. For example, many centers treat lung recipients for bacterial pathogens isolated from donor lungs to prevent the development of pneumonia. Nonetheless, multiple instances of bacterial donor-derived infection have been reported [19–22]. In the DTAC experience, clusters of transmission often occur, with about one half of exposed patients developing infection [19, 21••]. Outcomes can be poor, mycotic aneurysms are frequent, and death and graft loss are common [19–22]. Multidrug-resistant pathogens (*Acinetobacter baumanii, Pseudomonas aeruginosa*, vancomycin-resistant *Enterococcus*) are typically reported. In the DTAC report, no infected recipients received antibiotics as prophylaxis that would have treated the transmitted pathogen, prior to pathogen identification [19]. Donors with open abdomens (trauma victims requiring abdominal packing) may be at particular risk of transmitting multidrug-resistant organisms [22]. Less common bacterial organisms, such as *Ehrlichia spp*., *Brucella spp*., *Borrelia burgdorferi*, *Ricketsia rickettsia*, and *Treponema pallidum* (syphilis), have caused donor-derived infection as well [2, 19].

Mycobacterial Pathogens

Following primary infection with *Mycobacterium tuberculosis*, the disease typically disseminates, and infectious organisms may be present in a latent state in any organ. Immunosuppression increases the risk of reactivation of latent tuberculosis, and SOT recipients are at 20 to 74 times the risk of developing active tuberculosis [23]. Thus, it is not surprising that donor-derived transmission of tuberculosis has been reported. Transmission may occur from either donors with undiagnosed active tuberculosis (e.g., tuberculous meningitis) or donors with latent tuberculosis. Since the performance of both interferon gamma release assays and the purified-protein derivative test (PPD) are not validated in deceased donors and PPD could not realistically be used, testing deceased donors for latent tuberculosis is not practical currently. In the living donor, an assessment for risk factors for MTB and application of a test for latent TB (universal testing or based on risk factors) is reasonable and practical. Thirty cases of confirmed or probable donor-derived tuberculosis have been reported to DTAC or otherwise described in the literature [1, 3, 4••, 24]. As was expected, disease was often extrapulmonary, with a distinct minority with disseminated (miliary) disease. Granulomatous disease or "cold abscess" involving the graft occurred in a number of recipients as well. Death occurred in 5/30 (17 %), and graft loss occurred in several cases as well [1, 3, 4••, 24]. As of 2009, no confirmed cases of transmission of atypical mycobacteria have occurred in the DTAC experience [4••]. Theoretically, such transmission would be most likely in lung transplants, since disseminated atypical mycobacteria would be very uncommon in the donor population.

Fungal Pathogens

Donor-derived infections with molds such as *Aspergillus and Rhizopus spp*. have resulted in recipient infection and

death; submersion death in the recipient was the likely risk factor in some cases [1, 3]. *Candida spp*. (as well as *Aspergillus*) may infect the anastomotic site, resulting in pseudoaneurysm that may be complicated by graft loss and life-threatening hemorrhage. Renal transplant recipients are at highest risk, and the preservative solution may be the source in some cases. Among the endemic fungi, *Coccidiodes* is the most commonly reported, and infected recipients have a high death rate (possibly related to delays in recognizing infection) [25, 26]. Proven transmission of *Hisptoplasma spp*. has been reported as well [27], and cases series describe early infection after transplantation but not proven donor origin [28–30]. *Cryptococcus* may be transmitted particularly from nonhepatic donors with known cirrhosis, since cirrhosis is a risk factor for disseminated cryptococcosis.

Parasitic/Tropical Pathogens

Parasites confirmed to have caused donor-derived infection include *Babesia*, *Balamuthia mandrillaris* (see below), *Trypanosoma cruzi*, *Naegleria fowleri*, *Schistosoma,* and strongyloides . Due to prolonged latency and the growing Hispanic population in the United States (it is estimated that 300,000 infected persons live in the U.S.) [31], *Trypanosoma cruzi* (Chagas disease) has received particular attention. A multidisciplinary working group met in 2008 to provide guidance regarding Chagas disease in organ transplantation [32•]. Recommendations included targeted serological screening of donors born in endemic areas, avoiding transplant of hearts from infected donors, periodic screening using NAT (consultation with Division of Parasitic Diseases, CDC 770-488-7775), and blood microscopy in recipients of seropositive donors. Suspected or confirmed transmission can be treated with benznidazole or nifurtimox obtained from the CDC.

Selected Pathogens That Cause Meningoencephalitis

Particular concern has centered on the appropriate evaluation of donors with unrecognized meningoencephalitis. This reflects a high mortality rate and difficulty treating many of the pathogens that cause meningoencephalitis. Two clusters of donor-derived infection resulting from transmission of the amoeba *Balumuthia mandrillaris* illustrate the risk associated with the donor with meningoencephalitis. In one case, the donor was a 4-year-old boy who died with brainstem herniation and subarachnoid hemorrhage following an episode of influenza A. He did have a lymphocytic pleocytosis and enhancing central nervous system (CNS) lesions on magnetic resonance imaging. He was given a presumptive diagnosis of acute demylelinating encephalitis (ADEM) following influenza A, and his kidneys, liver, and heart were transplanted into four different recipients. Autopsy revealed amoebic encephalitis, and one kidney recipient died of amoebic encephalitis; the other had a significant episode of amoebic encephalitis but partially recovered. With multidrug prophylaxis, neither the heart nor the liver recipient has developed disease [33]. The second case occurred in Arizona; a 27-year-old landscape worker was given a diagnosis of stroke, but both the kidney–pancreas and liver recipient from the donor died of proven *Balamuthia mandrillaris* encephalitis [34].

Over the past decade, West Nile virus (WNV) emerged as a pathogen of major concern for donor-derived infection, and a number of transmissions with fatal results in the recipients have been reported [35–37]. Most infected individuals are asymptomatic, and donors with another cause of death may have viremia but exhibit no or limited signs or symptoms of WNV infection. For example, in one cluster, the donor died of a traumatic brain injury, but after the transplant, his spouse reported significant mosquito exposure and possible fever prior to the head injury. Three of four recipients developed WNV, with two developing severe neurological disease [35]. Infection of the donor via blood products transfused the day prior to transplantation has also been reported; in that case, all four recipients developed WNV disease [37]. Immunoglobulin (both standard preparation and a high-titer Israeli product, Omr-IgG-am; Omrix Biopharmaceutical) have been used to treat or prevent disease, with variable results [35–37].

A review of the DTAC experience from 2008 to 2010 demonstrated in 5 donors with unrecognized meningoencephalitis, transmission to 9/13 (69 %) recipients occurred, and 3 recipients died. Pathogens included *Balamuthia, Cryptococcus*, and WNV. Listed causes of death for the donors included stroke, intracerebral hemorrhage, anoxia, and ADEM [38]. CNS causes of death are very common in deceased donors; among 40,058 donors recovered from 2006 to 2010, CVA was a cause of death in 40.2 %, head trauma in 35.6 %, and anoxia in 21 %. Onehundred fifty-three, or 0.4 %, of recovered donors had CNS infection as a listed cause [38]. In general, donors with recognized treatable causes of meningitis (e.g., *Streptococcus pneumonia*) can safely donate organs, and most experts continue antibiotics in the recipients for a brief period. However, as the cases above illustrate, potential donors with unrecognized meningoencephalitis have resulted in fatal or severe disease in donors. Recognizing these donors is difficult, and a clear set of risk factors is not available. The DTAC created a guidance document providing some commonsense recommendations. Donors at low risk for cerebrovascular accident (e.g., pediatric donors) and those admitted with fever and mental status changes or seizure require careful evaluation, to exclude untreatable causes of meningoencephalitis. Environmental risk factors (e.g., bat exposure for rabies) should be considered. If overall evidence suggests an untreated or

undiagnosed meningoencephalitis, caution is warranted in offering or accepting these organs.

Prevention

Deceased Donor Screening

The OPTN/DTAC set policy for the screening of donors prior to transplantation. Required testing includes HIV, hepatitis B surface antigen, hepatitis B core antibody, hepatitis C antibody, VDRL or RPR, and serological testing for CMV and EBV [39]. With the exception of the VDRL/RPR, testing must utilize Food and Drug Administration cleared or approved screening tests. On the basis of reports of serious donor-derived disease, some centers test all donors or donors selected on the basis of risk factors for infection with other pathogens, such as WNV, *Typanosoma cruzi*, or *Coccidioides*.

While not yet required by policy, the use of NAT has increased in OPOs due to concern for window period infection to some degree prompted by highly publicized reports of donor-derived hepatitis C and HIV transmission [10–11, 12•]. As compared with traditional serological methods, NAT testing reduces the window period for HIV from 17–22 to 5–6 days, for hepatitis C from 70 to 3–5 days, and for hepatitis B from 35–44 to 20–22 days [40••]. Most OPOs use NAT testing in addition to standard serological screening, for all or some potential deceased donors [41]. Concern regarding potential false positive tests and ensuing loss of uninfected organs, as well as the cost and practicality of NAT testing in less sophisticated OPOs, has been suggested by several to advocate for limiting use of NAT to increased risk donors [40••]. Proposed U.S. Public Health Service (PHS) recommendations advocate for more extensive use of NAT testing, particularly for increased risk deceased donors (HIV) and all potential donors (hepatitis C) [13].

Living Donor Screening

Currently, OPTN/UNOS does not require any specific testing of living donors for transmissible infections, but most centers perform screening similar to that performed with deceased donors. However, in the living donor situation, adequate time is available to perform confirmatory testing (e.g., HTLV immunoassays), and a more extensive history can be obtained directly from the donor to assess epidemiologic risk for geographically or occupationally restricted infections. Some centers test at-risk donors for potentially transmissible latent infections, such as *Trypanosoma cruzi*, tuberculosis, Leishmaniasis, and strongyloides.

A recent case of window period HIV transmission from a living donor has generated significant discussion regarding the optimal HIV, hepatitis C, and hepatitis B testing of living donors [12•]. The U.S. PHS recommends screening of all living donors with NAT testing for HIV and hepatitis C and repeating testing within 7 days of the transplant procedure [13]. Others have argued that, given the rarity of window period infection (one known HIV transmission from a living donor in over 100,000 living donors), most positive tests will be false positives, resulting in potential delay or cancellation of transplantations.

Increased Risk Deceased Donors and Recipient Follow-up

In 1994, the CDC published a list of risk factors placing donors at higher risk for recent acquisition of HIV, and these criteria have been used to define a population of potential donors at increased risk for window period infections with hepatitis C, hepatitis B, or HIV that might be transmitted to recipients despite negative serological screening. These criteria include donors with a history of any of the following: men who have had sex with men in the preceding 5 years, users of nonmedical injectable drugs, persons with hemophilia, commercial sex workers, sexual contacts of any of the above, recent inmates of correctional facilities, and persons with recent occupational HIV exposures [42]. The PHS is currently working on a revision of these risk factors that should be finalized by late 2012 [13]. A recent meta-analysis demonstrated that for hepatitis C, the highest risk (without NAT screening) of window period infection was 300/10,000 donors for injection drug users [43]. For window period HIV infection, the highest risk was for men who have sex with men (10.2/10,000) and intravenous drug users (12.1/10,000) [44]. Due to the increased risk of donor-derived infection in OPTN-defined increased donors, routine follow-up testing for hepatitis B, hepatitis C, and HIV is indicated in this group. While the optimal testing strategy is not known, follow-up testing at 1, 3, and 12 months post-transplant using both NAT (since serologic conversion may be absent or delayed in immunosuppressed patients) and serologic testing is a reasonable strategy. Routine testing is supported by the pre-HIV testing experience where outcomes in recipients with donor-derived HIV were poor and opportunistic infections/AIDS defining illnesses occurred as early as 2–4 months posttransplantation. Despite this, a 2007 survey found that most centers do not routinely test recipients of high-risk donors and those that do may not use NAT testing.

Conclusions

Unexpected donor-derived infection remains relatively uncommon in SOT recipients. Viral organisms are most commonly transmitted, followed by fungal, bacterial, parasitic,

and mycobacterial pathogens. In many cases, the administration of preventative antimicrobials can prevent or ameliorate symptomatic disease in the recipients. Outcomes, however, can be poor in patients with confirmed transmission, with death rates of about 25 % overall. Further morbidity including graft loss may result from the donor-derived infection or treatment of the infection. Pathogens that do not have proven treatments or are not quickly recognized in the recipient (e.g., multidrug-resistant bacteria, rabies virus, amoebic organisms) create the highest risk of morbidity and mortality. While controversial, NAT testing provides the opportunity to reduce the risk of transmission of window period infection of common blood-borne pathogens from both deceased and living donors and will likely be required in the near future. Focused testing for geographically or epidemiologically restricted pathogens (e.g., *Coccidiodes, Trypanosoma cruzi*) should be considered, particularly for living donors, when practical.

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- •• Of major importance

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