Medical management of atraumatic *Mycobacterium abscessus* cutaneous infection: A case report

Chong Wei Tiong[a],*, Thomas Nack[a], Alex Y.C. Tai[a,b], N. Deborah Friedman[a,b]

[a] *Department of General Medicine, Barwon Health, Geelong, Victoria, Australia*
[b] *Department of Infectious Diseases, Barwon Health, Geelong, Victoria, Australia*

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ABSTRACT

Treatment for cutaneous infection from *Mycobacterium abscessus* is fraught with poorly established evidence. Given its antibiotic multi-resistance, surgical intervention is often recommended. We report a case of cutaneous *M. abscessus* infection that was successfully managed with medical therapy alone. A 55-year-old immunocompetent woman from the Bellarine peninsula in Victoria, Australia presented to our hospital with a 2-week history of a non-healing ulcer on her left forearm. The patient had no history of trauma or procedures to the skin. On presentation, the patient had a punch biopsy, which was culture positive for *M. abscessus*. The isolate was susceptible to clarithromycin and amikacin, had intermediate susceptibility to ciprofloxacin, cefoxitin and linezolid and was resistant to doxycycline, imipenem, cotrimoxazole and moxifloxacin. The tigecycline MIC was 0.25 μg/ml. The patient received a total of 12 weeks of oral clarithromycin 500 mg twice daily, 4 weeks of intravenous amikacin 500 mg daily, 6 weeks of intravenous tigecycline 100 mg over 24 hours via Baxter pump, and 4 weeks of oral clofazimine 100 mg daily. The patient made a good clinical recovery and had her medical therapy ceased after 12 weeks. *M. abscessus* cutaneous infection in an immunocompetent individual without antecedent trauma or surgery is rare. Our case illustrates the successful treatment of a deep *M. abscessus* cutaneous ulcer with relatively short duration macrolide-based antibiotic therapy without any surgical intervention.

1. Introduction

Treatment of *Mycobacterium abscessus* infection is fraught with poorly established evidence and is often difficult. Outcomes are dependent on the location and extent of disease, host immune and antibiotic susceptibility status. Macrolide activity remains an important prognostic factor in treatment outcomes[1,2]. As *M.abscessus* is intrinsically resistant to numerous antibiotics, surgical intervention is often recommended as an adjunct to antibiotics. We report a case of cutaneous *M.abscessus* infection that was successfully managed with medical therapy alone.

2. Case report

A 55-year-old immunocompetent woman from the Bellarine peninsula, in Victoria Australia, was referred by her general practitioner (GP) to the Infectious Disease Clinic at the regional tertiary care hospital with a 2 week history of a non-healing purulent ulcer on her left

forearm, which started as a pustule and rapidly worsened with increasing pain, erythema and edema over the subsequent few days (Fig. 1). Her past medical history included lumbar disc prolapse, and bipolar mood disorder, which was effectively managed with oral quetiapine 150 mg daily. The patient had travelled to Thailand on vacation 4 weeks prior to admission but had no history of skin trauma or water contact. The patient also denied any skin tattooing or surgical cosmetic procedures on the trip. She had been initially commenced on oral cephalexin by her GP for the ulcer.

On examination, the patient had an ulcer 1 cm × 1 cm (0.4 inch × 0.4 inch) with surrounding erythema of her forearm. Aside from a raised C-reactive protein at 74.4 mg/L and mildly raised alkaline phosphatase at 146 U/L, her laboratory tests were unremarkable (Table 1). A wound swab performed prior to commencement of cephalexin was acid-fast bacilli smear positive, with cultures pending.

A punch biopsy was performed, which revealed features of acute inflammation with a perivascular lymphocytic infiltrate in the mid and deep dermis and a heavy neutrophil infiltrate throughout the dermis

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* Corresponding author at: Department of General Medicine, University Hospital Geelong, 272-322 Ryrie Street, Geelong, 3220, VIC, Australia.
E-mail addresses: CHONG.TIONG@barwonhealth.org.au (C.W. Tiong), THOMAS.NACK@barwonhealth.org.au (T. Nack), alexyctai@gmail.com (A.Y.C. Tai), DEBORAHF@barwonhealth.org.au (N.D. Friedman).

Fig. 1. Successful treatment of *M. abscessus* cutaneous infection with 12 weeks of antibiotic therapy.

Table 1
Laboratory results.

Blood test	Week 0	Week 6	Week 7	Week 8	Week 9	Week 12	Reference range
Hemoglobin (g/dL)	12.1	13.4	12.3	14.0	12.4	13.0	11.5–17.0
White cell count (μL)	7700	5600	6200	6100	5300	7100	4000–11,000
Neutrophil count (μL)	5300	1900	2100	2800	2100	4300	2000–8000
Platelets (μL)	226,000	177,000	194,000	214,000	186,000	307,000	150,000–500,000
Sodium (mEq/L)	141	142	143	141	142	142	136–145
Potassium (mEq/L)	3.5	4.1	4.3	4.1	4.3	4.2	3.5–5
Bicarbonate (mEq/L)	28	27	26	27	27	24	22–32
Urea nitrogen (mg/dL)	7	27.73	37.25	25.89	25.77	10.64	7–19.61
Creatinine (mg/dL)	0.86	0.94	1.26	1.02	1.03	0.9	0.34–1.02
eGFR (mL/min)	76	69	48	62	61	72	>90
C-reactive protein (CRP) (mg/L)	74.4	<2.9	<2.9	<2.9		<2.9	>2.9
Albumin (g/dL)	3.6				2.6	4.1	3.4–5.0
Alkaline phosphatase (U/L)	146				256	94	20–120
Gamma-glutamyl transpeptidase (U/L)	19				36	50	<30
Alanine transaminase (U/L)	28				54	42	<45
Total bilirubin (mg/dL)	0.23				0.88	0.47	0.06–1.17
haemoglobin (g/dL)	12.1	13.4	12.3	14.0	12.4	13.0	11.5–17.0
White cell count (microL)	7700	5600	6200	6100	5300	7100	4000–11,000
Neutrophils (microL)	5300	1900	2100	2800	2100	4300	2000–8000
Platelets (x10³/microL)	226	177	194	214	186	307	150–500
Sodium (mEq/L)	141	142	143	141	142	142	136–145
Potassium (mEq/L)	3.5	4.1	4.3	4.1	4.3	4.2	3.5–5
Bicarbonate (mEq/L)	28	27	26	27	27	24	22–32
Urea (mg/dL)	7	27.73	37.25	25.89	25.77	10.64	7–19.61
Creatinine (mg/dL)	0.86	0.94	1.26	1.02	1.03	0.9	0.34–1.02
eGFR (mL/min)	76	69	48	62	61	72	>90
CRP (mg/L)	74.4	<2.9	<2.9	<2.9		<2.9	>2.9
Albumin (g/dL)	3.6				2.6	4.1	3.4–5.0
ALP (U/L)	146				256	94	20–120
GGT (U/L)	19				36	50	<30
ALT (U/L)	28				54	42	<45
Total bilirubin (mg/dL)	0.23				0.88	0.47	0.06–1.17

and all levels of the subcutaneous tissue. No acid-fast bacilli were identified on Ziehl-Neelsen or Wade-Fite stains. As the ulcer was clinically suspicious for *Mycobacterium ulcerans*, the patient was empirically commenced on rifampicin and clarithromycin. The quetiapine dose was reduced to 50 mg daily due to potential interactions with clarithromycin causing QT prolongation.

The *M.ulcerans* polymerase chain reaction (PCR) returned as negative, and preliminary results of the wound swab revealed a rapidly growing mycobacterium, with suspicion of *M.abscessus*. Rifampicin was therefore ceased, and replaced with doxycycline 100 mg twice daily to accompany clarithromycin at 500 mg twice daily. A third antimicrobial agent, oral linezolid, was added to the antibiotic regimen at a dose of
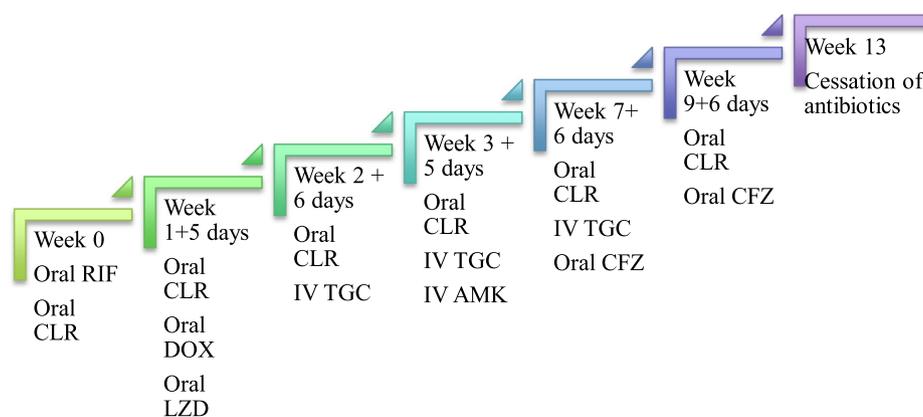
RIF=Rifampicin; CLR= Clarithromycin; DOX= Doxycycline; LZD= Linezolid; TGC=
Tigecycline; AMK=Amikacin; CFZ= Clofazimine

Fig. 2. Summary of antibiotics received.

400 mg daily (instead of 600 mg daily due to the potential drug inter-action of clarithromycin increasing serum concentrations of linezolid) [3]. After 7 days, *Mycobacterium abscessus* was isolated from wound culture. The isolate was susceptible to clarithromycin and amikacin, had intermediate susceptibility to ciprofloxacin, cefoxitin and linezolid and was resistant to doxycycline, imipenem, cotrimoxazole and moxi-floxacin. The minimum inhibitory concentration (MIC) for tigecycline was 0.25 μg/ml.

The patient was thus commenced on intravenous (IV) tigecycline 100 mg over 24 hours, continued on clarithromycin, while doxycycline and linezolid were ceased. One week later (week 4), with no im-provement in the ulcer clinically (Fig. 1), IV amikacin 500 mg daily was commenced, while continuing IV tigecycline and oral clarithromycin. Further susceptibility testing for clofazimine was requested at an in-terstate laboratory, and demonstrated an MIC of < 2 μg/mL. At the end of week 7, mild nephrotoxicity necessitated cessation of IV amikacin and replacement with oral clofazimine 100 mg daily (Fig. 2). The pa-tient developed mild liver function derangement (see Table 1) and peripheral edema, which were thought to be attributable to either ti-gecycline or the combined effect of both clofazimine and tigecycline. Her liver function improved after cessation of tigecycline at week 9, by which time there had been significant clinical improvement in the ulcer (Fig. 1). The quetiapine dosage was increased to 100 mg daily, as there was a decline in the patient's mood, presumed to be due to clofazimine reducing the serum concentration of quetiapine.

The patient made good clinical recovery and had all remaining antibiotics (clofazimine and clarithromycin) ceased after 12 weeks (Fig. 2). No surgical debridement was performed. Complete healing took place at 13 weeks with the skin defect healing via secondary in-tention (Fig. 1).

3. Discussion

Mycobacterium abscessus complex is a rapidly growing atypical my-cobacterium, which may be difficult to treat due multiple factors in-cluding; a scarcity of evidence for optimal treatment regimens, a poor correlation between in vitro antiobiotic sensitivities and in vivo results, multidrug resistance, and often long duration multi-antibiotic regimens which increase the risk of antibiotic adverse effects[4,5]. Latest taxo-nomic studies have subclassified *M. abscessus* into *M. abscessus sensu stricto*, *M. massiliense*, and *M. bolletii*, all of which differ in terms of antibiotic resistance profiles [6,7].

M. abscessus infection has a wide array of clinical manifestations, with the most common ones being pulmonary infections (especially in individuals with cystic fibrosis, chronic obstructive pulmonary disease

and bronchiectasis), and cutaneous soft tissue infections, from post-traumatic wounds or surgical wounds. Soft tissue infections may man-ifest as abscesses, ulcers, erythematous violaceous nodules and plaques, cellulitis, and sinuses. Post-surgical soft tissue infections make up 45% of extrapulmonary infections, and there have been previous large out-breaks of healthcare-associated extrapulmonary disease[4]. Other *M. abscessus* infections that have been described in the literature include: endocarditis, osteomyelitis, tympanoplasty infections, mastoiditis, en-dophthalmitis, keratitis, disseminated and deep seated infections[4,8]. *M. abscessus* is found in soil and in both tap and distilled water, as it is chlorine resistant [1,9].

M. abscessus is highly resistant to many antimicrobials in vitro and in vivo, including conventional antituberculous drugs [6]. Due to sub-optimal treatment outcomes in studies, there is insufficient clinical evidence for standardised treatment guidelines [4]. Similar to pul-monary infection, current practice for managing cutaneous infections is surgery and combination antibiotics. For cutaneous infections, the re-commended antibiotics by The American Thoracic Society and The Infectious Diseases Society of America are clarithromycin 1 g/day or azithromycin 250 mg/day together with parenteral antibiotics (cefox-itin plus either amikacin or imipenem). The minimum recommended duration is 2 weeks for amikacin and 4 months for total antibiotic therapy [10].

Macrolides like clarithromycin are cornerstone agents against *M. abscessus*, While *M.massiliense* is usually macrolide susceptible, con-ferring better treatment outcomes, *M. abscessus sensu stricto* commonly expresses the *erm(41)* gene, which may induce clarithromycin re-sistance [6,7]. Intrinsic resistance may occur due to point mutations in the *rrl* gene [4,6–8]. Resistance to trimethoprim/sulfamethoxazole and fluoroquinolones is highly prevalent in *M. abscessus* [6,7], and the or-ganism is resistant to most other beta lactams, except imipenem and cefoxitin, due to its production of the broad spectrum beta-lactamase Bla$_{Mab}$. Imipenem and cefoxitin typically have moderate in-vitro ac-tivity against M. abscessus [11,12]. Imipenem is not hydrolysed by Bla$_{Mab}$, but slowly inactivated, hence addition of an effective beta-lac-tamase Bla$_{Mab}$ inhibitor, such as avibactam, is recommended [12,13]. Classical beta-lactamase inhibitors, like clavulanate, sulbactam and tazobactam are however ineffective against Bla$_{Mab}$. [13]. For isolates that are susceptible to imipenem, combination therapy with rifabutin can be considered, as there is moderate in-vitro synergistic activity [13].

Amikacin has good activity against most *M.abscessus* strains, but as is the case with other aminoglycosides, the use of amikacin is limited by the potential for causing nephrotoxicity and ototoxicity [6,14,15]. *M. abscessus* is sensitive to linezolid in-vitro, however long-term use is

problematic due to the hematological and neurological side effects [4]. A newly developed oxazolidinone antimicrobial, tedizolid, an intravenous once daily agent, has more effective *in-vitro* bacteriostatic activity against *M. abscessus*, with less hematological and neurological toxicity compared to linezolid [16–18].

Clofazimine, primarily an antileprosy drug, has resulted in 81% treatment response rates in *M. abscessus* pulmonary disease [19]. However the clinical breakpoint for clofazimine testing is not certain, with the susceptible reference range of $< 2 \, \mu g/ml$ being extrapolated from previous studies[20,21]. Via the microdilution method and the agar dilution method in two separate studies, the MIC of clofazimine was identified as $0.063 \, \mu g/ml$ and $5 \, \mu g/mL$ respectively [20,21]. Tigecycline, a glycylcycline which has been used successfully as salvage therapy for difficult-to-treat infections works synergistically with clarithromycin and clofazimine. However, it can cause severe gastrointestinal side effects [4,21,22]. Omadocycline, a novel oral once-daily tetracycline has good *in-vitro* activity against *M. abscessus*, with bactericidal effect at concentrations $\geq 16 \, mg/L$, and possibly less gastrointestinal side effects. However, its *in-vitro* bactericidal effect is not as potent as that of tigecycline [23]. Bedaquiline has demonstrated promising results for the treatment of *M.abscessus* infections, usually in combination with other drugs such as clofazimine [24,25]. There is also promising research using engineered bacteriophage therapy to treat M. abscessus infection [26].

It is difficult to successfully treat *M. abscessus* cutaneous infection with medical therapy alone, although this practice is becoming more common. A study of an outbreak of 240 post-surgical *M. abscessus* cutaneous infections in Colombia, showed that clarithromycin monotherapy is less effective compared to a combination of surgery and antibiotic therapy, with a treatment response of 23% and 95% respectively [9]. A case report by Chan et al.[27] described a 79-year-old woman with an erythematous plaque with pustules in the antecubital fossa, who was treated with 6 months of clarithromycin 500 mg, ciprofloxacin 500 mg and doxycycline 100 mg twice daily. Another report of a 55-year-old woman who developed a papular and nodular rash from *M. abscessus* along her cholecystectomy incisional scar, was treated empirically with cefoxitin, clarithromycin and moxifloxacin, and completed a 6-month course of clarithromycin and moxifloxacin [28]. And Llenas-Garcia et al. [29] reported the case of a 32-year-old woman who developed nodules along her abdominoplasty incisional scar, and received initial treatment with cefoxitin, amikacin, clarithromycin and levofloxacin for 8 weeks, followed by linezolid and clarithromycin for a total treatment duration of 38 weeks.

Our case illustrates the successful treatment of a deep *M. abscessus* cutaneous ulcer with relatively short duration macrolide-based antibiotic therapy but without any surgical intervention. The success of treatment in this case was likely heavily influenced by the localized cutaneous nature of the disease, the fact that our patient is immunocompetent and the susceptibility of the organism to clarithromycin. It is rare to have a *M. abscessus* cutaneous infection in an immunocompetent individual without antecedent trauma or surgery [30–34]. Further clinical studies will be helpful in formulating clinical guidelines for the treatment of *M. abscessus* cutaneous infections.

Funding

Ethical approval

None required.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images/photographs.

Data for reference

Not applicable.

Declaration of Competing Interests

None.

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