Strongyloides stercoralis infection in solid organ transplant recipients

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Purpose of review
Strongyloides stercoralis infection remains of concern due to its high associated morbidity among solid organ transplant recipients (SOTR) and the risk of donor-derived infection (DDI). We review key aspects of epidemiology to inform screening for and treatment of chronic infection among organ transplant candidates to reduce the risk of infectious complications in the posttransplant setting.

Recent findings
In this work, we offer guidance regarding the optimal management of *Strongyloides* hyperinfection syndrome and disseminated infection and offer recommendations regarding posttreatment surveillance and the potential need for repeat treatment during subsequent periods of augmented immunosuppression. This review also provides updated recommendations for screening of deceased and living donors as recently proposed by the Organ Procurement and Transplantation Network's Ad Hoc Disease Transmission Advisory Committee.

Summary
Risk reduction of *Strongyloides* infection in the SOTR population can be further enhanced by optimized treatment of infection, posttreatment surveillance during at-risk periods and recent proposed policy shifts to universal donor screening.

Keywords
Strongyloides stercoralis, strongyloidiasis, transplantation

INTRODUCTION

Recognized for its unique lifecycle, significant clinical consequences, and specialized management, *Strongyloides stercoralis* has emerged over the past several decades as a formidable pathogen afflicting solid organ transplant recipients (SOTR). A thorough understanding of the epidemiology associated with this organism helps identify patients at risk for infection. Knowledge of this infection's multifarious clinical manifestations can inform a comprehensive diagnostic evaluation. As experience with strongyloidiasis in immunocompromised hosts has grown, transplant programs have updated management practices to improve clinical outcomes and prevent relapse of this infection. Further, recent changes in the screening of organ donors and recipients have the potential to significantly reduce the incidence of infection occurring after transplant. In this work we review our current understanding of *S. stercoralis* infection in SOTR and offer guidance regarding its diagnosis, management, and updated prevention strategies.

Description of the pathogen

First described in French soldiers returning from modern day Vietnam, strongyloidiasis is the clinical syndrome associated with infection related to members of the *Strongyloides* nematode genus [1]. The name *Strongyloides* derives from the Greek "strongylos" meaning "round" and "eidos" meaning "similar," terminology indicating a resemblance to *Strongylus*, another nematode [1]. Similar to other common roundworms including *Ascaris*, *Ancylostoma*, *Necator*, and *Trichiuris*, *Strongyloides* is classified as a soil-transmitted helminth.

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KEY POINTS

- Strongyloidiasis is a common global infection with substantial associated morbidity and mortality in the setting of hyperinfection and dissemination that warrants deliberate consideration and management in immunocompromised transplant recipients.
- Oral ivermectin remains the preferred antihelminthic treatment for *S. stercoralis* hyperinfection or dissemination but alternate routes of ivermectin therapy via compassionate or investigational use may be needed in some cases and in combination with albendazole therapy.
- Complete eradication of *S. stercoralis* in the immunocompromised host can be difficult to confirm and therefore posttreatment surveillance is recommended during periods of augmented immunosuppression.
- Universal donor screening and either universal or enhanced targeted pretransplant recipient screening are key approaches to prevention of *Strongyloides* infection in organ transplant recipients.

Whereas a number of *Strongyloides* species infect various vertebrates, *S. stercoralis* is the primary species that infects humans, though canines may also serve as definitive hosts [1]. The *S. stercoralis* lifecycle is depicted in Fig. 1. Filariform larvae in the environment infect a suitable host via penetration into skin, the most common route of entry, or consumption via contaminated food or water [2]. Once they penetrate either the skin or the mucosa of the gastrointestinal tract, filariform larvae migrate through the circulatory system to the lungs, ascend the respiratory tract, and are swallowed into the gastrointestinal tract. Adult females that have reached the small intestine, commonly the duodenum, lay eggs in the mucosa that mature and release rhabditiform larvae in the lumen which are shed in the stool.

Critical to infection in the human host, rhabditiform larvae that hatch in the gut lumen may also develop into infectious filariform larvae before leaving the host (Fig. 1). This unique ability gives rise to a cycle of autoinfection as these filariform larvae can then penetrate through the intestinal mucosa or perianal skin, enter the circulatory system, and perpetuate infection ad infinitum in the untreated host. This leads to a lifetime infection even if the host is no longer being exposed in an endemic region. A multitude of virulence factors involved in proteolysis, inhibition of host proteases, and prevention of gut expulsion are likely involved in this process of invasion and persistence within the host [3]. The *Strongyloides* cuticle may also promote immune evasion [4]. When the host is not able to control the rate of autoinfection massive repeat penetration and larval migration can lead to disseminated disease.

A variety of immunocompromising conditions are associated with progression and persistence of infection. Solid organ transplantation (SOT) and immunomodulatory medications including corticosteroids are risk factors for complications related to *S. stercoralis* [2]. Coinfection with human T-lymphotrophic virus type 1 (HTLV-1), a retrovirus with a specific geographic predilection that is estimated to infect several million people worldwide, serves as another risk factor for complications related to *Strongyloides* infection [5]. Whereas helminth infection typically elicits a robust Th2 response, HTLV-1 coinfection in patients with strongyloidiasis is associated with a diminution in interleukin (IL)-5, IL-4, IL-13 and immunoglobulin E (IgE) production, a finding suggesting a transition to a potentially less effective Th1-predominant response [5].

Epidemiology

The association of strongyloidiasis with soil, food, or water contaminated with fecal matter contributes to its disproportionate impact on communities afflicted by socioeconomic challenges such as inadequate infrastructure, suboptimal hygiene practices, and limited access to protective footwear [6▪]. The utilization of night soil, human feces used as fertilizer, remains a common practice in some parts of the world [7]. Contamination of raw fruits and vegetables occurs frequently in some regions [7].

On a global scale, multiple factors have hindered efforts to accurately characterize the epidemiology of *S. stercoralis* infection. Screening for infection is challenging in resource-limited settings, and small volume stool collection mars the sensitivity of studies relying on coproparasitological examination for diagnosis [8]. Furthermore, the sensitivity and specificity of serologic screening assays varies between available platforms, and cross reaction with other helminth species may occur with some tests [9].

S. stercoralis is estimated to infect >600 million people worldwide with a predominance in tropical and subtropical regions [10]. In some communities within Latin America, Africa, and the Western Pacific, prevalence may exceed 70% [11]. Southeast Asia, Africa, and the Western Pacific account for approximately 76% of infections globally [10]. Though *S. stercoralis* bears the World Health Organization's designation as a neglected tropical disease, autochthonous transmission may also occur in temperate and arid climates [6▪,12].

In the developed world, prior residence in an endemic region has been identified as a risk factor

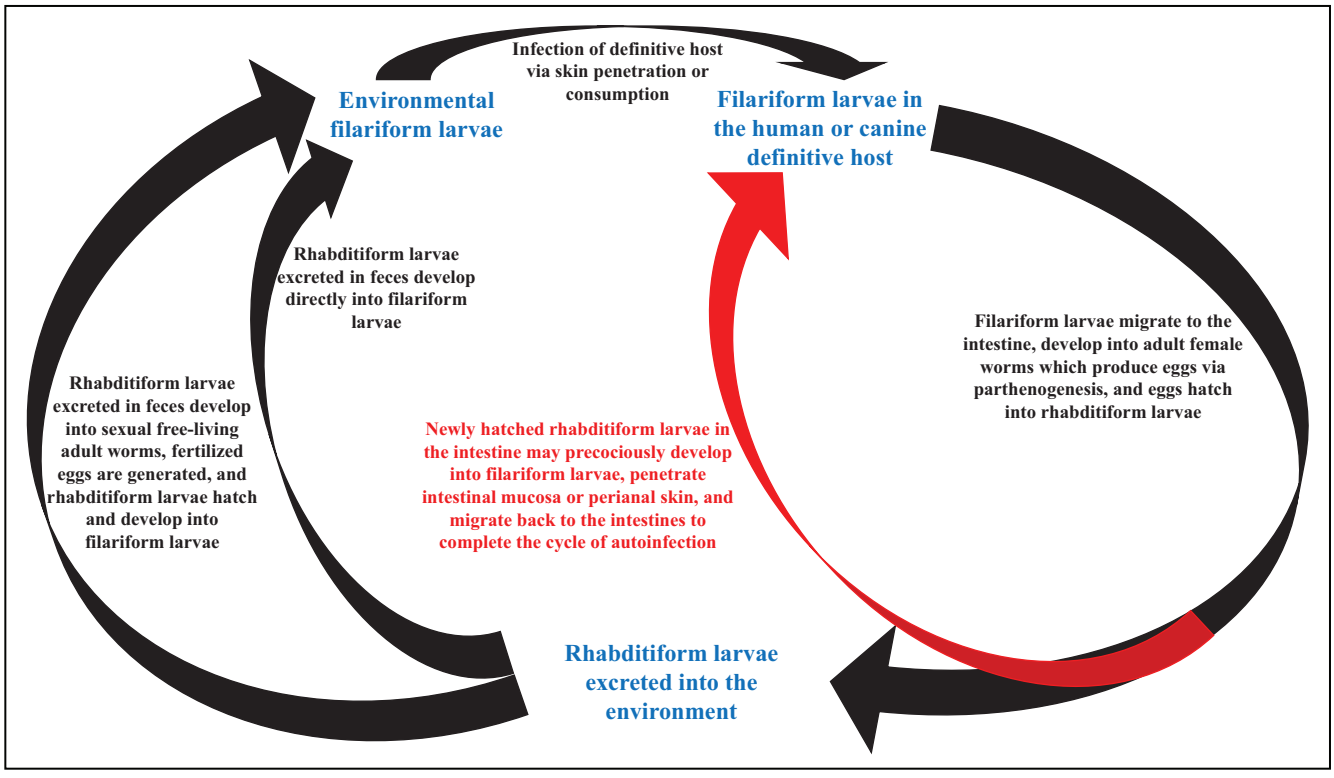

FIGURE 1. *S. stercoralis* lifecycle[28].

that may account for the vast majority of cases [13]. However, clinicians should consider other potential risk factors including dietary habits, occupational exposures, canine exposure, and the presence of household contacts who may have emigrated from endemic regions. Within the US, *S. stercoralis* has primarily been identified in the southeast. In particular, rural areas of the Appalachian region, especially within Kentucky, may exhibit significant prevalence rates [14]. Prevalence is also notably higher among individuals who have been institutionalized related to cognitive or psychiatric conditions [15]. Recent modeling by Singer and Sarkar suggests that multiple other states within the US may also harbor conditions conducive to autochthonous transmission [16].

Though donor-derived infection (DDI) is overall rare, recent data suggest that transmission via organ transplantation is more common than previously recognized [17,18]. Despite the availability of improved diagnostic tools, strongyloidiasis currently represents the most common parasitic DDI following SOT [19]. In a Centers for Disease Control and Prevention review of seven clusters of donor-derived strongyloidiasis, 86% of donors were born in Central or South America [20]. Whereas intestinal transplant may pose the most significant risk of transmission, other organs may also transmit infection [20,21].

Clinical manifestations

Heavily influenced by host immunity, *S. stercoralis* infection may present with a wide spectrum of clinical manifestations. In the general population, asymptomatic infection is most common. When immunocompetent hosts do experience symptoms, abdominal discomfort, vomiting, bloating, and loose stool, sometimes alternating with constipation, are frequent complaints [2]. Cutaneous manifestations are also common. *Larva currens*, a raised, erythematous, often serpentiform exanthem that has a predilection for the abdomen and extremities, is associated with the transit of filariform organisms through the skin. This rash is pathognomonic for *S. stercoralis*. In contrast to cutaneous *larva migrans* associated with hookworm infection that often lasts weeks, may progress up to two centimeters per day, and is typically bright red, *larva currens* is typically evanescent, may progress several centimeters per hour, and more often takes on a lighter color [22,23]. In cases of disseminated disease, periumbilical purpura, sometimes in a pattern reminiscent of thumbprints, and sometimes characterized by centrifugal spread, may occur as exemplified in Fig. 2 [24]. Purpura are thought to be secondary to invasion of the dermis by larvae that migrate through the vessel wall. In chronic cases, cutaneous manifestations including urticarial or pustular lesions may occur and are thought to represent

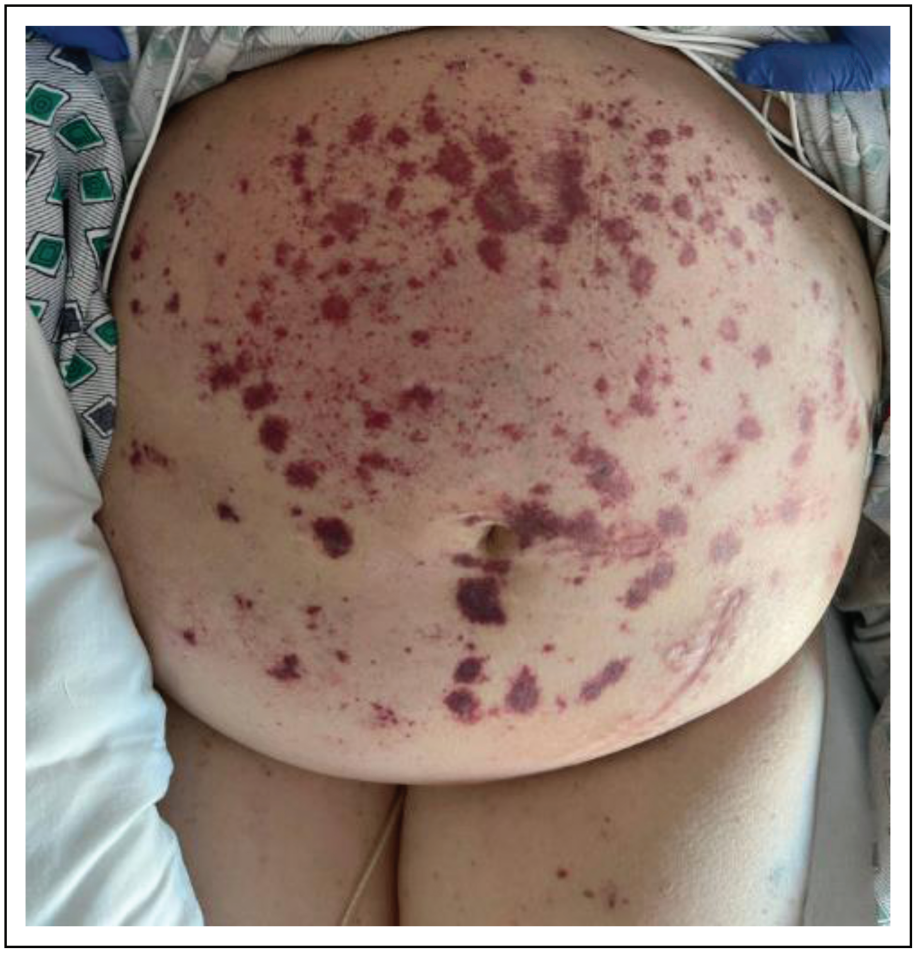

FIGURE 2. Purpuric abdominal rash in a solid organ transplant recipient with *S. stercoralis* hyperinfection syndrome.

immunologic sequelae [2]. Angioedema, similarly thought to be an immunologic phenomenon, may also be observed [2]. Symptoms related to pulmonary involvement including cough, wheezing, or throat irritation may be noted [2]. Eosinophilia is a common but inadequately sensitive and nonspecific finding associated with strongyloidiasis [25].

Immunocompromised hosts have a greater risk of progressive, complicated, persistent, and recurrent infection. In SOTR, immunomodulatory medications including corticosteroids may accelerate the *S. stercoralis* autoinfection cycle, potentially with devastating effect [26]. Hyperinfection syndrome, characterized by the rapid proliferation of larvae in the gut and migration between the respiratory and gastrointestinal tracts, carries a nearly 50% risk of mortality [25]. Patients often present with systemic symptoms such as fever, prominent gastrointestinal symptoms including vomiting, diarrhea or ileus, hematochezia, and abdominal pain, and pulmonary symptoms including cough, dyspnea, and hemoptysis. In cases with significant lung involvement, CT imaging may demonstrate a variety of findings. Ground glass opacities as depicted in Fig. 3a and prominent interlobular septal thickening (crazy paving pattern) as depicted in Fig. 3b are common in patients with hyperinfection syndrome [27]. The acute respiratory distress syndrome may further complicate pulmonary disease. Disseminated disease occurs when a patient with hyperinfection syndrome progresses to develop organ involvement outside the respiratory and alimentary tracts. This condition is associated with high morbidity and a nearly 70% risk of mortality if not recognized and treated early [25]. A massive helminth burden penetrating through the gut wall may contribute to secondary bacterial infections including polymicrobial bacteremia, meningitis due to enteric organisms, and concomitant sepsis.

DDI can manifest with a wide spectrum of symptomatology, can be acute or chronic, and can range in clinical severity. Gastrointestinal manifestations are most common [17]. Notably, the latency period between the time of transplantation and the subsequent development of symptoms related to DDI is long, ranging from seven to 33 weeks in one study [20]. Strongyloidiasis in SOTR in the early post-transplant period may represent newly acquired

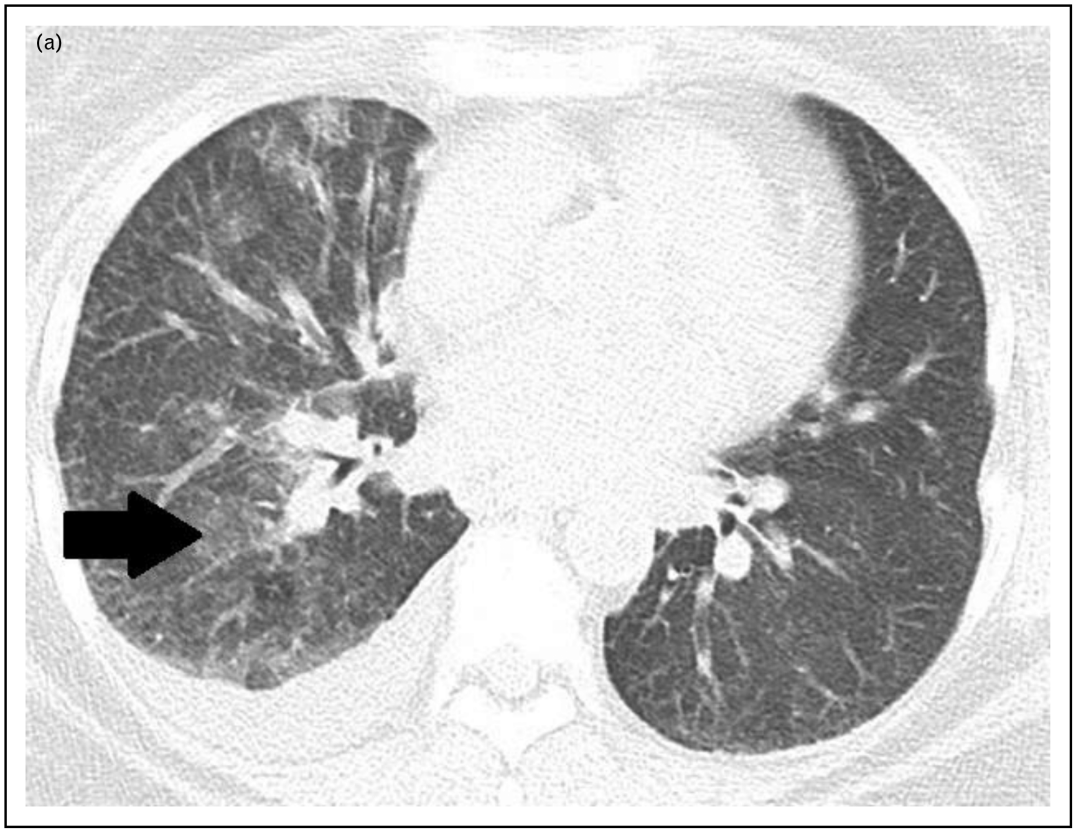

FIGURE 3. (a) Ground glass pulmonary opacities (arrow) in a solid organ transplant recipient with *S. stercoralis* hyperinfection syndrome complicated by hypoxemic respiratory failure. (b) Prominent pulmonary interlobular septal thickening in a "crazy paving" pattern (arrow) in a solid organ transplant recipient with *S. stercoralis* hyperinfection syndrome complicated by hypoxemic respiratory failure.

infection, reactivation of chronic infection, or donor-derived disease. Therefore, a detailed history, a careful review of donor and recipient risk factors, and a high degree of clinical suspicion may be required to confirm a diagnosis of DDI.

Diagnosis

S. stercoralis is challenging to detect with the naked eye as its size ranges from 600 μm in filariform larvae to nearly 3 mm in adult parasitic females [28]. Coproparasitological analysis, though useful, has limitations. The sensitivity of stool microscopy increases with the volume of fecal matter assessed, and patients may need to produce up to seven samples to achieve optimal results [25]. Spreading stool on a nutrient agar plate and assessing for worm trails is a sensitive test, but this assay is not widely performed and can be resource intensive as detection requires days of visualization and microscopic analysis [2]. Requisite technical expertise may limit the availability and clinical utility of all traditional stool studies. Additionally, it may take several weeks before larvae are detected in the stool of a newly infected patient [2]. PCR and antigen testing may enhance the sensitivity of stool analysis but these diagnostic options are not widely available [29,30].

In patients with symptomatic disease involving sites outside the gastrointestinal tract, diagnosis can be made by direct microscopic examination of clinical samples including skin/tissue biopsies, ascites fluid, cerebrospinal fluid, and bronchoalveolar lavage fluid. Importantly, just as peripheral eosinophilia may be diminished or absent in the setting of corticosteroid use, immunosuppressive agents may also dampen or completely abrogate the expected inflammatory histological changes observed on tissue samples obtained from sites of infection [24].

Serologic testing circumvents the need for cumbersome stool collection and can be useful as a screening tool in many clinical scenarios. Multiple serology platforms are available, and their sensitivity and specificity vary by assay [9]. Though many commercially available tests offer favorable performance characteristics, all serologic testing carries a series of caveats [9]. Most importantly, testing may be negative in the setting of acute infection and it is more likely to be positive in the setting of

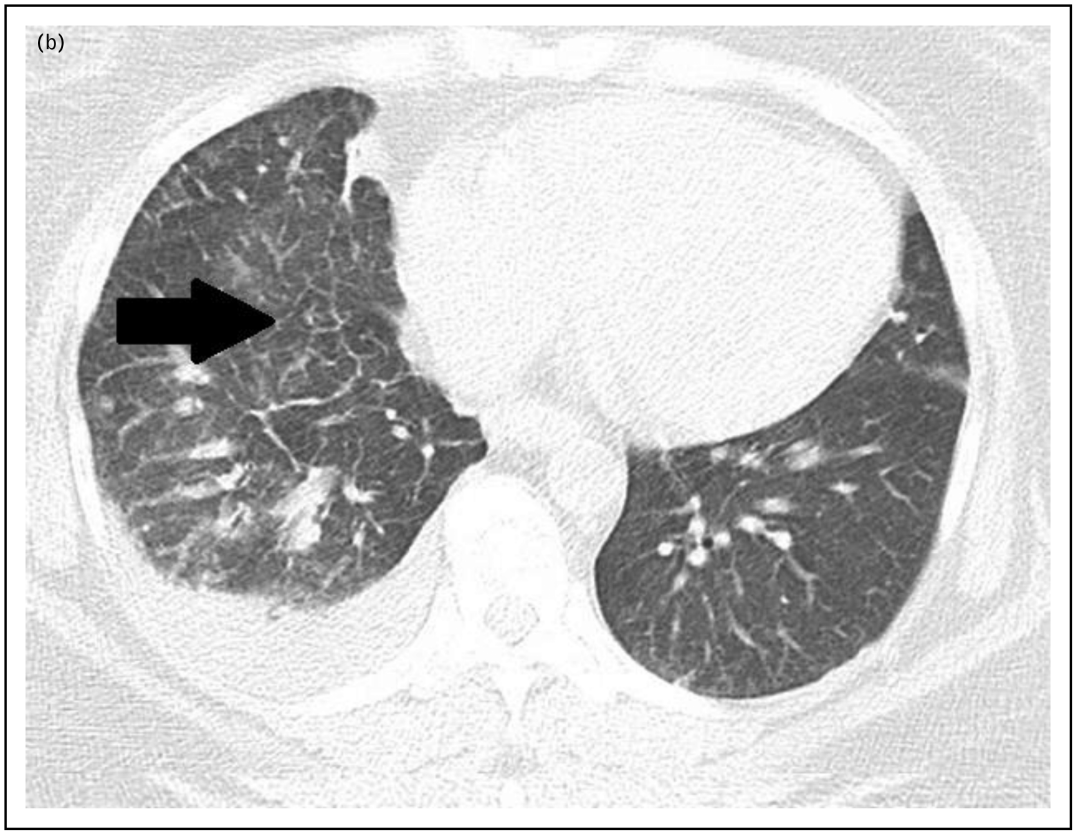

FIGURE 3. Continued.

longstanding infection [31,32]. The sensitivity of serology may decrease substantially in immunocompromised hosts [33]. The use of medications that have a greater impact on humoral immunity, e.g. rituximab, are of particular concern. Conversely, passive antibody transfer that occurs with transfusion of blood products including intravenous immunoglobulin in the peri- and posttransplant setting may confound the interpretation of serologic testing. In patients who experience hemodilution due to significant blood loss and subsequent transfusion, false negatives may occur [34]. Lastly, though antibody levels often gradually decline for several months after appropriate therapy, a detectable antibody titer, especially one collected soon after treatment, may not necessarily reflect persistent infection [35].

Given the association between HTLV-1 and *S. stercoralis* treatment failure, serologic screening for this viral pathogen is recommended in patients diagnosed with strongyloidiasis who have epidemiologic risk factors for coinfection [5].

Management

The significant risk of hyperinfection and dissemination from *Strongyloides* infection necessitates treatment of asymptomatic infection in transplant candidates and recipients. The recommended treatment for chronic, asymptomatic strongyloidiasis in transplant patients is oral ivermectin 200 μg/kg daily for two doses, repeated again after 14 days [25]. This treatment regimen of repeat doses after 14 days is based on the duration of one autoinfection cycle and ivermectin's more significant activity against the intestinal stages of *S. stercoralis* [25]. When considering the use of ivermectin in patients with epidemiologic risk factors for microfilarial coinfection, clinicians may consider the risk of precipitating complications including encephalopathy and the Mazzotti reaction [25]. If ivermectin is not available, a second-line regimen for chronic strongyloidiasis associated with a lower cure rate is oral albendazole 400 mg twice daily for a minimum of 10 to 14 days [25].

In transplant recipients with hyperinfection syndrome or disseminated disease, oral ivermectin at a dose of 200 μg/kg daily represents first-line therapy [25]. The treatment duration for severe disease is for a minimum of 2 weeks, but often until there has been evidence of 2 full weeks of negative stool examinations. In patients for whom oral therapy is not possible due to ileus, anatomic problems, or lack of enteral access, treatment with subcutaneous ivermectin is appropriate. A challenge of

subcutaneous therapy is procurement of ivermectin in this formulation and guidance for appropriate administration in humans. Subcutaneous ivermectin is licensed and frequently administered in veterinary practice. While licensed human formulations are not available, use of veterinary subcutaneous ivermectin under an investigational drug exemption granted through the Food and Drug Administration has facilitated use in humans with reported success [36]. Though specific human studies to guide dosing of subcutaneous ivermectin are not available, experts recommend 200 μg/kg once daily, similar to the dosing of the oral formulation. Rectal enema preparation and administration of ivermectin is also an option for patients unable to tolerate oral therapy, though efficacy data are limited [37,38]. Guidelines establishing target serum concentrations of ivermectin for the treatment of *S. stercoralis* are not available. Therefore, while serum ivermectin levels may be measured by specialized laboratories, the interpretation of levels when assessing for toxicity or for therapeutic drug monitoring is problematic and can also be affected by hypoalbuminemia [39]. When ivermectin is given by nonenteral routes, co-administration of albendazole may be of benefit [40]. Some experts recommend dual therapy with ivermectin and albendazole in critically ill patients until clinical improvement is noted [25]. Combination therapy may be particularly reasonable in cases characterized by wide dissemination, especially when the central nervous system is involved, for several reasons. First, ivermectin is most active against the intestinal form of the parasite. Secondly, P-glycoprotein efflux pumps along the blood-brain barrier actively remove ivermectin from the nervous system, and ivermectin concentrations in cerebrospinal fluid have been reported to be much lower than serum levels [25,41,42]. Albendazole, on the other hand, achieves much higher cerebrospinal fluid levels and represents an important, established component of therapy for neurocysticercosis, another helminth infection of the nervous system [43].

Clinical improvement and clearance of parasite from sites of infection guide the duration of therapy in patients with severe disease. In patients with hyperinfection syndrome, treatment should continue for 7–14 days after documented clearance of larvae from all identified sites of infection [25]. For disseminated infection, antihelminthic therapy should continue for minimum of 14 days from the first negative microscopic exam. Patients with HTLV-1 coinfection often cannot clear the infection, in part due to HTLV-1 related impaired eosinophilic response, and therefore expert opinion also recommends repeat treatment every 2–4 weeks during periods of increased immunosuppression, such as treatment for rejection, for these patients. There may also be a benefit to repeat treatment during periods of increased immunosuppression for HTLV-1 uninfected patients as complete eradication of *Strongyloides* is difficult to confirm.

As with many infections among solid organ transplant recipients, reduction of immunosuppression is a key component of *Strongyloides* hyperinfection syndrome management. Immunosuppression, particularly corticosteroids, should be tapered to the degree possible while still maintaining allograft tolerance.

Continued surveillance by ova and parasite exam after completion of therapy and clinical resolution of infection may be prudent, particularly in patients on potent immunosuppression. The recommended frequency of such surveillance is not defined, and the patient burden of sample collection must be considered when outlining a surveillance plan. Monthly stool ova and parasite exam in the outpatient setting may be a reasonable frequency to detect persistent infection before symptom onset. Patients who remain hospitalized after the end of treatment for hyperinfection syndrome or disseminated strongyloidiasis are likely experiencing other acute or critical illnesses, and more frequent weekly surveillance may be justified.

Prevention

Strategies to prevent *S. stercoralis* primary infection or reinfection are well described and include use of closed-toe footwear and avoidance of contaminated water or food. In transplant candidates, pretransplant screening identifies patients who would benefit from therapy. Acknowledging the risk of poor outcomes associated with hyperinfection syndrome and the favorable adverse effect profile of ivermectin, some programs have recently shifted to universal pretransplant serologic screening for all SOT candidates [44,45].

Guidelines published through the American Society of Transplantation in 2019 offer an approach to targeted serologic screening in deceased donors with risk factors for strongyloidiasis [25]. Some organizations have employed a targeted strategy with success. However, a 2016 survey suggested that only 10% of organ procurement organizations (OPO) routinely screened deceased donors for *S. stercoralis* [46]. More recently, a 2021 survey noted that only 24% of OPO in the US screened deceased donors for this pathogen [47]. Multiple other factors may also limit the effectiveness of targeted donor screening. Targeted screening requires a detailed knowledge of *S. stercoralis* epidemiology, and some programs may accept organs without the direct

input of infectious disease specialists. Donor exposure history may be remote or unavailable as patients are often incapacitated and family may be unavailable. Clinicians may not consider acquisition of infection in areas of the United States. Lastly, geographic risk factors may be absent in cases transmitted via contaminated food rather than soil.

Acknowledging the challenges associated with a targeted approach to donor screening, the Ad Hoc Disease Transmission Advisory Committee (DTAC) recently approved a policy shift that mandates universal *S. stercoralis* serologic screening for all deceased donors [48▪]. Current available data suggest that prophylactic ivermectin is associated with favorable outcomes in patients who receive organs from seropositive donors [20]. Understanding that the DTAC plans to monitor for transmission events as organizations implement this new policy, it is reasonable to accept organs from seropositive donors with a defined recipient risk mitigation strategy. Clinicians should be aware of the possibility of both false positive and false negative serologic results in donors. Importantly, negative donor serologic screening does not preclude the possibility of DDI. Living donor programs may have the benefit of predonation infectious diseases consultation for donor candidates with thorough assessment of risk factors to follow a targeted screening approach. In the absence of such a framework, living donor evaluations may want to consider universal *Strongyloides* screening for candidates ultimately approved for organ donation.

CONCLUSION

Chronic infection with *S. stercoralis* can be a common infection among SOTRs and donors requiring a systematic approach to pretransplant screening, treatment, and posttransplant surveillance. Timely management of strongyloidiasis among organ transplant recipients can prevent devastating complications of disseminated disease. Limitations of currently available microbiologic tools for detection of strongyloidiasis require careful clinical consideration of risks and benefits during diagnostic evaluation and posttreatment monitoring, particularly in immunocompromised patients.

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Conflicts of interest

There are no conflicts of interest.

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- ▪ of special interest
- ▪▪ of outstanding interest

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This OPTN Ad Hoc DTAC briefing proposes an important policy shift to universal screening for Strongyloides for deceased donors which can lead to recipient treatment to prevent donor derived strongyloidiasis.