Omadacycline for the Treatment of *Mycobacterium abscessus* Disease: A Case Series

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Key Points

We describe longer-term use of omadacycline as part of a treatment regimen for four patients with Mycobacterium abscessus disease and summarize the evidence to-date for the use of omadacycline in mycobacterial diseases.
Abstract

Background

Omadacycline is an aminomethylcycline antimicrobial approved by the United States Food and Drug Administration in 2018 for community-acquired bacterial pneumonia and acute bacterial skin and skin structure infections. It has in vitro activity against nontuberculous mycobacteria, including Mycobacterium abscessus complex, but clinical data for this indication are lacking.

Methods

Omadacycline use was reviewed at an 804-bed academic medical center. Patients were included if they received omadacycline for culture-proven M. abscessus disease in 2019.

Results

Four patients received omadacycline for the treatment of culture-positive M. abscessus disease in 2019. Two patients had cutaneous disease, one had pulmonary disease, and one had osteomyelitis and bacteremia. The patients received omadacycline for a median duration of 166 days (range 104-227) along with a combination of other antimicrobial agents. Omadacycline-containing regimens were associated with a clinical cure in three of four patients, with one patient improving on ongoing treatment. Omadacycline’s tolerability was acceptable for patients with M. abscessus disease, with one patient discontinuing therapy in month six due to nausea.
Conclusions

Omadacycline is a novel oral option for the treatment of *Mycobacterium abscessus* disease, for which safe and effective options are needed. While this case series is promising, further data are required to determine omadacycline’s definitive role in the treatment of *Mycobacterium abscessus* disease.

**Keywords:** omadacycline, *Mycobacterium abscessus*, mycobacterial infections, nontuberculous mycobacteria
**Introduction**

*Mycobacterium abscessus* complex, consisting of subspecies *abscessus*, *massiliense*, and *bolletii*, most frequently causes pulmonary or skin and soft tissue diseases [1]. The American Thoracic Society/Infectious Diseases Society of America nontuberculous mycobacterial (NTM) diseases guidelines recommend a combination of a macrolide with amikacin and either high-dose cefoxitin or imipenem for the initial treatment of serious skin, soft tissue, and bone diseases caused by *M. abscessus* [2]. Pulmonary disease-specific NTM guidelines recommend a combination including a macrolide and at least three active antimicrobials for the initial treatment phase [3]. However, resistance to multiple antibacterial classes is common for *M. abscessus*, making management particularly challenging [4]. For example, while a macrolide is considered one of the cornerstones of therapy, *M. abscessus* subsp. *abscessus* often has a functional erythromycin ribosomal methylase gene 41 (*erm41*), which confers inducible resistance to macrolides [5]. Treatment is prolonged and includes multiple active antimicrobials, several available only in intravenous formulations and/or limited by tolerability [2].

Omadacycline is a novel aminomethylcycline antimicrobial related to tigecycline available in both intravenous and oral formulations. It was approved by the United States Food and Drug Administration in 2018 for the treatment of community-acquired bacterial pneumonia and acute bacterial skin and skin structure infections. It shows *in vitro* activity against both gram-positive and gram-negative organisms [6]. Since its approval, multiple studies have shown omadacycline has favorable *in vitro* minimum inhibitory concentrations (MICs) against nontuberculous mycobacteria (NTM), most notably *M. abscessus* [7–9]. We report our clinical experience with the use of omadacycline as part of combination therapy for *M. abscessus* disease.
Methods

We reviewed omadacycline use at our 804-bed academic medical center and included patients who received omadacycline for culture-proven *M. abscessus* disease in 2019. Follow-up data were collected up until June 24, 2020. The University of Texas Health Science Center at Tyler performed mycobacterial susceptibility testing for three of the four isolates (cases 2-4), while the Laboratory Corporation of America provided susceptibility for case 1. Both laboratories were able to evaluate for inducible macrolide resistance, and both followed Clinical and Laboratory Standards Institute (CLSI) M24 performance standards for susceptibility testing [10]. Meropenem/vaborbactam testing for case 4 was done at a research laboratory at Harvard Medical School using an alamarBlue cell proliferation assay. This case series received exempt status from the Partners HealthCare Institutional Review Board (protocol 2019P003244).

Results

Four patients received omadacycline for the treatment of *M. abscessus* in the 12-month study period.

Case 1

A 45-year-old female underwent elective silicone breast implantation, liposuction and liposculpting of both arms, chest wall, upper lateral buttocks, back, and brachioplasty in the southern United States. Four weeks later, she developed persistent breast and back wound drainage and dehiscence requiring multiple debridements and implant replacements. After *M. abscessus* subsp. *bolletii* was cultured from the breast and back, empiric treatment with clarithromycin and trimethoprim-sulfamethoxazole was initiated. Three months into treatment, her implants were removed, and her
regimen was changed to clarithromycin and linezolid due to poor response. Shortly thereafter, she experienced worsening of her skin abscesses and was hospitalized to initiate amikacin, clarithromycin, and tigecycline. Her treatment was changed to azithromycin, linezolid, and tigecycline once susceptibilities resulted (Table 1). Since her organism’s tigecycline MIC was 0.25 mcg/mL, it was inferred that omadacycline may be active against the isolate. She was changed to oral omadacycline 450 mg daily for two doses as a loading dose, followed by 300 mg daily, and was ultimately discharged with an all-oral regimen of azithromycin, linezolid, and omadacycline.

During treatment, she required three additional drainage procedures in her lower back. She experienced cytopenias and paresthesias in her hands and feet, attributed to linezolid, which improved with a change to tedizolid. She now has minimal residual nodules and noticeable healing in areas of prior surgical drainage. She completed six months of omadacycline therapy in combination with azithromycin and an oxazolidinone, tolerating omadacycline without adverse events and to date has no evidence of disease recurrence eight months after antibiotic completion.

Case 2

A 72-year-old male developed head and neck cancer for which he underwent surgery and radiation therapy resulting in chronic aspiration events. He later developed a rheumatological disease characterized by weight loss, elevated inflammatory markers, and FDG avid thoracic and abdominal lesions consisting of dense lymphoplasmacytic infiltrates that were negative by mycobacterial culture. This disease remained undifferentiated despite extensive multidisciplinary evaluation. He was ultimately treated with one year of glucocorticoids with resolution of his symptoms and biochemical and radiographic abnormalities. Near the end of his glucocorticoid course, he developed fatigue, night sweats, and dyspnea and a computed tomography (CT) scan of the chest revealed a
large right middle lobe consolidation. Endobronchial biopsy demonstrated granulomatous changes with abundant acid-fast bacilli and culture identified *M. abscessus* subsp. *abscessus*.

His corticosteroid dose was tapered, and he completed a two-month induction course of amikacin, imipenem, clofazimine, and oral omadacycline dosed at 450 mg daily for two days, then 300 mg daily. He developed significant hearing loss and renal dysfunction on this regimen due to amikacin. After the two-month induction phase, his treatment was changed to omadacycline, clofazimine, and tedizolid for an all-oral regimen. Shortly after tedizolid initiation, he developed increased fatigue and dizziness, and tedizolid was therefore discontinued within days of initiation. Clofazimine and omadacycline were continued.

Despite medical management, he continued to experience fatigue and weight loss, and his chest CT showed a persistent right middle lobe consolidation. There was concern for ongoing infection, and he therefore underwent thoracoscopic right middle lobe resection 5 months into omadacycline therapy. During his hospitalization, he was unable to tolerate oral therapy for one week, so his clofazimine and omadacycline were briefly substituted with imipenem and eravacycline. Following surgery, he continued clofazimine and omadacycline, and there was gradual improvement in his fatigue and weight loss. Pathology from the lobectomy revealed granulomas, scarring, and interstitial chronic inflammation. There were no acid-fast bacilli on standard and modified stains and immunohistochemistry showed degenerated and fragmented mycobacterial forms. With negative lobectomy and BAL cultures, his antimicrobial therapy was discontinued three months post-operatively. Overall, he tolerated omadacycline for over seven months and has had no evidence of disease recurrence in four months since discontinuation.
Case 3

A 35-year-old female underwent abdominoplasty and back and buttocks liposuction in the Dominican Republic. One month later, she developed a post-operative wound infection treated empirically with moxifloxacin and clindamycin. Her infection worsened, and she subsequently transferred to our hospital with sepsis, wound dehiscence, and a clinical concern for a necrotizing skin and soft tissue infection. She had extensive deep tissue involvement in the flanks, abdominal wall, and thighs. She underwent multiple abdominal wall washouts, and intra-operative cultures resulted in mixed bacteria, including *M. abscessus* subsp. *abscessus*. At the time of discharge, she was treated with azithromycin, amikacin, imipenem, and linezolid. Susceptibility testing ultimately demonstrated resistance to linezolid and macrolides, and linezolid was discontinued (Table 1). Imipenem was discontinued after she complained of knee and ankle pain and chills suggestive of a serum sickness reaction. At that point 19 days into her treatment course, oral omadacycline 300 mg daily was added to azithromycin and amikacin. She experienced mild nausea upon omadacycline initiation which resolved after several days without medical management. Shortly thereafter, the patient reported tinnitus, so amikacin was discontinued and clofazimine initiated for an all-oral regimen of omadacycline, clofazimine, and azithromycin.

Her initial debridement procedures in the hospital had left her with open wounds in both flanks, abdominal wall, and thighs. These wounds demonstrated slow improvement, but during the first two months of therapy, she required several additional debridements in areas that continued to demonstrate poor healing. Her infection has slowly improved without recent flares but has not fully resolved. Her most recent operative cultures are negative for *M. abscessus*. In month six of omadacycline therapy, she developed nausea and vomiting for which she discontinued omadacycline. Since then, she has continued on clofazimine and azithromycin with no additional adverse events noted.
Case 4

A 41-year-old male with Guillain-Barré syndrome (GBS), chronic inflammatory demyelinating polyneuropathy, erythromelalgia, and prior lengthy antibiotic therapy for Lyme disease underwent a below the knee amputation for noninfectious complications of neuropathy. He had a chronic indwelling intravenous catheter for ongoing intravenous lidocaine for pain control and plasmapheresis for GBS. A month after his surgery, he developed a fever and progressive back pain. He ultimately presented to our hospital where *M. abscessus* subsp. *abscessus* was cultured from the blood. A lumbar spine MRI revealed findings compatible with vertebral discitis and osteomyelitis.

His indwelling catheter was removed, and subsequent blood cultures were negative. Diagnostic aspiration cultured *M. abscessus* from the disc and vertebral bone. He began treatment with cefoxitin, amikacin, and tigecycline and underwent epidural abscess evacuation and vertebral debridement without placement of hardware. Tissue cultures were positive for *M. abscessus*. Cefoxitin was changed to imipenem due to worsening transaminitis and clofazimine was added to his regimen. Amikacin was changed to bedaquiline when he developed vestibular toxicity and a mild creatinine elevation. Due to ongoing back discomfort, MRIs were obtained two months and again five months into therapy which revealed ongoing discitis and a small fluid collection, but a repeat aspiration was culture-negative at 60 days. Clofazimine was discontinued due to QT prolongation and imipenem, tigecycline, and bedaquiline were continued. Shortly thereafter, imipenem was changed to meropenem/vaborbactam based on a lower MIC (Table 1), and tigecycline was discontinued due to continued transaminitis.
He continued to have mild intermittent drug-induced transaminitis and thrombocytopenia. A switch to an all-oral regimen was planned; he did not tolerate a trial of linezolid due to finger and toe paresthesias, so omadacycline was added to his bedaquiline to complete a two-year total course of therapy. Oral omadacycline was initiated at 300 mg daily without a loading dose. Since omadacycline initiation, his transaminases have improved. He recently completed 24 months of therapy, of which three and a half months were with omadacycline, with no evidence of disease recurrence since treatment discontinuation six months ago.

**Summary**

Four patients received omadacycline for the treatment of culture-positive *Mycobacterium abscessus* disease in 2019 at our institution. Two patients had cutaneous disease, one had pulmonary disease, and one had osteomyelitis and bacteremia. Omadacycline was overall well-tolerated for over seven months of treatment (median 166 days, range 104-227) and was associated with clinical cure in three of the four patients, with the other patient improving with ongoing treatment off omadacycline.

**Discussion**

Intrinsic and acquired resistance to multiple antimicrobials make *M. abscessus* disease challenging to treat, leading one review to describe it as an “antibiotic nightmare” [1]. Due to the prevalence of resistance, susceptibility testing and prolonged combination therapy are recommended for all clinically significant *M. abscessus* diseases. However, *in vitro* susceptibility testing may not necessarily correlate with patient outcomes, particularly in pulmonary disease [2,3,11]. In a 2011 study, Jarand and colleagues found that only 48% of patients with *M. abscessus* pulmonary disease converted to negative cultures without recurrence after treatment, despite ≥85% susceptibility to
azithromycin and amikacin, two of the treatment mainstays and the only agents considered to have adequate correlation between *in vitro* activity and microbiological response [3,12].

Parenteral therapy is used in the majority of *M. abscessus* diseases, posing a significant challenge since patients are on therapy for at least 4 months, and often ≥12 months [2,3,13]. The introduction of novel oral antibiotics with *in vitro* activity against mycobacteria offers the opportunity to complete an extended treatment duration with an all-oral regimen. Oral antimicrobials with *in vitro* activity against rapidly growing mycobacteria include macrolides, fluoroquinolones, oxazolidinones, rifamycins, clofazimine, bedaquiline, and, most recently, omadacycline [7,13]. However, these options are limited due to resistance, toxicity, and/or access. For example, inducible resistance against macrolides is common [5]. Oxazolidinones may cause myelosuppression, peripheral neuropathy, and optic neuritis [14] and clofazimine and bedaquiline may cause QTc prolongation [15,16]. Clofazimine also requires an investigational new drug submission for use in the United States, and bedaquiline has only been approved for treatment of multidrug-resistant tuberculosis [17,18]. While *in vitro* data support these options, clinical data with combinations of these oral antimicrobials are lacking.

In our case series, all four *M. abscessus* isolates were identified to the subspecies level, which is not always performed routinely in clinical practice despite expert recommendation [19]. Three of the four patients had *M. abscessus* subsp. *abscessus*, which has lower response rates compared to *M. abscessus* subsp. *massiliense* [20]. The most active antimicrobials using CLSI M62 breakpoints were amikacin, linezolid, imipenem, and cefoxitin (Table 1). All but one patient had a macrolide-resistant isolate, with the *M. abscessus* isolates for cases 2-4 all returning positive for the inducible *erm41* gene. The MIC of tigecycline ranged from 0.12 to 0.25 mcg/mL. There are currently no clinically...
defined breakpoints to help interpret these values for tigecycline, but a susceptibility breakpoint of ≤0.5 mcg/mL has been proposed [21]. Omadacycline susceptibility testing was performed on one isolate, resulting in the same MIC as tigecycline (0.25 mcg/mL).

Given the variability in susceptibility profiles of the mycobacterial isolates and adverse events experienced, a number of treatment regimens were employed. This mirrors the lack of standard treatment options described by Novosad and colleagues in 2016, where a total of 21 different combinations were utilized in a cohort of 65 patients with *M. abscessus* disease [4]. The most commonly used antimicrobials in our case series besides omadacycline were amikacin (four cases), imipenem (three), and clofazimine (three). All four patients had at least one antimicrobial regimen adjustment, primarily due to potential drug toxicities.

All four patients developed antimicrobial-related toxicities during their extended treatment courses (Table 2). In phase 3 clinical trials, the most common adverse events seen with intravenous omadacycline were nausea (14.9%), vomiting (8.3%), and increased transaminases (5.4%) [22]. Compared to the variety of toxicities seen in patients from our study, omadacycline was relatively well-tolerated. One patient experienced nausea for several days after initiation, and then ended up discontinuing omadacycline in month six of treatment due to recurrent nausea. Notably, that patient did not receive an omadacycline loading dose. In the phase 3 randomized controlled trial that investigated oral omadacycline with a loading dose for acute bacterial skin and skin structure infections, 30% of patients experienced nausea [23], more than double the rate seen with omadacycline use in other phase 3 trials [24,25]. Based on a pharmacokinetic analysis, a dose of 300 mg daily would reach similar concentrations without a loading dose by day 5, which may be adequate in the treatment of NTM infections given their slow growth kinetics as compared to typical
bacteria [26]. Loading doses may be beneficial when the inoculum is high; this strategy was used in two patients who initiated omadacycline prior to source control without adverse events.

*M. abscessus* isolates have been tested against omadacycline in four recent *in vitro* studies [7–9,27]. In one study, the activity of omadacycline for *M. abscessus* treatment was directly compared to the activity of tigecycline using one mycobacterial strain [7]. The MIC for both medications was 4 mcg/mL, but the mycobacterial killing concentration for omadacycline was ≥16 mcg/mL, while tigecycline showed mycobacterial killing at lower concentrations of ≥4 mcg/mL. This *in vitro* difference may not be clinically relevant, however, as omadacycline’s total drug exposure *in vivo* is around three-fold higher than that of tigecycline in plasma, epithelial lining fluid, and alveolar cells [28]. Because of this, the authors concluded that omadacycline and tigecycline may have similar clinical activity against *M. abscessus*.

Another recent study tested tigecycline, omadacycline, and eravacycline against 28 *M. abscessus* isolates [8]. The MIC$_{50}$/MIC$_{90}$ of tigecycline, omadacycline, and eravacycline were 1/2 mcg/mL, 1/2 mcg/mL, and 0.5/1 mcg/mL, respectively. Given the difference in total drug exposure clinically, both omadacycline and eravacycline had more favorable pharmacokinetic-pharmacodynamic profiles than tigecycline. We have utilized omadacycline in our patients over eravacycline due to the convenience of outpatient oral administration as eravacycline is only available in an intravenous formulation. However, in admitted patients with *M. abscessus* infection, eravacycline may be another promising agent, which we used briefly in case 2.

A third published study tested omadacycline, tigecycline, and doxycycline against 24 *M. abscessus* isolates [9]. The MIC$_{50}$/MIC$_{90}$ of omadacycline and doxycycline were 1/2 mcg/mL and >64/>64 mcg/mL, respectively. Tigecycline was tested against 14 of these isolates and showed similar MICs.
omadacycline (MIC$_{50}$/MIC$_{90}$ 1/2 mcg/mL). The tigecycline MICs recorded in our four patients were all ≤0.25 mcg/mL, lower than the MIC data presented in this paper.

The final published study obtained MICs of omadacycline, minocycline, and 11 other antimicrobials against 20 *M. abscessus* isolates [27]. The MIC$_{50}$/MIC$_{90}$ of omadacycline and minocycline were 16/128 mcg/mL and 256/>256 mcg/mL, respectively. The omadacycline MICs in this study differed from the previous *in vitro* studies. Of note, this was the only study to use Middlebrook 7H9 supplemented with 10% oleic acid dextrose citrate (OADC) as a growth medium, while the other studies utilized cation-adjusted Mueller-Hinton broth. The authors point out that omadacycline may potentially degrade during incubation over time, which could explain the higher-than-expected MICs. Degradation was addressed in only one of the previous *in vitro* studies [7].

In the first three studies discussed above, omadacycline MICs correlated well with tigecycline MICs. Despite the growing literature of favorable *in vitro* data for *M. abscessus*, there has been only one report of omadacycline clinical use in this situation [29]. This case series provides the most clinical data published on the use of omadacycline for *M. abscessus* disease to date. This is also the largest data set describing the long-term use of omadacycline in humans. Omadacycline was well-tolerated for up to 13 weeks in toxicity studies in rats and monkeys [30]. Here, we show tolerability for over seven months of omadacycline therapy, with all four patients receiving at least three months of omadacycline and only one patient experiencing an adverse event in month six of treatment.

As a small single-center retrospective case series, no definitive conclusions can be made from our use of omadacycline in *M. abscessus* disease. With a diverse group of patients and no control group, conclusions about efficacy cannot be drawn. All patients received combination therapy with multiple
antimicrobial agents, and two of the four patients received omadacycline for less than half of their total treatment durations. Surgical source control is also likely just as important as medical management in *M. abscessus* disease, and all four of our patients underwent surgical management [31].

**Conclusions**

In four patients who received omadacycline for *M. abscessus* disease at our institution, omadacycline was relatively well-tolerated in long-term treatment. This case series and the published *in vitro* data make omadacycline a promising alternative agent in the management of nontuberculous mycobacterial infections, especially as part of an all-oral regimen. Further data are required to determine omadacycline’s definitive role in the treatment of *M. abscessus* disease.
Conflicts of Interest

All authors report no conflicts of interest relevant to this work

Patient Consent Statement

All patients provided verbal consent for this case series to be published. The case series received exempt status from the Partners HealthCare Institutional Review Board (protocol 2019P003244). HIPAA privacy rules have been followed.

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Funding

S.J.V. received funding from the Multidisciplinary AIDS Training Program supported by the National Institute of Allergy and Infectious Diseases (NIAID) at the National Institutes of Health [grant number T32 AI007387]. G.E.V. received funding from the NIAID [grant numbers K08 AI141740, L30 AI120170, and P30 AI060354], the Dr. Lynne Reid/Drs. Eleanor and Miles Shore Fellowship at Harvard Medical School, the Burke Global Health Fellowship at the Harvard Global Health Institute, and the Harvard University Center for AIDS Research.
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Table 1: Minimum Inhibitory Concentration (MIC) Results

| Antimicrobial                  | Isolate 1 | Isolate 2 | Isolate 3 | Isolate 4 |
|--------------------------------|-----------|-----------|-----------|-----------|
| Amikacin                       | 32        | I         | 8         | S         | 8         | S         | 16        | S         |
| Cefoxitin                      | 128       | R         | 32        | I         | 32        | I         | 64        | I         |
| Ciprofloxacin                  | >4        | R         | >4        | R         | >4        | R         | >4        | R         |
| Clarithromycin                 | 2         | S         | >16       | R         | R         | R         | R         | R         |
| Doxycycline                    | >16       | R         | >16       | R         | 16        | R         | >16       | R         |
| Imipenem                       | 32        | R         | 8         | I         | 16        | I         | 8         | I         |
| Linezolid                      | 16        | I         | 16        | I         | 16        | I         | 8         | S         |
| Minocycline                    | >8        | R         | >8        | R         | >8        | R         | >8        | R         |
| Moxifloxacin                   | >8        | R         | 8         | R         | >8        | R         | >8        | R         |
| Trimethoprim/ Sulfamethoxazole | >8/152    | R         | 4/76      | R         | 8/152     | R         | 8/152     | R         |
| Tigecycline                    | 0.25      | 0.12      | 0.25      | 0.25      |
| Clofazimine                    | 0.5       | 0.5       | 0.5       |
| Bedaquiline                    | 0.12      | 0.12      |
| Meropenem/vaborbactam          | >8/8      | 4/8       |
| Omadacycline                   | 0.25      |           |

\(^a\) All susceptibility testing was performed at The University of Texas Health Science Center at Tyler (cases 2-4) or the Laboratory Corporation of America (case 1) following CLSI M24 performance standards for susceptibility testing.

\(^b\) Meropenem/vaborbactam susceptibility testing for isolate 4 was performed at a research laboratory at Harvard Medical School using an alamarBlue cell proliferation assay.
Table 2: Case Summaries

| Case # | Organism Isolated | M. abscessus Infection Location | Omadacycline Loading Dose | Omadacycline Maintenance Dose | Antimicrobials Prior to Omadacycline | Concurrent Antimicrobial s | Surgical Management | Treatment Duration (days) | Resolution | Possible Treatment-Related Adverse Events |
|--------|-------------------|--------------------------------|---------------------------|-------------------------------|--------------------------------------|---------------------------|---------------------|-------------------------|-----------|------------------------------------------|
| 1      | *Mycobacterium abscessus* subsp. *bolletii* | Skin (back, breast) | 450 mg daily x2 doses | 300 mg daily | • clarithromycin, TMP-SMX  
• clarithromycin, linezolid  
• amikacin, clarithromycin, tigecycline  
• azithromycin, linezolid, tigecycline | • azithromycin, linezolid  
• azithromycin, tigecycline  
• amikacin  
• clarithromycin  
• linezolid | Yes: implant removal, multiple debridements & drainages | Total: 264  
Omad: 173 | Cure | Linezolid: cytopenias, paresthesias |
| 2      | *Mycobacterium abscessus* subsp. *abscessus* | Pulmonary | 450 mg daily x2 doses | 300 mg daily | N/A | • amikacin, imipenem, clofazimine  
• clofazimine, tedizolid  
• clofazimine, imipenem & eravacycline | Yes: wedge resection, lobectomy | Total: 227  
Omad: 227 | Cure | Amikacin: hearing loss, renal impairment  
Tedizolid: fatigue & dizziness |
| 3      | *Mycobacterium abscessus* subsp. *abscessus* | Skin (abdominal wall) | N/A | 300 mg daily | • azithromycin, linezolid, amikacin, imipenem | • azithromycin, amikacin  
• azithromycin, clofazimine | Yes: multiple abdominal wall washouts & debridements | Total: >332  
Omad: 159 | Ongoing, but improving | Imipenem: serum sickness  
Amikacin: tinnitus  
Omadacycline: nausea & vomiting |
|   | Mycobacterium abscessus subsp. abscessus | Blood, Vertebrae | N/A | 300 mg daily | amikacin, cefoxitin, tigecycline | imipenem, tigecycline, clofazimine, bedaquiline | meropenem-vaborbactam, bedaquiline, tigecycline | bedaquiline | Yes: epidural abscess evacuation & vertebral debridement | Total: 731 | Omada: 104 | Cure | Cefoxitin, tigecycline, bedaquiline: transaminitis. Clofazimine: QTc elevation. Amikacin: vestibular symptoms & mild creatinine elevation. Bedaquiline: thrombocytopenia. Linezolid: numbness of extremities |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|

*During case 2's omadacycline course, there was a brief switch to imipenem & eravacycline when the patient was NPO for 7 days.*

TMP-SMX: trimethoprim-sulfamethoxazole