Table 2. Clinical presentation and laboratory findings at initial presentation

| Clinical presentation          | N = 46 |
|-----------------------------|--------|
| Upper extremity             | 44 (95) |
| Redness (%)                 | 28 (61) |
| Pain (%)                    | 25 (54.3) |
| Abscess (%)                 | 9 (19.6) |
| Skin manifestations* (%)    | 34 (74) |
| Symptomatics (%)            | 12 (26.1) |
| Constitutional symptoms (%) | 11 (23.9) |
| Time to evaluation (days), median | 24 (0-366) |

Laboratory findings

| WBC, mean ± SD | 6.14 (2.23) |
| Platelets, mean ± SD | 252 (94.5) |
| CRP, mean (range) | 5.0 (0-60) |
| Time of positive culture, mean ± SD | 28.2 ± 13 |

*Skin manifestations include nodules, papules and plaques

Table 3. Complicated versus non-complicated

| Uncomplicated n = 35 | Complicated n =11 | P value |
|----------------------|-------------------|---------|
| Gender               |                   |         |
| Male (%)             | 19                 | 9       | 0.160 |
| Female (%)           | 16                 | 2       |       |
| Immunosuppression (%)| 5                  | 0       | 0.658 |
| IMI (%)              | 5                  | 0       | 0.317 |
| WBC, mean           | 6.14               | 6.42    | 0.731 |
| Platelets, mean     | 253.1              | 251.3   | 0.959 |
| CRP, median         | 5                  | 5       | 0.825 |
| Duration of symptoms| 2.9                | 4.7     | 0.026 |
| Number of drugs used| 1                  | 2       |       |
| Length of treatment  | 3.6                | 5.7     | 0.031 |

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1385. Mechanism-Based, In Vitro Inhibition of Mycobacterium abscessus: Assessing β-Lactam Therapy
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Background. M. abscessus (Mab) is an emerging pathogen, a highly drug-resistant rapidly-growing nontuberculous mycobacteria. Mab L,D transpeptidases (LdtMab, LdtMab2), D-carboxypeptidase and BlaMab-β-lactamase are important targets. Herein, we tested the susceptibility of cetefaroline (TAR) and imipenem (IMI) alone and in combinations with two diazabicyclooctanone β-lactamate inhibitors (BLI), relebacant (REL) and avibactam (AVI), against representative clinical isolates belonging to the Mab complex and assessed the mechanism of inhibition using mass spectrometry (QTOF-MS).

Methods. Minimum inhibitory concentrations (MICs) of TAR and IMI with or without AVI and REL and a TAR-IMI combination with and without REL were determined using microdilution. Approximately 5 x 10^6 colony-forming units (CFU) per milliliter were inoculated into Middlebrook 7H9 broth supplemented with 10% (vol/vol) oleic albumin dextrose catalase and 0.05% (vol/vol) Tween 80. AVI or REL were added at fixed concentration of 4 μg/mL to serial dilutions of TAR or IMI. For the TAR-IMI combinations, IMI at 1 μg/mL, and serial dilutions of TAR were used. Mab isolates were incubated with test agents at 30°C for 48 h, and MIC was defined as the lowest antibiotic concentration that prevented visible bacterial growth. (QTOF-MS) was used to assess intermediates of BlaMab, LdtMab1, and LdtMab2 bound the AVI, REL, but acyl complexes with TAR or IMI were not detected. LdtMab1, LdtMab2 form stable acyl complexes with REL, TAR, IMI, and IMI is commercially available and thus might be considered as part of a rescue regimen.

Results. Addition of IMI to TAR lowers MICs of TAR against Mab to therapeutically achievable concentrations. It would be welcome news for clinicians who are treating patients with highly resistant Mab infection that the combination of TAR and IMI lowered MIC's of all clinical isolates to <0.06 μg/mL. Addition of REL or AVI lowered MIC's of all clinical isolates to <0.06 μg/mL. Further studies are warranted to establish the efficacy of these combinations in vivo.

Conclusion. Addition of IMI to TAR lowers MICs of TAR against Mab to therapeutically achievable concentrations. It would be welcome news for clinicians who are treating patients with highly resistant Mab infection that the combination of TAR and IMI lowered MIC's of all clinical isolates to <0.06 μg/mL.
other NTM isolates. Most patients (n = 410, 62.8%) had none of the evaluated comorbidities prior to NTM diagnosis. Median follow-up time was 1252 days (IQR 449–2688). Median survival in our cohort among persons with and without comorbidities was 1973 days (95% CI 1487–2995) and 3952 days (95% CI 3496–5186), respectively. Median expected survival in the demographically-matched population cohort was significantly lower than expected in the general population. The impact of NTM infection itself vs. other comorbidities on survival requires further study.

Conclusions. NTM pulmonary infection in our cohort was associated with significantly lower survival than expected in the general population. The impact of NTM infection itself vs. other comorbidities on survival requires further study.

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1387. Women Living with HIV (WLWH) Lose IFNγ Responses Diagnostic of Late Latent TB Infection (LTBI) during Pregnancy and after INH Prophylactic Treatment (IPT)

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Background. TB is the most common opportunistic infection in PLWH. IPT is recommended for PLWH in endemic areas and for those with LTBI diagnosed by QuantiFERON gold-in-tube (QGIT) or tuberculin skin test (TST) in other areas. We report on the performance of QGIT and TST in pregnant WLWH who received IPT antepartum (AP) or postpartum (PP).

Methods. WLWH participating in IMPAACT P1078, a randomized, double-blind, placebo-controlled study comparing 28 weeks of IPT AP vs. PP, were tested by QGIT at entry (14–34 weeks gestation) and by QGIT and TST at delivery (L&D) and 44 weeks PP. Serial QGIT positivity was assessed by logistic regression using generalized estimating equations.

Results. Among 944 women with study entry mean (SD) of 29 (6) years of age, 521 (245) CD4+ cells/µl, on ART, including 63% with undetectable HIV plasma RNA, 284 (443) (30%) were QGIT+ AP 215 (62) (25%) at L&D and 246 (154) (32%) PP (P = 0.001), while 127 (15%) were TST+ at L&D and 126 (17%) PP. QGIT was more likely positive than TST at L&D (Odds ratio = 4.3, 95% CI = 2.8–6.8) and PP (6.4, 3.9–10.7; P = 0.001). QGIT and TST agreement coefficients (95% CI) were 0.4 (0.3–0.5) at L&D and 0.5 (0.4–0.5) PP. Among women QGIT+ AP, 59 (24%) reverted to QGIT- or indeterminate at L&D. However, 37 (63%) reverters recovered QGIT+ results PP, suggesting transient suppression of IFNγ responses during pregnancy. Responses to the mitogen-positive QGIT kit control were absent in 60 (7%) women AP, 116 (16%) at L&D, but 3 (0.4%) PP (P = 0.01), supporting the notion of transient immune suppression during pregnancy. Among women QGIT- AP, 33 (7%) converted to QGIT+ PP. Among AP QGIT+ women, 24 (11%) reverted to QGIT- PP after finishing IPT. None of the results differed between treatment arms (F 0.13).

Conclusion. In WLWH on ART, the loss of IFNγ responses to TB antigen and mitogen in pregnancy decreased the diagnostic value of QGIT. TST was similar at L&D and PP but was less sensitive than QGIT. QGIT conversions likely resulted from a combination of PP immune reconstitution and new TB infections. QGIT reversion might represent a change in TB-specific immunity in response to IPT. Reversions have been reported in patients without HIV after treatment of active TB. The clinical significance of QGIT reversions in PLWH needs further investigation.

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1388. Characteristics of Non-Tuberculous Mycobacterial Infections in Hematopoietic Stem-Cell Transplant Patients

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Background. Hematopoietic stem cell transplantation (HSCT) recipients are at increased risk for non-tuberculous mycobacterial infections (NTMI), but little data exists regarding NTMI in this population. This study aimed to better define the clinical and epidemiological characteristics of NTMI in HSCT patients.

Methods. We performed a retrospective review of patients age ≥ 18 who underwent HSCT between 2000 and 2017 at Yale-New Haven Hospital, and who subsequently had an NTMI based on ATS/IDSA criteria or a culture that grew NTM. We reported patient demographics, illness severity, treatment, and outcomes.

Results. Of 1371 HSCT recipients between 2000 and 2017, 17 (1.2%) had an NTMI or were culture positive. Most patients were male (76.5%), received allogeneic HSCT (70.6%), had graft vs. host disease (GVHD) (70.6%), and received immunosuppression (64%). Mycobacterium avium complex (MAC) was isolated in 16 (94.1%). Cultures were mainly positive in respiratory specimens (8 bronchoalveolar lavage, 2 sputum, and 1 lung tissue). Nine of 17 patients (52.9%) were deemed colonized whereas 8 (47.1%) were considered infected. In the infected group, isolated pulmonary infection was the most common presentation (n = 5, 62.5%). Two of the 8 infected patients (25%) had MAC bacteremia. Of those with NTMI, MAC was isolated in 6 (75%), MAC and M. abscessus/chelonae in 1 (12.5%), and M. kansasi in 1 (12.5%). Among those with NTM 3 were hypoxic (37.5%) and 4 (50%) had sepsis, though only 1 of the 4 had sepsis was directly attributable to NTM. Seven infected patients received antymycobacterial therapy. Four patients (50%) died but none were directly attributed to NTM. Diagnosis was often delayed in these patients, with a median of 44 days (range 14–155 days) from time of initial presentation to diagnosis of NTMI. Median time to death from time of NTM diagnosis was 75 days (range 16–1825 days).

Conclusion. NTMI, including disseminated infection, was uncommon in a large cohort of HSCT patients. Careful evaluation of positive cultures in these patients is needed to distinguish infection from colonization. High mortality in HSCT patients may not be directly attributable to NTMI, but the presence of NTM may be a predictor of poor outcomes.

Table 1. Characteristics of HSCT Patients with NTM Infection

| Patient | Type of Infection | Antibiotic Therapy | Duration of Therapy | Outcome |
|---------|-------------------|-------------------|--------------------|---------|
| 1 | Disseminated MAC (pulmonary and bacteremia) | Clarithromycin Erythromycin Rifampin Followed by Amphotericin B | 3 months | Death |
| 2 | Pulmonary MAC | Amphotericin B | 9 days | Death |
| 3 | Pulmonary M. kansasi | Unknown* | Unknown* | Death |
| 4 | Pulmonary MAC | Amphotericin B | 3 months | Unknown |

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