Other Life-Threatening Infections

39

Rodrigo Martino

39.1 Toxoplasmosis

39.1.1 General Concepts

Toxoplasma gondii is a protozoan that commonly infects animals and birds. Primary *T. gondii* infection in humans and other mammals is usually asymptomatic but leads to lifelong latent infection. Transmission to humans occurs by ingesting tissue cysts from undercooked meat or oocysts (released in the feces of cats). Latent cysts can give rise during immunosuppression to a severe localized reactivation producing, for example, toxoplasma encephalitis or chorioretinitis, with dissemination being common. (Martino et al. 2000; Martino et al. 2005; Tomblyn et al. 2009; Martino 2016).

Although toxoplasmosis is the most common systemic parasitic infection in EBMT centers, it is a relatively rare opportunistic infection following HSCT. Currently we are aware that the patients' seroprevalence explains the wide range of incidences published. Table 39.1 summarizes selected case series of toxoplasmosis in HSCT published to date.

39.1.2 Risk Factors and Incidence in HSCT

The seroprevalence for *T. gondii* varies greatly between and even within countries, ranging from <15% in Japan and in pediatric wards, 30% in urban adults in North America and the UK, and to 40–80% of adult HSCT recipients in countries with high endemicity such as France or Turkey. This varying seroprevalence is the main reason for the great variability in the incidence of toxoplasmosis after HSCT, which has been estimated to average 0.8%, with <0.4% in areas of low endemicity to 2–3% in those with high-antibody prevalence.

Toxoplasmosis occurs mainly in allo-HSCT recipients, although cases after auto-HSCT have been published. Reactivation of latent tissue cysts in previously infected individuals is the usual mechanism implicated. Thus, it is important to determine the patients' serostatus prior to transplant. However, the disease may also develop if primary (or re-)infection after transplant may occur.

Ninety-five percent of the cases occur within the first 6 months after the procedure, and acute GVHD and its treatment are the main risk factors. Late cases may occur, again usually in patients with chronic GVHD requiring IST. In addition, seropositive patients without GVHD but with severe cellular IS due to in vivo or ex vivo TCD are also at risk.

R. Martino (✉)
Hospital de la Santa Creu I Sant Pau, Autonomous University of Barcelona, ES, Barcelona, Spain
e-mail: rmartino@santpau.cat

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Table 39.1 Selected case series of toxoplasmosis after HSCT (Martino 2017)

Author (year)[a]	Cases	Number of HSCT (% frequency)	% of sero (+) pre-HSCT	Median (range) day onset
Derouin et al. (1992)	7	296 allo (2.4)	65	74 (55–180)
Slavin et al. (1994)	12	3.803 allo (0.31) 509 auto (0)	15	59 (35–97)
Bretagne et al. (1995)	2	550 allo (0.3)	70	NS
Maschke et al. (1999)	20[b]	655 (3.1)	NS	73 (14–689)
Martino et al. (2003)	41	4.391 allo (0.93) 7.097 auto (0)	Variable (multinational study)	64 (4–516)
Small et al. (2000)	10	463 allo (2.2)	23	78 (36–155)
Aoun et al. (2006)	7	121 allo (5) 204 auto (0.4)	69	45 (13–140)
de Medeiros et al. (2001)	9	789 allo-HSCT (1.14)	NS	69 (13–265)
Mulanovich et al. (2011)	9	3.626 Allo (0.25) – U.S. pt 0.15% – Non-U.S. pt 1.6%	18% U.S. pt >50% non-U.S. pt	56 (12–122)
Bautista et al. (2012) and Martino et al. (2015)	9	148 adult CBT (4%)	45	39 (7–98)
Sumi et al. (2013)	6	279 allo (1.8%) 87 auto (1.1%)	10	NS
Hakko et al. (2017)	5	170 allo (2.9%)	70	42 (26–119)

[a]Not all in references
[b]4 definite and 16 possible cases of toxoplasmosis

39.1.3 Most Common Clinical Presentations

The CNS is the main site of disease, but pneumonitis and myocarditis are also frequent findings.

Toxoplasma encephalitis typically presents with focal neurologic abnormalities of subacute onset, frequently accompanied by non-focal signs and symptoms such as headache, altered mental status, and fever. Meningeal signs are very rare. CT brain scans often show multiple bilateral cerebral lesions, although MRI is more sensitive than CT in the early diagnosis of this infection. Toxoplasma pneumonitis may develop in the absence of extrapulmonary disease. Toxoplasma chorioretinitis is rare compared to AIDS patients.

39.1.4 Diagnosis

In HSCT recipients, the utility of serology is mainly to identify those at risk for developing toxoplasmosis post transplant.

PCR techniques are currently the standard method for its diagnosis. These techniques are applicable in blood, CSF, and BAL, the usual samples that are available in HSCT recipients with this infection. Most centers use qPCR with a level of detection as low as 20 parasites/mL, with parasite loads of >600/mL reported in most patients with toxoplasmosis.

Since histologically proven toxoplasmosis is a very difficult-to-obtain diagnosis, various levels of diagnostic certainty have been proposed. Histologically defined cases are considered as definite cases of toxoplasma disease, PCR-defined cases as probable, and CNS imaging-defined cases as possible ones.

39.1.5 Treatment and Prognosis

Table 39.2 details the recommended treatment and prophylaxis of toxoplasmosis in HSCT recipients. Most patients respond to one or another of these regimens, and neurologic improvement of toxoplasma brain involvement usually occurs within 7 days. If appropriately treated, up to 60% of patients may show clinical–radiologic improvement or even a complete response to therapy. This highlights the importance for a high index of suspicion for toxoplasmosis in immunocompromised patients.

39.1.6 Specific Screening and/or Prophylactic Strategies Available

Current data suggest that infection may precede disease in most cases of toxoplasmosis. Thus, monitoring sero(+) patients with weekly qPCR of blood samples has been advocated, especially when prophylaxis is not being used, in an effort of using a preemptive-type therapeutic approach, as used for CMV infection. Although an optimal qPCR technique has not been standardized, several studies support the usefulness of this approach. Patients on TMP/SMX prophylaxis should not be monitored.

TMP/SMX is useful in minimizing the risk of reactivation of toxoplasmosis, although there are well-reported cases of toxoplasmosis breaking through this prophylaxis in HSCT recipients. Suboptimal dosing may have contributed to some of these "breakthrough" infections, since these cases occur when TMP/SMX is taken less than 3 days per week. Thus, using either one standard-dose tablet (80/400 mg) daily or a double-strength tablet (160/800 mg) 4 days per week is the recommended dosing, as shown in Table 39.2.

Avoiding primary or reinfection after HSCT is always important, avoiding the most common sources of infection: uncooked meats of any type and drinking contaminated water.

Table 39.2 Suggested treatment and prophylaxis for toxoplasmosis in HSCT recipients

Treatment	Dose
Pyrimethamine (*plus* folinic acid)	Oral, 200 mg loading dose, then 50–75 mg q.d. (folinic acid, oral or IV, 10–15 mg q.d.) + *one of the following*
Sulfadiazine	Oral, 1–1.5 g q6–8h, *OR*
Clindamycin	Oral or IV, 600 mg q6h
Prophylaxis	Dose
TMP/SMX[a,b]	1 double-strength tablet (160/800 mg)/day, 4 day × week, *OR* 2 double-strength tablets (160/800 mg)/day, 3 day × week, *OR* 1 standard-dose tablet (80/400 mg) daily, *OR*
Pyrimethamine and sulfadoxine (fansidar)[a]	2–3 tables per week
Dapsone[b]	100 mg daily
Atovaquone[b]	1500 mg daily

[a]Also effective for PJP prophylaxis, and possibly listeriosis, nocardiosis, and, in some geographic areas, partly effective in preventing gram-positive cocci and gram-negative bacillary (enterobacterial and non-glucose fermenting) infections

[b]The dose can be reduced in patients with mild renal insufficiency

39.2 Tuberculosis (TBC)

(de la Cámara et al. 2000; Cordonnier et al. 2004; Yao-Chung et al. 2016; Young and Weisdorf 2016; Beswick et al. 2018).

39.2.1 General Concepts

TBC, and especially, multidrug-resistant (MDR) TBC, continues to be a worldwide major health problem. This may surprise many EBMT HSCT physicians, who may have never seen a case of TBC.

39.2.1.1 Mycobacterium Tuberculosis

Mycobacterium tuberculosis causes nearly all cases of TBC, and these acid-fast bacilli differ from other bacteria in that they can live only in an infected human. Outside of the human body, they have a very short survival, and infection is transmitted by the inhalation of aerosolized particles from a patient. In addition, its isolation from clinical samples should never be considered as a colonization or sample contamination. TBC is not an opportunistic infection, and thus its detailed description is outside the scope of this manual.

39.2.2 Risk Factors and Incidence in HSCT

The risk of developing TBC is directly proportional to the TBC present in the geographic area of the HSCT center and the patients' residence (Fig. 39.1). A few studies have analyzed its incidence with respect to the general population, and most have found that allo-HSCT recipients have 2–10 times higher risk than the general population, while auto-HSCT recipients do not have a significantly higher risk (De la Cámara et al. 2000) (Table 39.3).

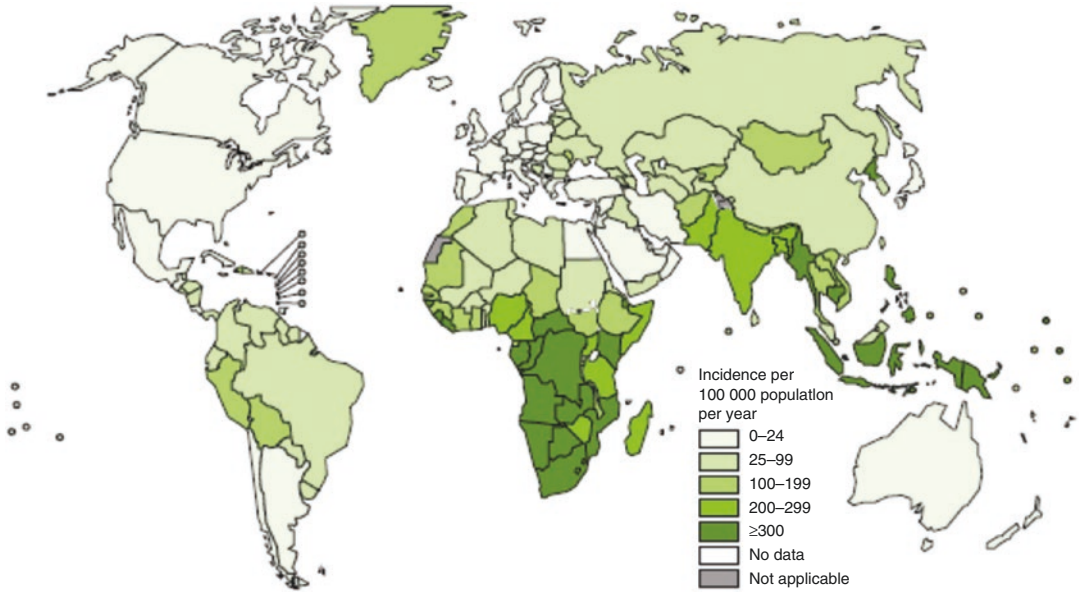

Fig. 39.1 TBC estimate incidence rate 2016 (WHO webpage)

Table 39.3 Selected case series of mycobacterial infections after HSCT before 2018

Author (year)[a]	Country	TBC/HSCT x risk with GP[b]	NTM/HSCT x risk with GP	Outcome of infection
Lee et al. (2017)	Korea	21/824 (allo) × 9.1 GP	NA	1 died
Liu et al. (2016)[c]	Taiwan	5/422 (allo)	21/422 (allo)	11 died
Beswick et al. (2018)	Canada	NA	30/1097 (allo) × 35 GP	NA
Fan et al. (2015)	Taiwan	32/1368 (allo) × 7 GP 7/672 (auto) × 2.5 GP	0	20 died
Garces-Ambrossi et al. (2005)	USA	4/577 (allo) × 10 GP	0	NA
Cordonnier et al. (2004)	Multiple (EBMT)	23/1513 (allo) 8/3012 (auto)	8	5 died
Ku et al. (2001)	Taiwan	8/255 (allo) × 13.1 GP 0/95 (auto)	0	ND
de la Cámara et al. (2000)	Spain	12/2866 (allo) × 2.2 GP 8/5147 (auto) = to GP	NA	3 died
Budak-Alpdogan et al. (2000)	Turkey	5/351 (allo) × 3.9 GP	0	No deaths
Gaviria et al. (2000)	USA	3 /6529 (3 allo)	0	No deaths
Aljurf et al. (1999)	Saudi Arabia	4/641 (allo)	0	2 died
Roy et al. (1997)	USA	2/1486 (allo) 0/755 (auto)	7/1486 (allo) 2/755 (auto)	No deaths
Martino et al. (1996, 2011)	Spain	2/698 (allo) 0/637(auto)	0	No deaths

NA Details not available in the study
[a]Not all in references
[b]x risk with GP, studies in which the relative risk of suffering TBC was compared to that in age-/sex-matched normal individuals from the general population
[c]Abstract

39.2.3 Most Common Clinical Presentations

The clinical presentation of TBC in HSCT recipients is the same as in the general population, although it may have a more rapid progression, and the ratio of pulmonary to extrapulmonary disease has been reported 34/5 to 11/10, which surely represents a publication bias, with a median of 75%/25%. The most common extrapulmonary disease is meningitis.

39.2.4 Diagnosis

Culture of even a single colony from an affected organ is diagnostic for TBC. Direct microbiologic examination for acid-fast bacilli is of course mandatory, but its sensitivity is probably low. In addition, the results of positive cultures take many days to weeks, and the use of highly sensitive and specific PCR methods is now the usual methods for the initial diagnosis.

The quantiFERON-TB Gold test is not reliable in the diagnosis of TBC in HSCT recipients due to their T-cell immunodeficiency.

39.2.5 Treatment and Prognosis

With appropriate treatment, TBC in HSCT recipients has a low attributable mortality (<30%). The author suggests that HSCT physicians contact ID physicians immediately when diagnosis of TBC is made. Empirical treatment should be started if this consultation will not be replied immediately, but herein we cannot recommend a "one fits all" drug combination, since this varies greatly according to the level of drug resistance in each geographical area.

39.2.6 Specific Screening and/or Prophylactic Strategies Available

Even in areas where TBC is endemic, pre-HSCT screening with the tuberculin skin test or the gamma-interferon quantiFERON-TB Gold test is not done in most HSCT centers. In addition, specific antibiotic prophylaxis in patients with past and cured TBC is not warranted.

Two special scenarios do, however, require contacting an ID specialist pre- or post-HSCT in order to analyze whether "prophylaxis" may be indicated, the drugs to use, and their duration:

1. Highly IS HSCT recipients or candidates who have been substantially exposed to someone with active infectious TBC
2. HSCT recipients or candidates with a positive tuberculin skin test or the gamma-interferon quantiFERON-TB Gold test who were not previously treated and have radiological evidence of TBC lung disease

39.3 Nontuberculous (or Atypical) Mycobacterial (NTM) Infections

(Cordonnier et al. 2004; Young and Weisdorf 2016; Beswick et al. 2018).

39.3.1 General Concepts

Atypical mycobacteria are fastidious microorganisms that are ubiquitous in nature and can simply colonize any body surface and secretions and often contaminate clinical samples from the environment. There are a very large number of NTM species with varying geographical distributions. However, with respect to infections in HSCT recipients, NTM can be divided into two different categories:

(1) *Mycobacterium avium-intracellulare* complex.
(2) Anonymous or atypical NTM, subdivided into the rapidly growing NTM and the slow growing NTM: the most commonly reported species from EBMT centers are *M. fortuitum, M. chelonae, M. abscessus, M. xenopi, and M. kansasii*.

39.3.2 Most Common Clinical Presentations and Risk Factor

A large number of atypical NTM infections are CVC infections, followed by skin infections. However, in patients with severe cGVHD, severe infections of any organ can occur, as well as disseminated cases. *M. avium-intracellulare* complex, on the other hand, usually causes pulmonary disease or disseminated infections, with blood cultures being positive in >50% of cases. Such infections almost always occur in severely immunocompromised allo-HSCT recipients, such as those with severe steroid-dependent cGVHD.

39.3.3 Diagnosis

Diagnosis requires isolation of a NTM from the affected organ(s). Differentiating colonization from contamination and disease can be difficult with NTM. Depending on the species, cultures can be positive in very few days or take many days, as with TBC. Thus, the use of specific PCR methods and/or special biochemical methods is now the usual method for the diagnosis of NTM infections.

39.3.4 Treatment and Prognosis

With appropriate treatment, most NTM infections have a good outcome and a low attributable mortality, although the data are very scarce (Table 39.3).

As in the case of TBC, the author suggests that HSCT physicians contact ID physicians immediately when diagnosis of NTM infection is made. In CVC infections, the catheter should probably always be removed. While awaiting for the ID specialists, empirical therapy with a macrolide (clarithromycin or azithromycin) plus moxifloxacin or levofloxacin can be started.

39.3.5 Specific Screening and/or Prophylactic Strategies Available

Screening and prophylaxis have no role in NTM infections.

39.4 Listeriosis

(Safdar et al. 2002; Boyle 2014; Martino et al. 1996).

39.4.1 General Concepts

Only one species, *Listeria monocytogenes*, produces all cases of this mostly "bacterial foodborne" infection. *L. monocytogenes* is a pseudo-"diphtheroid" gram-positive bacillus. This organism is widespread in nature and in tap water, sewage, the microbiota of pets and farm animals, and nearly all types of fresh foods. The fact that it grows well in refrigerator temperatures adds yet another variable which favors ingestion by humans, which appears to be universal worldwide. At any specific moment, 5% of healthy humans have *L. monocytogenes* in feces. With these premises, it is surprising that listeriosis is an uncommon infection in HSCT recipients.

39.4.2 Risk Factors and Incidence in HSCT

The only risk factor is the combination of ingesting colonized food or water and having a severe cellular IS.

Its incidence is unknown, and only two studies are available. At the MSKCC in New York, six cases occurred in 1315 allo-HSCT recipients from 1985 to 1997, with an incidence of 0.47% (Safdar et al. 2002). At the FHCRC in Seattle, three cases occurred among 4069 HSCT recipients (<0.1%) during the first 100 days post transplant (Boyle 2014). Finally, in our center, we have had three cases of listeriosis among 2360 adult HSCT recipients (0.1%) (Martino et al. 1996). All other information has been reported as isolated case reports.

39.4.3 Most Common Clinical Presentations

Listeriosis in HSCT recipients is almost always a sepsis syndrome with bloodstream infection,

with CNS involvement in 40–60% of cases, which can present as meningitis, encephalitis, or brain abscess, and with several cases of rhombencephalitis reported (Chang et al. 1995).

39.4.4 Diagnosis

The diagnosis is made after the bacterial microbiology laboratory informs the clinicians that the patient has positive blood and/or CSF cultures for this organism. The putative source of the infection cannot be identified in outpatients.

39.4.5 Specific Screening and/or Prophylactic Strategies Available

Screening has no role in preventing listeriosis. Standard approaches to food safety handling and preparation are, of course, the main preventive measures.

The routine use of TMP/SMX prophylaxis after HSCT surely has a role in preventing listeriosis, but its low incidence makes this impossible to prove.

Cases of listeriosis in long-term inpatients should, of course, activate the rapid intervention of the hospital infection control/prevention unit in the HSCT ward.

39.4.6 Treatment and Prognosis

The treatment of choice is high-dose ampicillin (or high-dose TMP/SMX in those allergic to penicillin) combined with an aminoglycoside during 3 weeks or 6 weeks in case of CNS infection. We also recommend consultation with ID specialists.

The prognosis of listeriosis in HSCT recipients is unknown, although 20% of the reported cases died, while 10% had a CNS recurrence.

39.5 Nocardiosis

(Coussement et al. 2017; Shannon et al. 2016; Bambace et al. 2013).

39.5.1 General Concepts

Nocardia spp. (any of the dozens of currently accepted species may be involved, but most cases in Europe appear to be due to *N. asteroides*, *N. brasiliensis*, and *N. nova*) are aerobic gram-positive rods that grow in characteristic filamentous, branching chains and being acid fast, and their appearance makes them easily identifiable by microbiologists, with its acid-fast staining properties differentiating it from *Actinomyces* spp. *Nocardia* spp. grow in soil and decaying matter, and human infection usually occurs from inhalation of airborne bacilli.

39.5.2 Risk Factors and Incidence in HSCT

Nocardiosis is a late post-HSCT infection, occurring months to years after HSCT, mostly allo-HSCT. Patients usually have steroid-dependent chronic GVHD, secondary diabetes mellitus, and/or bronchiolitis obliterans or bronchiectasis from the numerous post-HSCT infections suffered. There are no specific risk factors in HSCT, although being at the right time in a place where soil-living bacilli are made massively airborne is a common-sense mechanism of infection. Similar to *M. tuberculosis*, *Nocardia* spp. do not colonize the airways.

The incidence of nocardiosis has been reported to range from 0.3 to 1.7% in allo-HSCT, although many large centers have not had a single case. In auto-HSCT the median incidence is 0%, although occasional cases have been reported and surely occur in many centers.

39.5.3 Most Common Clinical Presentations

Pulmonary infection, with its accompanying signs and symptoms, and radiologically one or more nodular lesions with a tendency to cavitate occur in 90% of patients with nocardiosis. At presentation, however, around half of the patients have disseminated disease, usually to the skin and osteoskeletal organs, but around 1/3 will have CNS involvement up front. Since CNS

involvement is so common and can initially be asymptomatic, a CNS CT or MRI scan is mandatory in all HSCT recipients with pulmonary nocardiosis (in any IS host, in fact). Brain abscesses are the usual presentation, although severe hyponatremia due to SIADH is also common due to basal meningitis.

39.5.4 Diagnosis

Diagnosis, of course, requires culture of an affected organ, usually the lungs. Often, the characteristic ramified bacilli can be directly observed from sputum or a directed BAL, but culture-based diagnosis is made in at least 1/3 of the cases. This is of utmost importance, since cultures become positive at a median of 9 days after sampling but can take up to 2–4 weeks. Molecular-based methods are useful only to identify uncommon species of *Nocardia* with known multidrug resistance, but this is rarely required in clinical practice. The most common differential diagnosis is with invasive pulmonary mold infections.

39.5.5 Specific Screening and/or Prophylactic Strategies Available

Screening has no role in preventing nocardiosis, but its rapid diagnosis does have an impact on patient outcome.

The routine use of TMP/SMX prophylaxis after HSCT may prevent more cases of nocardiosis, but the 2–3-day per week schedules are not effective in preventing it. Of note, *Nocardia* spp. isolated in patients taking single-strength TMP/SMX prophylaxis 5–7 days per week have had a good in vitro susceptibility to TMP/SMX and have responded well to high doses of the drug.

39.5.6 Treatment and Prognosis

High-dose TMP/SMX is still the treatment of choice, although there have been good results with carbapenems, amikacin, second-generation cephalosporins, and/or linezolid.

When treated promptly, nocardiosis usually resolves with prolonged antibiotic therapy, but directly attributable mortality has been reported in up to 40% of cases; these are, of course, those cases that affect extremely debilitated allo-HSCT recipients due to prolonged severe GVHD and its numerous complications, as well as those with disseminated infection and extensive CNS involvement, including the brain stem. Overall mortality, however, is high, since around 40% of patients have severe coinfections when nocardiosis joins the club.

Treatment of nocardiosis usually requires at least 6 months of specific antibiotic therapy, and it is of course recommended that ID specialists are actively involved in the treatment and follow-up. Of note, most *Nocardia* isolates are susceptible to most of the too-often empirically/prophylactically used antibiotics in HSCT recipients (levofloxacin, moxifloxacin, amoxicillin-clavulanate), as well as tetracyclines and tigecycline.

Key Points

- The intense IS associated with allo-HSCT, especially when there is a chronic GVHD that requires a prolonged IST, favors the development of infections by very unusual pathogens.
- Despite its low incidence, it is necessary to know these pathologies in order to make an early diagnosis and to adapt the therapy to the causal pathogen.

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