Review Article

Tropical Infections in the Context of Kidney Transplantation in Latin America

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Abstract. Reports on tropical infections among kidney transplant (KT) recipients have increased in recent years, mainly because of the growing number of KT programs located in tropical and subtropical areas, and greater mobility or migration between different areas of the world. Endemic in emerging and developing regions, like most countries in Latin America, tropical infections are an important cause of morbidity and mortality in this population. Tropical infections in KT recipients may exhibit different pathways for acquisition compared with those in nonrecipients, such as transmission through a graft and reactivation of a latent infection triggered by immunosuppression. Clinical presentation may differ compared with that in immunocompetent patients, and there are also particularities in diagnostic aspects, treatment, and prognosis. KT patients must be screened for latent infections and immunized properly. Last, drug–drug interactions between immunosuppressive agents and drugs used to treat tropical infections are an additional challenge in KT patients. In this review, we summarize the management of tropical infections in KT patients.

INTRODUCTION

Infections are the main cause of death in kidney transplant (KT) recipients living in emerging and developing countries.^{1–3} Even in developed countries, where cardiovascular events are the main cause of death, infections are among the three leading causes of mortality.⁴ The increasing number of published reports on tropical infections in KT patients is remarkable. The growing number of transplant programs in emerging and developing countries in tropical and subtropical areas such as Brazil, Argentina, Mexico, India, Africa, and China partially justifies this higher incidence.⁵

In addition, improvement in patients' life expectancy and better quality of life results in greater mobility or migration between different areas in the world, with consequently greater exposure to environmental risks. Last, transplant tourism also contributes to this scenario.^{6,7} Most tropical infections are considered neglected diseases by the WHO and occur mainly in countries with fragile health systems, resulting in inadequate surveillance strategies and low notification rates. Given this big picture, the actual incidence of tropical infections in these countries must be greater.

In this setting, a better understanding of the interactions between tropical infections and KT has a pivotal role in improving health care and patient outcomes. This review aimed to address the peculiarities of the occurrence of tropical infections in Latin America in the context of kidney transplantation.

PATHWAYS OF TRANSMISSION

The transmission of tropical infections in the KT scenario may occur via three pathways: 1) *de novo* infection, 2) reactivation, or 3) donor-derived infection. *De novo* infection is defined as the direct acquisition of infection from the community of transplanted patients who had never been in contact with the pathogen before, or the re-exposure to the same

pathogen in previously exposed and properly treated patients. This pathway is more common after the first year after KT, when patients are fully recovered and engaged in their regular activities, such as jobs and social life. Similar to the general population, it occurs frequently among people living in or visiting tropical areas.^{5,6}

Another mechanism is reactivation or recrudescence of latent diseases, such as tuberculosis (TB), severe forms of strongyloidiasis, Chagas disease, leishmaniasis, and malaria. This phenomenon is more frequent during the first 6 months after KT and is a result of the intense immunosuppressive state that occurs during the early posttransplant period.^{5,6} Less commonly, tropical infections can be acquired by transmission from the donor through the allograft. Clinical presentation is usually early, but may occur at any time after KT. Chagas disease, TB, malaria, and human T-cell leukemia virus type 1 (HTLV-1) are examples of reported donor-derived infections.^{5,8,9}

PECULIARITIES OF CLINICAL PRESENTATION AND TREATMENT

The diagnosis is sometimes more difficult in KT patients than in immunocompetent patients, because the immune response is modified by immunosuppressive drugs, resulting in atypical presentation.¹⁰ Common findings, such as eosinophilia in strongyloidiasis, granulomas in TB and schistosomiasis, cyclical fever in malaria, and organomegalies in visceral leishmaniasis, can be absent.^{11–15} Furthermore, malaria, Chagas disease, and strongyloidiasis may exhibit a more aggressive and catastrophic evolution in KT patients, with a high risk for severe organ damage and death.^{5,12} Although some evidence has suggested that KT patients are less likely to develop severe dengue, depending on intense T-lymphocyte activity,^{6,9} a recently published systematic review reported greater incidences of severe dengue and greater mortality.¹⁶

The available diagnostic assays should be interpreted carefully, and false-negative results are common when serological methods are used because seropositivity may be delayed. Hypersensitivity-dependent skin tests, useful for the diagnosis of latent TB infection (LTBI) (Mantoux reaction or tuberculin

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skin test [TST]) and leishmaniasis (Montenegro test), are T-cell-dependent reactions and frequently reveal false-negative results. Therefore, interferon-gamma (IFN- γ) release assays (IGRAs)—blood tests that measure T-cell release of IFN- γ after stimulation with antigens—have emerged as an interesting alternative. They are also available for LTBI diagnosis and may eventually be promising in other diseases in which the immune memory is reached by an antigen reexposure.¹⁷ Last, for high-risk patients without a conclusive diagnosis, empirical treatment is often justified, especially in endemic countries, as well as when severe forms are present.^{18,19}

Renal allograft dysfunction is commonly detected upon diagnosis or throughout the treatment, with the main causes being hypovolemia, dehydration, sepsis, iodate contrast nephrotoxicity, and drug nephrotoxicity, with amphotericin B being a typical example.²⁰ Some drugs, such as azole derivatives used to treat fungal infection (fluconazole, itraconazole, and voriconazole), inhibit P450 cytochrome enzyme activity, leading to a greater exposure to calcineurin inhibitors (CNIs) and enhancing its nephrotoxicity. In contrast, rifampicin, used to treat TB, significantly increases P450 cytochrome activity, reduces CNI exposure, and favors rejection episodes. Drugs that inhibit or enhance P450 cytochrome activity may also influence exposure to steroids and the mammalian target of rapamycin inhibitors, resulting in toxicity or acute/chronic rejection.

Other causes of allograft dysfunction include the following: local inflammatory and fibrotic activities caused directly by the infectious agent, as described in renal TB and schistosomiasis; rhabdomyolysis in leptospirosis; acute interstitial nephritis in leptospirosis and leishmaniasis; and glomerulonephritis secondary to schistosomiasis, leishmaniasis, and malaria.^{18,20,21}

PECULIARITIES OF THE MOST FREQUENT TROPICAL INFECTIONS IN KIDNEY TRANSPLANT PATIENTS

In this section, we present the peculiarities of the most frequently reported tropical infections in KT patients in Latin America: TB, Chagas disease, leishmaniasis, arboviral diseases, and strongyloidiasis. Other relevant tropical infections, such as malaria, schistosomiasis, and leptospirosis, despite being endemic in this region, have rarely been described in KT patients and are not addressed here. Table 1 summarizes the central information discussed here.

Tuberculosis. The risk of TB infection after organ transplantation is 20 to 50 times greater than that in the general population, even in developing countries.⁹ In Brazil, an endemic area, the incidence of TB is 31 cases per 100,000 people in the general population and 1% to 5% in KT recipients.^{19,22} The three pathways of transmission have been reported; reactivation of LTBI is the most common, followed by community acquisition and, more rarely, donor transmission.²³

As in the general population, pulmonary forms are predominant, regardless of the region's endemicity. However, extrapulmonary and disseminated diseases are more common in KT patients than in immunocompetent individuals. Patients often present with atypical clinical symptoms, even those with typical pulmonary involvement.^{18,19,24} Not rarely, fever of unknown origin is the only symptom, and a high clinical suspicion is necessary, mainly in patients living in endemic areas.^{18,19} Treatment of active TB is based on recommendations for the general population and includes isoniazid, rifampicin, pyrazinamide, and ethambutol. Regarding the

main cautions in this population, it is often necessary to adjust the drug dosage for renal function, as hepatotoxicity is more common,^{25,26} and CNI exposure should be monitored closely. Changing rifampicin for rifabutin (a weaker inducer of cytochrome P450) has been suggested for patients with localized and non-severe forms of TB.²⁷ Some authors have suggested that rifabutin could be an alternative to replace rifampicin in patients with more severe disease.²⁸

Latent TB is the prolonged suppressed state of infection that results from a persistent immune response caused by *Mycobacterium tuberculosis* antigens, in the absence of clinical signs/symptoms.²⁹ Chronic kidney disease patients with LTBI are at a greater risk for developing active TB after KT when they are administered immunosuppressive drugs.^{29–31} Active TB is characterized by the presence of signs/symptoms and can affect graft and patient outcomes negatively, including a greater risk for kidney dysfunction and mortality.^{18,29,30} Therefore, efforts should be made to prevent TB. In high-endemicity areas, clinical suspicion should be high, and pre-transplant donor and recipient evaluation should be performed carefully.

All KT candidates should be screened for active TB by careful evaluation of clinical signs and symptoms. Screening for LTBI is also recommended and involves investigating the history of previously untreated or suspected TB, and near contact with patients with active infection, beyond TST or IGRAs and radiological chest exams.^{27,31,32} The cutoff for TST in this population is not clearly established, and induration of ≥ 5 mm has been suggested as positive.²⁷ Given the anergy of cellular immune response in patients with chronic kidney disease, a tuberculin test may reveal false-negative results. An IFN- γ release assay has emerged as an additional tool for improving screening sensitivity in this situation; however, the negative predictive value of IGRAs in high-risk patients is a concern, and pretest probability, including epidemiology and radiological findings, might be considered. Patients living in high-endemicity regions should undergo both the TST and IGRAs to maximize the sensitivity of detection.^{33–35}

The following options are recommended for LTBI treatment^{30,34}: 6 or 9 months of isoniazid, a 3-month regimen of weekly rifapentine plus isoniazid, 3 to 4 months of isoniazid plus rifampicin, or 3 to 4 months of rifampicin alone. When possible, LTBI treatment should be started before KT.

Graft transmission can be prevented through the screening of active or latent disease in donors, which is only possible in living donations. Active TB in living or deceased donor candidates contraindicates donation.^{34,35} Despite the lack of strong evidence, recipients of allografts from donors with untreated LTBI can be considered for preventive isoniazid chemoprophylaxis.^{36,37} LTBI treatment in potential living donors prior to donation is not clear; however, this might be considered for those living in high-endemicity countries.^{34–37}

Chagas disease. Chagas disease is one of the most severe protozoan infections after solid organ transplants—mainly, heart transplantation.^{13,38} The natural history involves an acute and a chronic phase. Acute infection occurs after an incubation period of 1 to 2 weeks following *Trypanosoma cruzi* transmission. Despite high parasitemia, the acute phase is mostly asymptomatic, mainly in immunocompetent patients, whose immune system controls parasitemia after a few weeks. After the acute phase, most patients progress to the chronic phase, characterized by low-level, intermittent

TABLE 1
Main tropical infections in the context of kidney transplantation

Disease	Frequency	Pathways of transmission	Clinical presentation	Diagnosis	Treatment	Additional comment
Tuberculosis	1–5%	Reactivation is the most common, followed by community acquisition and donor derived	LTBI: no signs/symptoms attributed to TB Active TB: pulmonary, extrapulmonary, and disseminated forms	LTBI: previously untreated or suspected TB, near contact with patients with active TB, radiological chest exams, TST, IGRAs Active TB: AFB positivity in fluids or tissues, <i>M. tuberculosis</i> detection in culture, PCR, suggestive histological findings	LTBI (KT recipients): 6 or 9H ₁ Active TB: 2EHRZ/4-7HR	It is suggested that one identify recipients with LTBI before KT to schedule prophylactic strategy. There is no clear recommendation for donors with LTBI. Donors and recipients with active TB have to be excluded for transplantation. Vigilance in seropositive patients and those receiving kidneys from seropositive donors after KT is recommended
Chagas disease	Identified in endemic areas or traveling patients; rare	Reactivation, donor derived, and vectoral transmission or by contaminated food in endemic areas	Symptomatic chronic infection: detected Ab and low-level parasitemia + cardiac/digestive disease Latent or indeterminate chronic infection: detected Ab and low-level parasitemia, but no evidence of organic lesion Acute form and reactivation: detected parasitemia and broad-spectrum clinical presentation of fever, myocarditis, inflammatory cutaneous lesions, CNS disease, and more	For Ab detection: serology For parasitemia detection: PCR is preferable; alternatively, xenodiagnoses or blood culture can be used.	Indicated when parasitemia is detected: benznidazole (preferable) or Nifurtimox for at least 2 mo	
Leishmaniasis	Not clear; rare even in endemic area	Vectoral infection, reactivation or, more rarely, infection by graft or blood transfusion	Visceral, cutaneous, and mucocutaneous; tegmental is uncommon	Presence of the parasite in smears, isolation of the organism in culture media, Montenegro skin test	Liposomal amphotericin B or pentavalent antimonials	Recurrence rate, 20–30%
Dengue	Unknown, probably underdiagnosed	De novo (mosquito bite); transmission by blood or graft seems to be possible, but never reported	Fever, arthralgia, myalgia, headache, diarrhea, ascites, pleural effusion, bleeding; severe forms are not common, except when occurring during the early-KT period	PCR, antigen testing, or serology	Supportive	It can be caused by four virus serotypes
Zika	Unknown, probably underdiagnosed	De novo (mosquito bite), sexual contact, transmission by blood or graft seems to be possible, but never reported	Scarce information is available	Serology can cross-react with Dengue; PCR might be helpful.	Supportive	Infected living donor must wait 120 d to donate kidney
Chikungunya	Unknown, probably underdiagnosed;	De novo (mosquito bite); transmission by blood	Fever, arthralgia, lymphopenia,	Serology or PCR	Supportive	It is recommended not to accept donors

(continued)

TABLE 1
Continued

Disease	Frequency	Pathways of transmission	Clinical presentation	Diagnosis	Treatment	Additional comment
Yellow fever	90% of cases diagnosed in Americans and published occurred in Brazil to be rare Unknown, appears to be rare	or graft seems to be possible, but never reported De novo (mosquito bite); unintentional live-attenuated vaccination; transmission by blood or graft seems to be possible, but never reported	thrombocytopenia; less severe than in immunocompetent individuals Fever, jaundice, bleeding, kidney and liver damage; high mortality rates	Serology or PCR	Supportive	until 30 d after the end of symptoms The vaccine is contraindicated after KT
Strongyloidiasis	10–40% in general population, exceeding 60% in low-income regions	Reactivation, de novo infection, donor derived	Acute or chronic intestinal forms, hyperinfection syndrome, disseminated form	Direct identification of larvae in stool or body specimens, serology, PCR	Ivermectin	Prophylaxis should be indicated for all patients in whom detection of the parasite occurred before KT. In the absence of routine parasite screening, prophylaxis should be prescribed, mainly in endemic regions. Mortality rates in severe forms: > 50%

2EHRZ/4-7HR = 2 months of ethambutol (E), isoniazid (H), rifampin (R), and pyrazinamide (Z) followed by 4 to 7 months of isoniazid (H) and rifampin (R); 6 or 9H = 6 or 9 months of isoniazid; Ab = antibodies; AFB = acid-fast bacilli; CNS = central nervous system; IGRAs = interferon-gamma release assays; KT = kidney transplant; LTBI = latent tuberculosis infection; PCR = polymerase chain reaction; TB = tuberculosis; TST = tuberculin skin test;
* Alternatives to LTBI treatment: 3-month regimen of weekly rifapentine plus isoniazid, 3 to 4 months of isoniazid plus rifampicin, or 3 to 4 months of rifampicin alone.

parasitemia. Notwithstanding maintaining positive serology assays, most chronically infected patients do not develop clinical symptoms, known as “indeterminate form” or “latent infection.” The rest of the patients who progress to the chronic form present organ damage, mainly with cardiac or gastrointestinal diseases.^{38,39}

Three transmission pathways have been described. De novo *T. cruzi* is acquired by a bite from kissing bugs, insects from the Triatominae subfamily, transfusion of contaminated blood, or consumption of infected food, such as an açaí pulp, an Amazonian fruit.^{39,40} The most common mode of infection after KT is reactivation, which can occur in 8% to 22% of seropositive recipients.^{41,42}

To avoid donor-derived infection, serological tests should be performed in all potential donors, mainly those living in countries where the disease is endemic. There is no consensus about routine serology performed in donors living in non-endemic regions. In the United States, for instance, less than 20% of donors in the last decade were screened for Chagas disease.⁴³

The risk of untreated KT recipients acquiring Chagas disease from seropositive donors might be considered. Untreated acute Chagas disease in KT recipients is associated with severe manifestations and high mortality rates. In some endemic regions, seropositive patients are not considered for organ donation.^{39,44} However, evidence suggests that when donor-transmitted Chagas disease is diagnosed and treated promptly, it is associated with acceptable outcomes.^{13,38,44}

In regions where KT with seropositive donors are allowed, strict laboratory monitoring and clinical surveillance should be adopted, and polymerase chain reaction (PCR) is the method of choice. Blood culture and xenodiagnoses are less sensitive and have a long turnaround time, hampering the timely detection of parasitemia.^{41,42,45} In settings where PCR is not available, surveillance should rely on the direct identification of the parasites in blood using blood smears or, preferably, microhematocrit or the Strout test. A suggested monitoring schedule, based on heart transplantation, is as follows: weekly in the first 2 months, biweekly between the third and sixth months, monthly up to year 2, and any period after immunosuppression intensification.³⁸ Preemptive treatment is only indicated if parasitic load is detected and there is no default threshold to start the treatment in asymptomatic recipients. Treatment is based on benznidazole (first option, better tolerability) or nifurtimox for at least 2 months.³⁸ Both drugs can interact with CNI, and immunosuppressive blood levels should be monitored closely. Regardless of whether reactivation occurs, tests should be performed weekly while on treatment until two negative results are obtained.³⁸ There are few reports describing the use of routine benznidazole prophylaxis for KT recipients of seropositive donors without parasitemia monitoring.⁴⁶

Chronically infected recipients should be considered at high risk for reactivation, mainly in the first year after KT, and should be monitored closely.³⁸ This risk after KT is less than that after heart transplantation; however, it must be considered. Monitoring and management of parasitemia is similar to that described earlier for KT using seropositive donors.⁴⁷

Leishmaniasis. Cellular immune response is critical in *Leishmania* infections. Immunocompetent patients who acquire this protozoan commonly remain asymptomatic. In contrast, immunosuppressed patients usually present symptomatic forms, including visceral (“kala-azar”), the most

frequently described clinical presentation; cutaneous; and mucocutaneous.^{48,49} The real incidence is not clear because it is an uncommon disease in areas with high transplantation activity. Even in endemic countries, reports are limited to single-center case series and case-control analysis.^{14,50} Pathways of transmission may involve acquisition by a vector, as in the general population; reactivation of latent infection; or, more rarely, transfusion with infected blood and donor derived.⁴⁸ If there is no evidence of an active infection, a careful epidemiological and clinical investigation must be performed in donors from endemic regions.

Patients with a previous clinical report of leishmaniasis should be monitored closely, because 20% to 30% of patients present with a recurrence after KT.⁴⁸ Secondary prophylaxis with a weekly dose of amphotericin B, monthly dose of anti-moniato, or daily dose of fluconazole can be considered, especially in the first year after KT.⁵¹ For patients traveling to endemic areas, using chemical or physical barriers for protection, such as mosquito repellents and special stockings, is recommended.

Some assays can be considered for diagnosis, depending on staff expertise and local availability, and include the Montenegro test, serology, direct examination of blood samples or bone marrow, or, more currently, PCR.⁵² Because the whole blood cells from patients with active visceral leishmaniasis secrete significant levels of IFN- γ and interleukin-10 when stimulated by soluble and specific antigens, IGRAs have emerged as a potential alternative, providing high specificity, but they are not yet used in routine clinical practice.⁵³ Liposomal amphotericin B is considered as the first-line treatment, and relapses are frequent.⁵⁴

Arboviral diseases. The most endemic arboviruses in tropical and subtropical areas are dengue, chikungunya, Zika, and yellow fever (YF), all of which have already been reported in KT recipients.^{55–59} Dengue, chikungunya, and Zika transmissions occur through a bite from *Aedes aegypti* or *Aedes albopictus* mosquito or, more rarely, through blood transfusion and grafts. Zika can also be transmitted through sexual contact and perinatally.⁶⁰

Dengue can be caused by four virus serotypes, and infection by one serotype does not provide immunity against others. There are a lot of reports and case series on dengue after KT, and it is likely that this infection is underdiagnosed because most cases are oligo- or asymptomatic.^{61,62} Symptomatic infection ranges from mild flu-like symptoms to infection with warning signs, such as abdominal pain, persistent vomiting, mucosal bleed, and lethargy; severe dengue is characterized primarily by plasma leakage with or without hemorrhage or severe organ impairment.⁹ Some evidence has suggested that KT patients are less likely to develop severe dengue.⁹ However, a recently published systematic review reported that pleural effusions and ascites were more frequent in that population, and a greater incidence of severe dengue and higher mortality rate was observed.¹⁶ Two cases of severe dengue were described in the early postoperative period, suggesting greater severity when the patient undergoes transplantation during the incubation period.⁶³ Diagnosis can be confirmed by antigen testing, PCR, or serology depending on the time of symptoms; antigen testing and PCR are used in the early stages and serology in the later stages of the infection.⁶⁴ Treatment is limited to supportive care.

Zika infection may result in neonatal microcephaly, neurological complications, and Guillain-Barré syndrome in the general population.⁶⁵ Diagnosis can be achieved via serology, and treatment is restricted to supportive measures. Thus far, there are only a few reported cases of Zika after KT, both occurring during the late post-transplant period and with favorable outcomes.⁶⁶ Living donor candidates with a history of Zika infection should wait at least 120 days after infection resolution to donate.⁶⁷

Currently, Brazil accounts for more than 90% of confirmed cases of chikungunya fever in the Americas.⁶⁸ After a mosquito bite, viremia can extend up to 3 weeks.⁶⁵ Recent reports have shown that patients receiving steroid therapy usually exhibit less severe disease; to date, only a few case reports and case series on chikungunya after KT have been published.^{69–72} Fever, arthralgia, lymphopenia, and thrombocytopenia are the most common manifestations.⁶⁸ Arthralgia, often affecting large and symmetric joints, is milder and has a shorter duration compared with immunocompetent individuals^{71,73,74}; no negative impact on graft outcomes has been described.⁷¹

The diagnosis can be confirmed through serology (IgM), but PCR is the gold standard. Treatment is usually restricted to symptom-based management.⁷⁵ It is recommended not to accept donors until 30 days after the symptoms end.⁷⁶

YF is considered one of the most severe arboviral infections and is associated with high mortality rates, independent of the baseline patient characteristics. There are two epidemiological cycles of YF disease: wild and urban. Vectorial transmission of the wild form occurs through the bite of *Sabethes* and *Haemagogus* mosquitoes. The urban form is transmitted by *Aedes aegypti*. Approximately 90% of all cases are in Africa, where both forms have been identified; the urban form is considered to have been eradicated in America.⁵⁶

In the elderly, children, patients with organ impairment (heart, lung, liver, or kidney), and those undergoing immunosuppressive therapy usually have worse prognosis for YF.⁵⁹ In the general population, peak viremia occurs 3 days after transmission, and after 10 days on average, patients present with fever, jaundice, hemorrhages, and kidney and liver damage.^{56,59} In a recent Brazilian YF outbreak, a myriad of organ and tissue disorders were observed, including fulminant hepatitis, coagulation disorders and bleeding, acute kidney injury, systemic vasculopathy, pancreatitis, intestinal ischemia, splenitis, hemophagocytosis, and immune dysfunction.⁷⁷ Two regions reported their experience with liver transplantation to manage fulminant hepatitis.^{77,78} Because there are only a few reports on YF after KT, there is no robust information on the clinical presentation and prognosis in this population. To date, no blood or graft transmission has been reported in this population.^{58,59} Treatment is based on supportive measures. The available vaccine is live attenuated and, therefore, contraindicated in immunocompromised patients.⁷⁹

Strongyloidiasis. Infection by *Strongyloides stercoralis* is endemic in some tropical regions, although its real prevalence has varied greatly among different countries. The global prevalence is heterogeneous, and evidence suggests that it can affect 10% to 40% of the population in many tropical and subtropical countries, exceeding 60% in low-income countries, where ecological, sanitary, and social situations are fragile.⁸⁰

Reactivation is the most frequent pathway of transmission, but de novo acquisition and transmission by grafts have also been reported.^{81,82} Clinically, these infections are classified

into four forms: acute intestinal form, chronic intestinal form, hyperinfection syndrome, and disseminated strongyloidiasis.⁸³ The acute intestinal presentation is characterized by nonspecific abdominal pain, followed by symptoms in the upper or lower gastrointestinal tract such as nausea, vomiting, or diarrhea, and is associated sporadically with cutaneous lesions and lung symptoms (Löfller syndrome). The chronic intestinal form is more frequent and is usually confounded by functional dyspepsia or irritable bowel syndrome. Few patients may progress to severe presentations, followed by paralytic ileus, intestinal obstruction, or gastrointestinal bleeding. These severe intestinal cases associated with severe lung manifestations such as dyspnea and hemoptysis are known as hyperinfection syndromes, which involve amplification of the natural cycle of the parasite and are more common in immunosuppressed patients. In these three forms (acute and chronic intestinal forms and hyperinfection syndrome), larvae are only found in organs involved in the infection cycle. Last, in disseminated strongyloidiasis, patients commonly present with systemic complications such as meningitis, cholecystitis, liver abscess, pancreatitis, or septic shock; thus, larvae are found at sites different from those during their natural cycle.⁸⁴

Severe forms are associated with mortality rates reaching 50%, especially during the early post-KT period or after acute rejection treatment, when high-exposure immunosuppression is used.⁸⁵ A recent multicenter Brazilian study reported a 30-day all-cause mortality rate of 28.3%, and Gram-negative bacteremia and respiratory failure were predictors of mortality.¹⁵

The diagnosis is based primarily on the isolation of *S. stercoralis* from biological samples: stool, duodenum aspirate, and other body fluids.⁸⁶ Because the sensitivity of this method is not ideal, serological assays and molecular biology techniques (PCR) have been suggested as alternatives or additional tools.^{87,88} Treatment is preferably accomplished with ivermectin for 2 days, and a second course can be indicated 1 or 2 weeks later. In patients with severe forms, treatment should be continued until symptoms improve and serial stool samples are negative for 2 weeks.⁸⁴

Routinely, *Strongyloides* screening and treatment should be considered as a strategy to prevent both recipient- and donor-derived strongyloidiasis. Available screening strategies are the parasitological stool sample exam and serology, both with limited accuracy.^{89,90} Preemptive treatment should be instituted for all KT recipients with positive serology or larvae in the stools assessed prior to KT. In endemic countries or in patients transplanted with unknown *Strongyloides* status, universal prophylaxis might be considered. Treatment is preferably indicated with ivermectin for 2 days 2 weeks prior to KT in cases of scheduled transplants (living donor transplants), or shortly after surgery in nonscheduled KT (deceased donor transplants). It is also indicated after treatment of acute rejection or when intensification of immunosuppression for any other reason is required.⁸⁴

GENERAL ASPECTS OF PREVENTION AND OTHER SPECIFIC SITUATIONS

Strategies for the prevention of infectious diseases in KT patients can be divided into three categories: 1) prevention of donor-derived transmission, 2) prevention of post-

transplant reactivation of latent infections, and 3) prevention of infections acquired in the community. During the pre-transplant clinical evaluation, two strategies should be used: screening for selected infectious diseases and updating the vaccination schedule. The immunization schedule must be updated in all KT candidates according to local recommendations. There is no robust information on the safety of YF immunization in patients undergoing dialysis or on a waiting list. A single-center Brazilian study reported only minor adverse events in 24% of 45 patients undergoing chronic dialysis who received the YF vaccine, suggesting that it is likely the vaccine is safe in this population.⁹¹ Thus, KT candidates should receive the YF vaccine before transplantation and wait for at least 8 weeks before undergoing transplantation.

It is important to mention infection by HTLV-1, which can cause tropical spastic paralysis, neurogenic bladder, and T-cell lymphoma, and that transmission via the graft can be expected. In some countries, HTLV-1 serology for kidney donors is no longer recommended because it is often false positive, and the probability of unexpected donor-derived transmission is low. However, donor-derived HTLV-1 has already been described and can be associated with significant morbidity.^{92,93} Therefore, HTLV-1-positive serology must be verified using more specific methods such as Western blot or PCR; and, if infection is confirmed, potential donors must be excluded. There is no standardized recommendation for follow-up in seropositive recipients or for patients infected inadvertently by graft transmission.⁹⁴

Regarding malaria, candidates for donation with active infection must be excluded, and in suspected cases, diagnostic confirmation via blood smear or PCR testing is recommended. Patients traveling to endemic areas should be advised of prophylactic environmental exposure measures, and, in some cases, chemoprophylaxis with doxycycline, chloroquine, mefloquine, or atovaquone may be indicated, and patients should be monitored closely for the potential interaction between these drugs and immunosuppressive agents.⁹⁵

There are no specific prophylactic recommendations to prevent dengue, Zika, and chikungunya fever. People who live in endemic areas or travel to these areas must be strongly encouraged to use mosquito repellents and physical barriers.

FUTURE PERSPECTIVES AND CONCLUSIONS

Tropical infections in KT patients have begun to receive great interest, mainly in regions such as Latin America, where transplant programs have improved. In contrast to the typical diseases that usually affect this specific population, tropical infections are more frequent in countries where there are particularly weak points in the public health system. Therefore, worldwide efforts to share advancement in strategies for case notification and, more importantly, for diagnosis are necessary. Furthermore, actions focused on prophylaxis must be improved and expanded, including the development of effective and safe vaccines, chemoprophylaxis when indicated, and public information. Considerable attention must be given to the anti-vaccination movement, which is a harmful new movement responsible for the decline in the number of vaccinated people worldwide. Last, further research aimed at developing new vaccines, new

prophylaxis strategies, and more sensitive diagnostic techniques should be encouraged in rich and poor countries alike.

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