Mediterranean Journal of Hematology and Infectious Diseases

Review Article

Managing Invasive Fungal Infections During Allogeneic Hematopoietic Transplantation: A 2025 Update

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Competing interests: The authors declare no competing interest.

Abstract. Invasive fungal infections (IFIs) mostly affect immunocompromised hosts and are responsible for high rates of complications and mortality. Prevalence of IFIs has been reported between 7 and 15% and is evolving due to the introduction of new drugs in the prophylaxis of high-risk patients. Invasive candidiasis has become less frequent, while cases of aspergillosis are increasing. The most important risk factors for IFIs can be divided into 3 categories: those related to the hematological neoplasm, those related to the patient's lifestyle, and those dictated by the transplant characteristics. In high-risk patients, prophylaxis is driven by both local epidemiology and the timing of engraftment. During the pre-engraftment period, a wide spectrum of drugs can be chosen as antifungals, while in the post-engraftment period, posaconazole is recommended for patients presenting with GvHD who are undergoing immunosuppression. Regarding treatment, voriconazole is still the recommended drug for invasive aspergillosis, although adverse events, toxicity, and drug interactions are particularly relevant. In the management of IFIs, international guidelines recommend the best drugs for prophylaxis and treatment, but the future holds new molecules that are already demonstrating excellent efficacy and tolerability.

Keywords: Antifungal agents; Antifungal prophylaxis; Invasive fungal infections; Invasive Aspergillosis; Candida; Allogeneic hematopoietic cell transplantation.

Citation: Quattrone M., Di Pilla A., Brunetti S., Giordano A., Fianchi L., Pagano L., Criscuolo M. Managing invasive fungal infections during allogeneic hematopoietic transplantation: A 2025 Update. Mediterr J Hematol Infect Dis 2025, 17(1): e2025064, DOI: http://dx.doi.org/10.4084/MJHID.2025.064

Published: September 01, 2025 **Received: July 22, 2025** **Accepted: August 14, 2025**

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Introduction. Invasive fungal infections (IFIs) are opportunistic diseases that typically occur in immuno-compromised hosts with multiple predisposing factors. Among them, patients with hematological malignancies and those undergoing allogeneic transplantation (allo-HCT) are at maximal risk.[1–4] In these patients, IFIs and particularly invasive aspergillosis (IA), represent a very important cause of morbidity and mortality.[5,6] IA is a serious condition caused by *Aspergillus* spp., a family of ubiquitous fungi that is often introduced in the respiratory system by inhalation of the spores and may also disseminate through angio-invasion to other organs such as the brain, the skin, the kidneys, and the heart.[7,8]

In recent years, the number of transplant procedures and the median age of HCT patients have increased, and both transplant-related complications and infections are

Table 1. IFI in HSCT patients.

Study	Patients	Donor type	Stem cell source	Antifungal prophylaxis	IFI incidence	IFI type	Mortality
Girmenia et al, Biol Blood Marrow Transplant 2014[5]	1858	MRD 46% MMRD 6% UD 39% CB 10%	BM 31% PB 59% CB 10%	Secondary 5% Primary (fluconazole) 75% Primary (mold-active) 14% None 6%	8.8%	IA 81% IC 11% Zygomycosis 4% Fusariosis 2% Others 2%	46%
Atalla et al, Transpl Infect Dis 2015[108]	345	MRD 74% MMR 13% UD 13%	BM 56% PB 35% CB 9%	99% (90% fluconazole)	12.7%	Fusariosis 54% IA 21% Hyalohypho 14% Mucor 7% Others 4%	n.n.
Sun et al, Biol Blood Marrow Transplant 2015[109]	1401	MRD 75% MMRD 31% UD 26% Autologous 25%	PB 66% BM 3% CB 2% Mixed 29%	Secondary 9% Primary 77%	26.7%	IA 71% IC 28%	19%
Fayard et al, BMT 2019[110]	381	MMRD 100%	PB 73% BM 27%	100%	7.0%	IA 44% IC 33% Pneumocystis 13%	5%
Oltolini et al, BMT 2020[111]	235	MRD 17% MUD 21% MMRD 62%	PB 100%	Secondary 15% Primary 85%	-	-	-
Souza et al, Mycoses 2021[112]	192	MRD 16% MMRD 5% UD 17% Autologous 42% Unknown 20%	-	Fluconazole 82% Micafungin 8% Voriconazole 8% Itraconazole 1% Posaconazole 1%	13.0%	IA 56% IC 24% Fusariosis 12% Mucor 4%	-
Papanicolaou et al, Transplant. Cell. Ther. 2024[69]	2765*	MRD 73%	PB 77%	-	7.4%	IA 2% IC 1.6%	1.6%
		MMRD 27%			4.1%	IA 1.4% IC 4.0%	0.8%

BM: bone marrow - CB: cord blood – IA: invasive aspergillosis – IC: invasive candidiasis - MMRD: mismatched related donor - MRD: matched related donor – PB: peripheral blood - UD: unrelated donor.

more frequent.[9] On the other hand, the incidence of IFI has changed significantly over the years, also due to the introduction of new drugs used in the field of prophylaxis and treatment.[4]

Considering the poor outcomes associated with infections due to IFIs in immunocompromised hosts, it is essential to identify risk factors that increase the likelihood of IFI development after allogeneic transplantation.[10] In particular, it is important to distinguish risk factors related to the patient's lifestyle, those connected to the patient's hematological disease and its specific treatment, and finally those depending on the characteristics of HCT itself, such as the type of donor, stem cell source, conditioning, and GvHD (graft versus host disease) prophylaxis.[4,11,12]

To reduce the incidence of IFIs, an appropriate prophylaxis plays a crucial role: recently, ECIL (European Conference on Infections in Leukemia), ESCMID (European Society for Clinical Microbiology and Infectious Diseases), and IDSA (Infectious Diseases Society of America clinical guidelines) have updated indications on the most effective prophylaxis in immunosuppressed patients.[13–15] In particular, for allo-HCT patients, it is crucial to distinguish between the pre-engraftment and post-engraftment periods, as they present different risk factors for IFIs.[13,14,16]

Regarding indications for treatment, different classes of drugs are available, with specific pharmacological interactions and side effects. Sixteen new compounds are also undergoing clinical trials to expand therapeutic possibilities for these patients.[17]

Our review aims to provide useful tools for risk stratification, primary prophylaxis, and therapeutic management of IFIs in HCT recipients.

Epidemiology. Over the last decade, the epidemiology of IFIs has undergone dramatic changes, largely due to the increasing number of immunosuppressive treatments, the use of invasive devices, and a higher volume of transplant procedures.[18]

Invasive candidiasis is the most common fungal disease among hospitalized patients in the world, with an

incidence of 1-14 cases per 100.000 inhabitants[19] and a mortality rate reaching up to 60% in different studies.[20–23] Aspergillosis, on the other hand, affects 1.1-1.8 people per 100.000 inhabitants, with a mortality up to 85% in patients with invasive infections in intensive care settings.[24,25] A recent study on prevalence of fungal infections showed that in 2018, more than 600.000 fungal infections were diagnosed in the United States, with the highest mortality observed in patients diagnosed with mucormycosis (18.6%) and invasive candidiasis (17%), followed by *Pneumocystis jirovecii* infection (12.9%) and invasive aspergillosis (12.5%).[26]

HCT patients are at high risk of IFI as they represent a distinct epidemiological niche for fungal infections. **Table 1** describes the reported incidence of IFI in HCT patients, considering stem cell donor type and source, antifungal prophylaxis used, involved pathogens, and mortality.

The incidence of IFI in HCT patients ranges from 7% to 15% across various studies, with a notable shift observed over the last 15 years due to the introduction of new antifungal drugs for both prophylaxis and treatment. 4 In the past, candidemia was one of the most frequent IFIs in hematological patients, with high mortality, up to 60%.[27–29] *Non-albicans Candida species* were responsible for more than half of *Candida* infections in HCT, with 33% of infections caused by *C. glabrata*, 14% *by C. parapsilosis*, 8% by *C. tropicalis*, and 6% *by C. krusei*. Recent studies, however, have shown that the incidence of candidemia is lower than in the past, with a significant reduction in the candidemia fatality rate among HCT patients.[15,31,32] A SEIFEM report published in 2015 compared hematological patients that received HCT (either autologous or allogeneic) in 2011-2015 with a historical cohort (1999-2003).[15] The survey showed an important decrease in candidemia case fatality rate both in patients treated with autologous HCT (44% vs 9.5%, $p = 0.01$) and in patients who underwent allogeneic HCT (57% vs 24%, $p = 0.02$).[13-15] *Aspergillus* is considered the most common invasive mold disease in HCT patients.[4,33,34] Invasive pulmonary aspergillosis is the most common clinical manifestation, but it can disseminate through vascular invasion to other organs, such as the brain, skin, kidneys, and sinuses.[35] The introduction of antifungal prophylaxis and the use of new and effective anti-mold treatments have changed the epidemiology of *Aspergillus* infection in HCT, with a prevalence of 43-64% of all IFIs.[36,37] Recent data shows an incidence of IA in allogeneic HCT patients of 8%, with lung localization in 93% of cases and overall mortality reaching 70% in some studies.[35,37,38] The most frequent strains are *A. fumigatus* (42%), followed by *A. niger* (26%), *A. flavus* (11%), and *A. terreus* (5%).[38] While *Candida* spp. and *Aspergillus* spp. are responsible for most IFIs in HCT patients, the emergence of non-*Aspergillus* molds, such as *Mucorales* (7-8% of cases), *Fusarium* (0.1-5.2%), *Scedosporium,* and a few documented cases of *Cryptococcus,* have been reported.

Risk factors for IFIs. Timely and correct identification of IFI risk factors is critical to improve patients' outcomes. Risk factors for IFIs in HCT candidates should be assessed over time, as they may differ from those initially identified at diagnosis. Transplant conditioning, post-engraftment complications, and prolonged follow-up may all modify the actual risk of IFI. Although risk factors may be already present at the time of the transplant, other less predictable variables may occur during the post-transplant clinical course. As shown in **Table 2**, three broad categories can be identified, deriving from patient comorbidities and lifestyle, as well as the primary hematologic disease.[11]

Among factors related to the patient's characteristics, the increasing average age of transplant candidates is a crucial consideration. Although no unambiguous threshold value has been identified, some studies have shown that an age of 50 years or older may be associated with an increased risk of IFIs.[42] Comorbidities that increase the risk of IFIs include diabetes, chronic obstructive pulmonary disease (COPD), high BMI, but also iron overload following transfusion dependence.[4,43] Moreover, free iron acts as a catalyst, causing mucositis and negatively influencing the activity of neutrophils, monocytes, NK cells, and macrophages. This mechanism is widely exploited by fungi to proliferate.[4]

Considering the patients' lifestyle category, a SEIFEM study conducted on 1192 patients with newly diagnosed acute myeloid leukemia (AML) reported a strong correlation between cigarette smoke, cocaine abuse, and invasive mold infections ($p = 0.02$ and $p = 0.006$, respectively).43 Moreover, either having hobbies and jobs involving high exposure to fungal agents or a recent (6 months) house renovation were associated with IFI ($p = 0.01$, $p < 0.001$ and $p < 0.001$ respectively).[43]

Risk factors related to the patient's hematologic disease and its treatment define the most variable and dynamic category. Neutropenia, disease response at the time of transplantation (partial vs complete response), previous chemotherapy, immunotherapy and target treatments (including CAR-T therapy), myeloablative conditioning and immunosuppression, choice of donor (haploidentical, mismatched unrelated donors or cord blood transplant) and cell source, occurrence of grade 3-4 GvHD present a specific weight in definition of risk.[4]

Prolonged neutropenia is one of the most significant risk factors for IFI, and it can be detected in more than one-third of HCT patients with a diagnosis of fungal infection.[14,43,44] This data has been confirmed not only during the pre-engraftment phase, but also after engraftment, considering that many infections can be diagnosed months or years after HCT.

Previous viral infections, including respiratory

Table 2. Risk of Invasive Fungal Infection in HSCT.

Risk of Invasive Fungal Infection in HSCT		
Risk factors according to patients' characteristics		
Lifestyle	Comorbidities	Hematologic Neoplasia
- Job occupation - Smoke, cannabis - Place of residence - Pets - Potted plants	- Age - Genetic predisposition (TLR4, Dectin-1, PTX3 - Diabetes - Iron overload - Diseases with organ damage (i.e., pulmonary fibrosis) - Previous viral infections - Previous fungal infections - HCT-CI ≥ 3	- Neutropenia - Induction CT - Malignancy phase at HSCT - Stem cell donor - HSCT conditioning - T-cell suppression - GvHD - Use of steroids and other immunosuppressants - Type of antifungal prophylaxis
Risk factors over time		
At diagnosis	Before conditioning	After transplant
- Lifestyle risk factors - Comorbidities risk factors - Neutropenia - Viral/bacterial infections	- Disease response - Viral/bacterial infections - Previous chemotherapy - Immunotherapy and target treatment	- Disease response - Viral/bacterial infections - Previous chemotherapy - Immunotherapy and target treatment - Myeloablative conditioning - Immunosuppression - Choice of donor - Stem cell source - GvHD staging and treatment

*CT: chemotherapy, HSCT: hematopoietic stem cell transplant.

viruses and *CMV*, as well as the drugs used to treat them, such as ganciclovir, may play a role in favoring IFIs. IFIs have also been described after severe community-acquired viral infections (*Influenza, Parainfluenza,* and *Respiratory syncytial virus*) complicated by respiratory failure, with an incidence of IA ranging from 7% to 30%.[45–48] More recently, IA has also been recognized as a significant complication in severe *SARS-CoV-2* pneumonia. According to recent data, the incidence of severe aspergillosis after *SARS-CoV-2* pneumonia ranges from 2% to 33% of cases, with a mortality of approximately 56%.[49-53] Conversely, the incidence of invasive candidiasis after COVID-19 is 0.8-14%, with a higher risk in ICU settings and a particularly significant mortality rate ranging between 40% and 70%.[54–57]

Regarding chemotherapy, it is essential to consider the destruction of the gut barrier induced by the drugs and their impact on the microbiota. In recent mouse models, the administration of chemotherapy resulted in gut barrier damage, characterized by phenomena of dysbiosis and bacterial translocation.[58] Maintaining the integrity of the intestinal epithelium and the diversity of the microbiota could reduce the risk of IFIs and other complications, including GvHD.[59] Recent data on patients with AML treated with CPX-351 showed a protective effect on mucosal barrier function compared to classic chemotherapy (3+7), with an enhancement of gut microbial activity and antifungal resistance and a more balanced microbial composition thanks to the activation of the pathway of IL-22 and IL-10 and the production of immunomodulatory metabolites by anaerobic bacteria.[59,60]

The use of antifungal prophylaxis during chemotherapy is also a protective factor against IFI, such as the use of posaconazole during acute myeloid leukemia induction treatment.[43]

Patients who undergo allogeneic HCT after CD19-targeted CAR-T therapy are exposed to a magnified infectious risk, with an incidence of IFI as high as 21% in recent studies.[61] Shadman et al described a cohort of 32 patients, with an incidence of IFI of 18% (6/32) and a mortality of 33% (2/6). Target therapies may also increase the risk of developing IFI:[63,64] it is higher with BTK inhibitors (ibrutinib, acalabrutinib, and zanubrutinib) and with alemtuzumab, moderate/high with PI3K inhibitors (idelalisib, copanlisib, duvelisib), only moderate with blinatumomab and with venetoclax monotherapy.[64-67]

Allogeneic transplantation characteristics are also crucial in risk stratification. In particular, transplantation from an unrelated or haploidentical donor is at higher risk of developing infections than from identical donors.[68–70] In a recent study, the incidence of IFIs in haploidentical donor transplants (haplo-HCT) was 5.2%. In comparison, in sibling identical transplants ranged between 1.9% and 2.2%, with an increased risk of transplant-related mortality and overall mortality.[70] The higher risk of IFIs in these patients could be explained by the increased incidence of GvHD and the slower rate of immune reconstitution after haploidentical transplantation, with a higher incidence of prolonged neutropenia.[71,72] It is also described as an increased number of *Candida spp*. Infections in cord blood transplantation are likely due to delayed engraftment compared to other donor sources.[73] Infection risk also increases with a second or third transplantation, because

failure to engraft after HCT is associated with prolonged cytopenia and a higher toxicity.[69]

Moderate to severe GvHD, referred to as grade 2 or over and requiring treatment with high-dose steroids, increases IFI's risk. In fact, corticosteroids impair the activity of macrophages and neutrophils. However, they also reduce the count of lymphocytes, leading to a deregulation of Th1/Th2.[69] Chronic severe GvHD is also an important risk factor, due to the prolonged immunosuppressive treatment that impacts regulatory immune T and B lymphocytic pathways.[69]

Furthermore, the risk of IFIs tends to lower over the years after transplantation, but it never disappears. In this respect, a study of Foord et al showed that, although infections are less frequent over time, the relative risk remains increased in 5-year HCT survivors versus other cancer survivors and the general populatio.[74] In particular, bacterial and fungal infections were each 70% more common in HCT versus non-HCT cancer survivors (IRR, 1.7; p = 0.01), with incidences of *Aspergillus* at 3.3% versus 1.3% and *Candida* at 4.1% versus 2.8%, respectively.[74]

Antifungal Antifungal prophylaxis in allogeneic HCT recipients. Regarding IFI prophylaxis in patients undergoing allogeneic HCT, it is valuable to distinguish between two phases characterized by significantly different risk factors for IFI: the pre-engraftment and post-engraftment phases. During the pre-engraftment period, fluconazole is likely the most valuable choice for antifungal prophylaxis, given its low rate of pharmacological interactions and toxicities. Unfortunately, the moderate efficacy of fluconazole often limits its use.[13,14,75] It is therefore reasonable to prefer fluconazole only in low-risk patients and in hospitals where the epidemiological records show a low risk of mold infection. It is also important to combine the use of fluconazole with a mold-directed diagnostic workup (biomarkers and/or CT scan-based).[13,14,75] On the other hand, using fluconazole in association with other antifungal drugs has not shown any advantages.[13,14]

In the post-engraftment period, in high-risk populations and in hospitals with a recurrence of mold infection, posaconazole has been shown to be more effective than fluconazole in preventing IFIs, particularly invasive aspergillosis, with a comparable rate of treatment-related serious adverse events.[76]

Among the azoles, voriconazole is one of the most effective antifungal agents. However, in clinical practice, its administration often presents several challenges in HCT patients, considering the variable pharmacokinetics, the narrow therapeutic window, and drug-to-drug interactions, particularly with immunosuppressive agents.[77,78] One important interaction to consider is with letermovir: voriconazole trough concentrations may decrease after starting letermovir, due to large inter-individual pharmacokinetic variability, which depends on body weight, genetic polymorphism in CYP, liver function, and serum C-reactive protein concentration.[79,80] Although voriconazole serum concentrations may vary during letermovir administration, the incidence of fungal infection does not increase in these patients.

Itraconazole has also shown better protection than fluconazole against invasive mold infections, but with a higher degree of toxicity and lower tolerability.[16,84] On the other hand, isavuconazole has shown conflicting results in terms of efficacy, with variable rates of breakthrough infections (ranging from 3.2 to 17.9%)[84,85] despite an encouraging safety profile.

Regarding echinocandins, evidence in literature mainly concerns micafungin, but comparative studies with fluconazole are often based on a low-risk population, resulting in a low incidence of IFI.[86]

Recent trials are ongoing to explore the efficacy of rezafungin, a new echinocandine derivative of anidulafungin with a safer profile and a longer half-life, approved for the treatment of candidemia.[88]

Antifungal prophylaxis should be continued in GVHD patients with chronic immunosuppression due to steroids (corticosteroid equivalent of > 1 mg/kg/day of prednisone for > 2 weeks) or other anti-GVHD therapies (such as lymphocyte-depleting agents) and in long-term neutropenic patients.

Secondary prophylaxis should also be considered for patients with successfully treated IFI who require subsequent immunosuppression.[14]

IFI treatment. The choice of antifungal treatment should consider various factors, including prior use of mold-active azole prophylaxis, existing comorbidities, the likelihood of azole-resistant *Aspergillus* infection, and clinical conditions.

Voriconazole is the drug of choice for the primary treatment of IA.[13,14] Plasma levels of voriconazole should be controlled 2–5 days after the first dose. If levels are sufficient (between 1–1.5 and 5–6 lg/mL), they should be monitored regularly due to high intraindividual variation.[97] Isavuconazole is an alternative first-line agent with high tolerability and fewer side effects.[98] Regarding posaconazole, data have shown non-inferiority to voriconazole in the treatment of IA, with a limited number of adverse effects.[99]

Treatment should be initiated as soon as possible in patients with strongly suspected IA and continued for at least 6-12 weeks, depending on the degree and duration of immunosuppression, the site of disease, and evidence of disease improvement. We advise against stopping treatment until complete clinical resolution and favorable radiological evolution are achieved. Adjunctive measures should reduce or eliminate immunosuppressive agents when feasible and consider colony-stimulating factors in neutropenic patients with

invasive aspergillosis that is refractory or unlikely to respond to standard therapy, and for an expected neutropenia of more than one week. Combining voriconazole and echinocandin may also be considered in selected patients with an incomplete response to first-line therapy and a documented infection.[100] Surgery for aspergillosis should also be considered for localized disease that is accessible to debridement.

Echinocandins, including the new drug rezafungin, are the recommended first-line treatment for candidaemia and all forms of invasive candidiasis, except for neurological and ocular sites, due to their broad activity and safety profile.[101] Second-option treatments include liposomal amphotericin B and fluconazole, although fluconazole resistance must be considered in accordance with local epidemiology.[101]

Cryptococcosis is a rare and lethal infection in HCT patients. Liposomal amphotericin B, 3–4 mg/kg daily, and flucytosine, 25 mg/kg four times a day, are the most effective therapy options for cryptococcal meningitis, disseminated cryptococcosis, and severe isolated pulmonary cryptococcosis in high-income settings.

Recent trials are investigating new drugs for the treatment of IFIs. Opelconazole is an inhibitor of the fungal dihydroorotate dehydrogenase that has shown efficacy against various fungal species, especially *A. fumigatus*, but also *Scedosporium*, *Lomentospora*, *Rasamsonia*, and *Talaromyces*.[88] Opelconazole has been designed for topical use and nebulized administration, with a low rate of drug interactions and toxicities and a promising efficacy.[88] Fosmanogepix is an inhibitor of the fungal enzyme Gwt1 with broad-spectrum activity against yeasts including *Cryptococcus* and *Candida*, as well as molds, such as azole-resistant *Aspergillus*.[103,104] Data show that it could be effective against pathogens that usually resist other drugs, such as *Scedosporium*, *Lomentospora prolificans*, and *Fusarium*.[103] It may also have a favorable profile in terms of drug interactions and adverse events.[103]

Ibrexafungerp is a non-competitive inhibitor of the β-(1,3)-D-glucan synthase enzyme, demonstrating activity against a range of pathogens, including *Candida* and *Aspergillus* spp., while retaining its activity against azole-resistant and echinocandin-resistant strains.[107] Data showed comparable responses to the standard of care in invasive candidiasis, with favorable preliminary results in *C. auris* infections in terms of efficacy and tolerability, as well as in refractory cases. Mild adverse reactions have been reported, including gastrointestinal symptoms.[107]

Refractory or Progressive Aspergillosis. Aspergillosis can be defined as refractory/progressive when there is a clinical, radiological, or serological worsening in patients who have already started a first-line treatment.[102] A sign of serological refractoriness could be a stable galactomannan (not fallen by either 1 unit or < 0.5 units based on measurements taken at least 7 days apart) after at least 8 days of treatment, or a positive galactomannan from BAL in a patient with a previously negative exam.[102] Other criteria to classify aspergillosis as refractory could include a new, distinct site of infection detected clinically or radiologically, or the progression of the original lesion by> 25% after at least 8 days of therapy.[102]

In case of refractory or rapidly progressive aspergillosis, it is mandatory to exclude the emergence of a different strain. Salvage therapy includes changing the class of antifungals, reducing or reversing the underlying immunosuppression (when feasible), and considering surgery in selected cases. It is also advisable to add another antifungal drug from a different class or combine antifungal treatments from other classes that the patient has not received yet.[97] In documented azole-resistant disease, it is beneficial to switch from voriconazole monotherapy to a combination with an echinocandin or amphotericin B.[96] Not only clinical monitoring, but also serial monitoring of serum galactomannan (at least every 7 days) should be used to monitor disease progression and therapeutic response.

Conclusions. In this review, we highlighted the main challenges of managing invasive fungal infections in HCT patients. In particular, we discussed the main risk factors for IFI and the most influential international guidelines for antifungal prophylaxis and treatment, according to the engraftment phase.

Refractory/progressive infections remain a challenge; therefore, combination therapy should be considered, potentially leading to a change in the class of antifungal agents. Reducing the underlying immunosuppression, excluding the emergence of a new pathogen, and surgical debridement are also options, although they are difficult to apply in daily practice.[14]

In the context of new drugs, the possible role of rezafungin as a prophylactic agent in HCT patients will be defined in the next years. Indeed, the data on the use of isavuconazole in the prophylaxis of fungal infections in HCT patients are promising, with good efficacy and an excellent tolerability profile.

Inhaled opelconazole could become very promising: ongoing trials are exploring both monotherapy and combination treatment with other antifungals, exploiting the virtually absent bloodstream absorption and potential for drug interactions. Scientific research on new drugs identified oteseconazole as less toxic than other azoles with a lower risk of drug interactions.

One additional research field is the emergence of rare fungal species, such as *Mucorales*, for which innovative diagnostic tools and therapeutic options are needed.

Exciting new perspectives concern the role of microbiota. Gut dysbiosis induced by chemotherapy and

antibiotics appears to be predisposing for fungal infections, especially by *Candida spp*. New studies will be needed to determine whether the use of probiotics and prebiotics, in association with antifungal drugs, may play a role in the prophylaxis treatment of IFIs.

Despite significant advances in the treatment of patients undergoing HCT, fungal infections still represent an important cause of morbidity and mortality. Exhaustive knowledge of risk factors associated with the occurrence of IFI has a significant impact on clinical practice, as it enables the identification of patients who require a targeted diagnostic approach, including serial monitoring of radiological and microbiological examinations, such as high-resolution CT scans and bronchoalveolar lavage. Early diagnosis and timely treatment represent available criteria for improving the outcome of these infections and patient survival.

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