

Terms. We excluded 80 HIV-infected and identified association of the infection with known risk factors.

Results. Of 570 patients, 491 HIV-uninfected patients with DMAC were studied. Underlying structural pulmonary diseases were COPD and bronchiectasis (51% and 47%, respectively). Two hundred ten patients had concomitant malignancy of which lung cancer was the most frequent (43%). Seventy-nine percent were receiving corticosteroids and 10 patients (2%) were on TNF inhibitors (2%).

Conclusion. In this study, majority of patients with DMAC are HIV-uninfected. Larger studies should focus on identifying the prevalence and risk factors of DMAC in the post-AIDS era.

Table: Distribution of the Sample According to Associated Conditions

	N = 490 (%)
Age, years	
Senior (>65)	330 (67%)
Adult (18–65)	160 (33%)
Female	310 (63)
Smoking history	420 (86)
Bronchiectasis	230 (47)
Previous pulmonary tuberculosis	40 (8)
COPD	250 (51)
HTN	310 (63)
Diabetes mellitus	130 (27)
Chronic kidney disease	110 (22)
Interstitial lung disease	90 (18)
Inflammatory bowel disease	20 (4)
Concomitant malignancy	210 (43)
Lung	60 (28)
GI	50 (23)
Head and neck	40 (19)
Hematological	40 (19)
Renal	30 (14)
Chronic oral corticosteroid treatment	390 (79)
Tumor necrosis factor alpha inhibitor therapy	10 (2)

COPD; chronic obstructive pulmonary disease, HTN; hypertension, GI gastrointestinal.

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785. Treatment of *Mycobacterium immunogenum* Skin and Soft-Tissue Infections: A Case in a Peritoneal Dialysis Patient

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Background. *Mycobacterium immunogenum* is a somewhat recently identified species of rapidly growing nontuberculous mycobacteria, genetically related to *M. abscessus* and *M. chelonae*. Resistance patterns of rapidly growing nontuberculous mycobacterium species can make them difficult to treat. This is particularly true of *M. immunogenum*, in part due to the infrequency of reported cases of human infection and limited data to guide therapy.

Methods. We present here a case of *M. immunogenum* skin and soft-tissue infection at the site of insertion of a peritoneal dialysis catheter in a patient with end-stage renal disease. He initially presented with nodular subcutaneous lesions around his catheter site that progressed through oral antibiotics. This led to sampling which confirmed the diagnosis of *M. immunogenum*. We conducted a review of the literature to identify previously reported cases of *M. immunogenum*, including skin and soft-tissue infections, and used these data to guide management.

Results. We reviewed 11 reports (cases and case series) of *Mycobacterium immunogenum* in the literature. Susceptibilities often take weeks to return, and so empiric therapy is based on case series, and then later adjusted based on susceptibilities. Patients received combined antimicrobial regimens with durations of 2 weeks to 12 months, with variable outcomes. Several required surgical debridement, as was the case with our patient. His PD catheter was removed and he was treated empirically with amikacin, azithromycin, and tigecycline intravenous induction. His ultimate long-term regimen was later switched to azithromycin, clofazimine, and tedizolid due to side effects and the eventually available susceptibility profile.

Conclusion. The treatment of *M. immunogenum* remains a challenge due to the relative scarcity of data to guide treatment, and consequent lack of systemic approach to therapy. Most reported cases involve the use of a macrolide, often in combination with an aminoglycoside or a fluoroquinolone. Several started with intravenous induction, followed by transition to oral therapy on the order of weeks to months. Others also require surgical debridement. More data are required to develop a standardized approach to the treatment of *M. immunogenum*.

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786. Ceftaroline and Avibactam? Is This a Potential Combination for *Mycobacterium abscessus* Infection?

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Background. *Mycobacterium abscessus* harbors a β -lactamase enzyme, Bla_{Mab}, able to hydrolyze penicillins, most cephalosporins and carbapenems. As of today, management of *M. abscessus* with β -lactams does not include combination of β -lactamase inhibitors. The potential benefit of combinations of several β -lactams with new diazabicyclooctane (DBO) inhibitors, such as relebactam and avibactam, has not been well studied. Based upon the ability to inhibit Bla_{Mab} by highly potent DBO inhibitors, our goal herein was to investigate the efficacy of a novel combination, ceftaroline (CEF) and avibactam (AVI), to restore susceptibility to β -lactam antibiotics and inhibit growth.

Methods. Minimum inhibitory concentrations (MICs) of CEF with or without AVI were examined using the microdilution method.

Results. MIC₅₀ and MIC₉₀ of CEF is 8 mg/L; in the presence of 4 μ g/mL of AVI, the MICs of CEF decreased to \leq 4 mg/L in 31 of 35 cases (table).

Conclusion. Our results add to the growing evidence of using β -lactams as agents effective against Mycobacterial infections. Inhibition of the hydrolytic activity of (Bla_{Mab}) using DBOs such as AVI suggest that this combination should be evaluated in animal and clinical models.

Table 1: MICs of *M. abscessus* Strains (mg/L).

	Avibactam (AV), Ceftaroline (CEF)				
	CEF	CEF+AVI	CEF	CEF+AVI	
<i>M. ab</i> 15-103	8	0.25	<i>M. ab</i> (ATCC)	16	0.25
<i>M. ab</i> 15-442	8	1	<i>M. ab</i> IDR1400012185	32	0.25
<i>M. ab</i> 137-10561	8	0.5	<i>M. ab</i> IDR1400011191	8	<0.25
<i>M. ab</i> 15-305	16	2	<i>M. ab</i> IDR130008519	16	2
<i>M. ab</i> (bolletti) 15-148	16	1	<i>M. ab</i> 138-4796	2	0.25
<i>M. ab</i> 16-49	2	1	16-49	16	1
<i>M. ab</i> (ATCC)	32	<2	15-228	16	1
<i>M. ab</i> 16-21	8	0.5	15-235	8	0.5
<i>M. ab</i> 15-228	8	0.5	137-1061	>128	8
<i>M. ab</i> 15-206	128	1	<i>M. ab</i> 15-206	32	<0.25
<i>M. ab</i> 15-235	8	0.5	<i>M. ab</i> 15-86	64	0.5
<i>M. ab</i> 138-4796	32	<2	15-305	16	1
<i>M. ab</i> IDR130008519	16	16	<i>M. ab</i> 16-21	2	0.5
<i>M. ab</i> IDR1400012185	16	4	<i>M. ab</i> 15-103	4	0.5
<i>M. ab</i> 15-86	16	2	<i>M. ab</i> 15-148	8	1
<i>M. ab</i> 132-10561	4	1	15-442	32	1
<i>M. ab</i> #3	4	1	<i>M. ab</i> 132-10561	4	0.5
<i>M. ab</i> N1	4	0.5	<i>M. ab</i> #1	4	0.5
<i>M. ab</i> 60-2016	32	32	<i>M. ab</i> #2	8	2
<i>M. ab</i> IDR1400011191	16	16	<i>M. ab</i> #3	4	0.5
<i>M. ab</i> #14	16	1	<i>M. ab</i> #4	16	2
<i>M. ab</i> W2	8	1	<i>M. ab</i> #5	>128	4
<i>M. ab</i> #12	0.5	<0.125	<i>M. ab</i> #6	64	4
<i>M. ab</i> #13	32	2	<i>M. ab</i> #7	4	0.5
<i>M. ab</i> #11	16	0.5	<i>M. ab</i> #8	8	1
<i>M. ab</i> #10	32	2	<i>M. ab</i> #9	64	8
<i>M. ab</i> #7	4	1	<i>M. ab</i> #10	64	8
<i>M. ab</i> #1	16	0.5	<i>M. ab</i> #11	4	0.5
<i>M. ab</i> #8	18	0.25	<i>M. ab</i> #12	4	0.5
<i>M. ab</i> #9	32	4	<i>M. ab</i> #13	4	0.5
<i>M. ab</i> #6	32	1	<i>M. ab</i> #14	8	1
<i>M. ab</i> #4	8	0.5	<i>M. ab</i> 60-2016	32	32
<i>M. ab</i> #2	4	0.5	<i>M. ab</i> W2	8	0.5
<i>M. ab</i> #5	>64	4	<i>M. ab</i> N1	4	0.5

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787. The Addition of Avibactam Augments the Activity of Piperacillin Against *Mycobacterium abscessus* in vitro, and Is Effective in Treating *M. abscessus* Infection in a *Galleria mellonella* in vivo Model

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Background. *Mycobacterium abscessus* is an emerging multi-drug-resistant pathogen, harboring the β -lactamase Bla_{MAB}. Avibactam is a non- β -lactam, β -lactamase