Table 2
IFI (EORTC/MSG Criteria)

UPN	Symptoms	Imaging	BAL	IFI
27	Yes	Ground glass opacity/ consolidation	Galactomannan +ve	Probable
46	Yes	New focal opacity in previous consolidation	-ve	Possible
85	Yes	Small b/l pleural effusion with ground glass opacity and septal thickening	-ve	Possible

2 (2%) patients developed other fungal related problems. One patient developed oral candidiasis treated with micafungin. One patient had splenic leisons (presumed fungal) treated with treatment dose posaconazole and improved.

None of these patients had aGVHD at time of IFI diagnosis. None of these patients died of fungal related causes. 4 out of 5 patients were alive at last follow up. One died of relapse.

Conclusion: Micafungin followed by posaconazole is effective as primary IFI prophylaxis in unrelated donor HCT.

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Nocardiosis in Allogeneic Hematopoietic Stem Cell Transplant Recipients: A Matched Case-Control Study of Risk Factors, Clinical Features and Outcomes

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Nocardial infection is emerging as an important cause of morbidity and mortality among hematopoietic stem cell transplant (HSCT) recipients. Risk factors and outcomes in this population remain undefined.

We performed a matched case-control study (1:2 ratio). Cases were defined as recipients of allogeneic HSCT with a microbiological diagnosis of nocardial infection. Control subjects were matched for age, timing and transplant type. Between January 2007 and December 2011, among 440 allogeneic HSCT recipients, 11 (0.03%) were diagnosed with nocardiosis.

Infection occurred at a median of 510 days (range 139-1042) after HSCT and was disseminated in 45% of cases. Diagnoses clustered in the fall (68%). Pulmonary involvement with nodular infiltrates occurred in 91% of cases. Final culture results were available 55.7 days after diagnostic testing (range 14-120 days). *Nocardia nova* was the strain most commonly isolated (27%). Co-infection with *S.aureus, Pseudomonas,* CMV and *Mycobacterium sp.* occurred in 73% of all cases. Trimetroprim-Sulfametoxazole was not protective in 4 out of 11 patients (36%) receiving it for *Pneumocystis jirovecii* pneumonia prophylaxis.

In univariate analysis, chronic GVHD (P= .011) was associated with nocardial infection. Other associated conditions included bronchiolitis obliterans syndrome (BOS) (P= .033), steroid-induced diabetes mellitus (p=< .001), and opportunistic infection within the preceding 6 months (p= < .001). Positive CMV serologic status or recent CMV infection were not significant variables. High-dose corticosteroid treatment within the preceding 6 months (P= .005), tacrolimus therapy (P= .002), antifungal prophylaxis (p = <.001), prior autologous transplant (P= .008), and rituximab treatment within 12 months (p = < .001) were specifically associated with nocardiosis. Patients with *Nocardia* infection had significantly higher mean tacrolimus levels (p = .043), LDH levels (p = .040), and neutrophil counts (p= .002) than controls.

Nocardial infection is an infrequent delayed complication of allogeneic HSCT primarily affecting recipients with chronic GVHD. This has biologic correlation, since these patients often require prolonged immunosuppressive therapies, targeting both B and T cells, and may have underlying anatomic factors interfering with microbial clearance, such as bronchiectasis in BOS. High-dose corticosteroid treatment and its consequences, notably steroid-induced DMII, may increase susceptibility to this infection. Nocardiosis should be promptly considered and carefully investigated in susceptible cGVHD patients, given its adverse impact on prognosis, as demonstrated by the significant reduction in the overall survival of the *infected* cohort (72.7% vs 100%, P= .013).

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Significance of Rh Mismatch in Allogeneic Hematopoietic Progenitor Cell Transplants

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The Rh D antigen is one of the most immunogenic red blood cell antigens know. Despite this, it is not considered in matching schemes for hematopoietic progenitor cell (HPC) transplants. The frequency of anti-D after Rh mismatched allogeneic HPC transplants is reportedly low. The objective of this study was to retrospectively study Rh mismatched HPC transplants and examine both allogeneic anti-D formation as well as transplant outcome measures. From January 1999 to April 2011, 104 consecutive adult Rh mismatched HPC transplants were performed at our institution and were available for review. We compared transplants with Rh+ recipients with Rh- donors (R+/D-, n=60) to those with Rh- recipients and Rh+ donors (R-/D+, n+44). There was no difference in underlying diagnoses, graft source, dose of HPC, degree of HLA matching, conditioning, or type of graft versus host disease (GVHD) prophylaxis. The median follow-up was 655.5 days (range 14 – 4264 days). Only 2 patients formed anti-D during the follow-up period, and both were in the R+/D- group of transplants. One was never exposed to Rh-positive blood products. The second patient was exposed to multiple Rh-positive apheresis platelet products prior to antibody formation. Ten patients formed other red cell alloantibodies with no statistical difference between groups. The most common antibody formed was anti-E. The overall use of rbc's and platelets post transplant was similar in both groups, although the use of Rh-positive rbc's was more common in the R+/D- group pre-transplant, and the R-/D+ group post-transplant. A low incidence of chronic GVHD was seen in both groups (82% of R+/D- transplants had no chronic GVHD, 64% of R-/D+ transplants had no chronic GVHD, P = .045). No difference in platelet and neutrophil engraftment was demonstrated.

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The Cost of Pediatric Unrelated HSCT

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