J Clin Tuberc Other Mycobact Dis 21 (2020) 100183

Contents lists available at ScienceDirect

J Clin Tuberc Other Mycobact Dis

journal homepage: www.elsevier.com/locate/jctube

Surgical cure of clarithromycin resistant *Mycobacterium chelonae* breast implant infection: A case report and review of the literature

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ARTICLE INFO

Keywords:
NTM
Mycobacterium chelonae
Rapidly growing mycobacteria
Breast implant infection
Lipotourism
Medical tourism

ABSTRACT

Clusters of patients who obtain cosmetic surgeries abroad have developed surgical site infections due to rapid growing non-tuberculous mycobacteria (NTM). These are usually treated with a combination of surgery and months of anti-mycobacterial therapy, but poor outcomes, including permanent scarring are common. We present a case of a 36-year-old female who developed a clarithromycin-resistant *M. chelonae* (CRMC) infection after undergoing breast augmentation in the Dominican Republic. She underwent debridement and explant of her silicone implants, but due to a series of complications including discordant antimicrobial susceptibility testing profiles, GI side effects, and then pregnancy, she was unable to receive typical multidrug anti-mycobacterial therapy after surgery. She received close clinical follow up and demonstrated full recovery without any evidence of recurrence of infection at 9 months of follow up. We searched the literature for cases of NTM surgical site infection after breast surgery. To our knowledge, this is the first case report of confirmed NTM breast implant infection being cured with surgery alone, and only the second report of clarithromycin resistant *M. chelonae* in a patient without disseminated infection or pre-exposure to macrolides. The increasing prevalence of drug resistant NTM infections is an emerging concern for clinicians treating patients with complications related to medical tourism.

1. Case presentation

A 36-year-old previously healthy female presented to our hospital with erythema and pain of the bilateral breasts one month after undergoing elective breast augmentation with textured silicone implants in the Dominican Republic.

After a reportedly routine intra-operative course, she developed a post-operative hematoma in the subsequent days on the left breast which was treated with percutaneous aspiration by the original surgeon. She denied other exposures such as swimming or hot tub use and returned to the United States two weeks after her surgery. Within days of her return, she started to notice erythema over the left breast. She presented for evaluation at two initial hospitals where she was diagnosed with cellulitis and prescribed short courses of antibiotics including dicloxacillin, trimethoprim-sulfamethoxazole and ciprofloxacin. When the erythema and pain progressed, she presented to a third hospital, and was given IV vancomycin. During this hospitalization, she developed a fever which prompted breast ultrasound that revealed bilateral fluid collections. She was transferred to our hospital on vancomycin and ciprofloxacin, about 1 month after her initial surgery.

During bilateral implant explantation, murky fluid was encountered, and the pockets were copiously irrigated and bilateral Blake drains were placed. Operative samples were sent for bacterial, fungal, and mycobacterial cultures. Gram's and Acid-Fast Bacilli (AFB) stains were negative. The aerobic/anaerobic bacterial culture grew mixed skin flora that included *Cutibacterium acnes* and *Micrococcus* species, which were thought unlikely to be pathogenic. Her fevers resolved after surgery and her surgical team transitioned her from vancomycin to trimethoprim/sulfamethoxazole and ciprofloxacin. She was not treated with macrolides at any point during this initial course. Four days post debridement, both implant AFB cultures were positive for growth of a rapid-growing mycobacterium; confirmed as *Mycobacterium chelonae*

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https://doi.org/10.1016/j.jctube.2020.100183

Table 1
Antimicrobial Susceptibility Testing profile of M. chelonae, performed twice at two different national reference laboratories. The second sets of AST were performed several months after the original isolate was obtained; the isolate used for this 2nd set of AST was re-isolated and grown from an original agar slant that was saved at our hospital's clinical microbiology laboratory. Clarithromycin was initially reported as susceptible by reference laboratory #2, but all other AST tests demonstrated resistance to clarithromycin. Interpretations for several antibiotics were provided by laboratory #2 based on a laboratory developed broth microdilution test that has not been cleared by the U.S. Food and Drug Administration (FDA).

Antimicrobial agent	Antimicrobial Susceptibility Testing results by laboratory:MIC (Interpretation)			
	Laboratory #1; initial AST	Laboratory #2; initial AST	Laboratory #1; repeat AST	Laboratory #2; repeat AST
Routinely tested antimicrobial agents with breakpoints				
Amikacin	32 (I)	< =8 (S)	16 (S)	< =8 (S)
Cefoxitin	> 128 (R)	> 128 (R)	> 128 (R)	> 128 (R)
Ciprofloxacin	> 4 (R)	2 (I)	> 4 (R)	8 (R)
Clarithromycin	**16 (R)**	**2 (S)**	**8 (R)**	**16 (R)**
Doxycycline	> 16 (R)	> 16 (R)	> 16 (R)	> 16 (R)
Imipenem	16 (I)	8 (I)	32 (R)	> 16 (TR)
Linezolid	16 (I)	8 (S)	16 (I)	16 (I)
Minocycline	> 8 (R)		> 8 (R)	
Moxifloxacin	> 8 (R)	2 (I)	> 8 (R)	> 4 (R)
Tobramycin	2 (S)	> 16 (R)	2 (S)	< =2 (S)
Trimethoprim/sulfamethoxazole		4/76 (R)		4/76 (R)
Additional antimicrobial agents, without established breakpoints [1–2]				
Clofazimine		< =0.5 (S)		< =0.5 (S)
Tigecycline	0.25 (NP)	< =0.25 (TS)	0.12 (NP)	< =0.25 (TS)
Kanamycin		< =8 (TS)		< =8 (TS)

Abbreviations: AST, antimicrobial susceptibility testing; S, susceptible; I, intermediate susceptible; R, resistant; TS, tentative interpretation susceptible; TR, tentative interpretation resistant; NP, not provided; TMP/SMX, Trimethoprim-Sulfamethoxazole.

using Matrix-Assisted Laser Desorption- Ionization-Time of Flight Mass Spectrometry (MALDI-TOF) at the State laboratory.

Given her clinical stability without fever, pain, or worsening erythema and unpredictable drug resistance pattern of *M. chelonae*, empiric anti-mycobacterial therapy was not pursued while antimicrobial susceptibility testing (AST) was pending. Surprisingly, the *M. chelonae* isolate was found to be resistant to clarithromycin upon initial AST at the primary reference laboratory (Table 1). Given the rarity of clarithromycin resistant *M. chelonae* (CRMC) in treatment naïve patients, AST was repeated at a second, independent reference laboratory, which reported that the *M. chelonae* isolate was clarithromycin susceptible (Table 1). The AST performed at a national reference laboratory included testing for routine antimicrobial agents with established breakpoints against RGMs, as well as newer antimicrobial agents, for which there is insufficient data to establish breakpoints [1,2]. During this period, the patient was followed in the clinic every two to four weeks and demonstrated no evidence of cellulitis or recurrent infection.

Given the patient's high burden of disease at presentation and general consensus that antimycobacterial therapy is warranted for this type of infection [3], our plan was to treat her with a prolonged course of combination antimycobacterial therapy. However, given the discordant clarithromycin susceptibility testing results, we requested repeat AST (for the *M. chelonae* isolate from our original agar slant) at both reference laboratories, which confirmed clarithromycin resistance (Table 1). It is unclear what lead to the initial discordant AST at the second laboratory. We hypothesize that this could have been due to a mixed population of NTMs, not identified by MALDI-TOF, since an AST error is less likely given that the MICs for several antibiotics were different.

Once the final AST results were available, two months after surgical washout at which time she remained stable without evidence of recurrence of infection, we started the patient on linezolid 600 mg daily and azithromycin 500 mg daily. Five days into this regimen, the patient developed intolerable GI side effects, not modifiable by taking medication with food, and the patient self-discontinued her antimycobacterial regimen. Due to the patient's strong desire to avoid intravenous therapy, we planned for an alternative oral treatment regimen of bedaquiline, omadacycline, and clofazimine [4,5]. We inferred that three active agents should be used to treat *CRMC* from the recent NTM guideline recommendations for the treatment of *M. Abscessus* [6]. Although there is a limited body of evidence correlating AST results with clinical outcomes for drug-resistant NTM such as M. chelonae, expert guidance suggests that phenotypic AST can inform treatment decisions, particularly when antibiotic options are limited [7].

After obtaining approvals for these antibiotics, when she returned for follow up, now four and a half months after the original surgical washout, the patient revealed that she was pregnant. We discussed that in the absence of clinical signs of infection and the potential fetal toxicity of this regimen, clinical observation was warranted.

The patient was seen again about nine months after her explantation and washout, at which time she was twenty-two weeks pregnant and there were no notable symptoms, erythema, or signs of infection. We discussed expectant management, with ID follow-up as needed, and recurrence has not been reported.

2. Literature review

2.1. Epidemiology and management of NTM infections after breast surgery

We searched PubMed and Embase for cases of NTM infection after breast surgery to identify cases with surgical cure. Using the search strategy outlined in the appendix, we identified 25 case reports, series, or reviews. We reviewed these 25 manuscripts with a total of 269 patients described [3–27]. NTM infection after "lipotourism," is a well described phenomenon in various parts of the world in the last two decades [14,16,20,26–30]. Most cases of NTM infection after breast surgery are treated with surgery (typically requiring explantation) and anti-mycobacterial therapy. Although cases of successful reimplantation have been reported [31,32], even with effective therapy, relapse rates are high [9,32]and scarring may lead to undesirable cosmetic outcomes [9]. Of these 269 patients, only three (0.01%) were successfully treated with surgical debridement alone. These three cases were all part of a 15 patient series that arose from an outbreak in Israel in 2003, finally leading to the isolation of a new species of rapid

Table 2
Reported cases of CRMC. Abbreviations: N, Number of Patients; M, Male; F, Female; RLE, Right Lower Extremity; LLE, Left Lower Extremity; c/b, complicated by; COPD, Chronic Obstructive Pulmonary Disease.

Author (Year)	N	Clinical History	Disseminated	Previous Macrolide Exposure	Surgery	Treatment Regimen	When CRMC identified	Clinical Outcome
Schwartz et. al (2018) [34]	4	Archived isolates from Cystic Fibrosis patients	Not described	Unknown, but likely	Not Described	Not described	Not described	Not described
Churgin et. al (2018) [33]	1	56 M scleral buckle (placed 20 years previous for retinal detachment) infection	No	Not described	Yes	linezolid, clarithromycin, IV Imipenem × 3 weeks	Prior to treatment	Improved
Mannelli et. al (2018) [41]	1	47 M prosthetic hip infection + skin lesions. Treated with Amikacin, Tigecycline, and Azithromycin initially; but hip collections grew and CRMC was identified, and patient pursued hospice	Yes	Yes; as part of initial therapy with amikacin, tigecycline, azithromycin	No	IV Amikacin, IV tigecycline, azithromycin	8 weeks into treatment	Declined further therapy after treatment failure; pursued hospice
Brown-Elliott et. al (2001) [35]	1	57 M chronic steroids (Myasthenia Gravis); multiple skin nodules on RLE; treated with clarithromycin monotherapy, developed worsening nodules and CRMC was identified and taken for debridement and given clarithromycin + tobramycin; eventually had worsening nodules again, then treated with IV linezolid effectively	No, but many lesions on RLE	Yes; as part of initial regimen; clarithromycin monotherapy	Yes; after first treatment failure	1) clarithromycin monotherapy2) Surgery + clarithromycin + tobramycin3) IV linezolid	1) 4 months into clarithromycin monotherapy2) After 2nd treatment failure after surgery	Improved with IV linezolid
Vemulapalli et. al (2001) [36]	1	65F chronic steroids (COPD), disseminated cutaneous lesions, developed resistance on clarithromycin monotherapy	Yes	Yes; as part of initial regimen of clarithromycin monotherapy	No	1) clarithromycin monotherapy2) TMP-SMZ + Ciprofloxacin	4 months into therapy when new nodules arose after initial response	Improved nodules; not fully resolved
Bañuls et. Al (2000) [37]	1	66F chronic steroids (dermatomyositis), disseminated cutaneous lesions, developed resistance on clarithromycin and ciprofloxacin	Yes	Yes; as part of initial regimen of clarithromycin monotherapy	No	1) clarithromycin and ciprofloxacin2) minocycline and clarithromycin	2 months into therapy when nodules recurred	Improved skin lesions; died of metastatic vulvar cancer
Driscoll et. al (1997) [38]	1	66F chronic steroids (pemphigus vulgaris), multiple lesions on L lower extremity (LLE); developed resistance on clarithromycin monotherapy	No, but multiple lesions on LLE	Yes; as part of second regimen of clarithromycin monotherapy	No	1) Minocycline (no response)2) Clarithromycin (rapid response, then recurrence)3) Erythromycin4) tobramycin (developed AKI)5) palliative ciprofloxacin and azithromycin (no improvement)	2 months into therapy when nodules recurred	Did not improve
Tebas et al. (1995) [39]	1	60 M orthotopic heart transplant c/b rejection (prednisone, azathioprine, cyclosporin), bilateral arm lesions, developed resistance on clarithromycin monotherapy	Yes	Yes; as part of initial regimen of clarithromycin monotherapy	No	1) clarithromycin monotherapy2) imipenem and tobramycin (tobramycin stopped due to AKI)	3 months on therapy	All antibiotic therapy was stopped due to lack of effective options, died of other causes
Wallace et al. (1993) [40]	1	39F with multiple sclerosis on immunosuppression (not specified), disseminated cutaneous disease, developed resistance after self-discontinuing clarithromycin monotherapy at 3.5 months	Yes	Yes; as part of trial regimen of clarithromycin monotherapy	No	Clarithromycin monotherapy, then self-discontinued	1 month after self-discontinuing her therapy	Not provided

growing NTM (*M. jacuzzi)* named after the fact the pathogen was isolated from one of the surgeon's hot tub. Notably, all three of these cases were "presumptive" cases identified in retrospective review, they were not sent for acid-fast bacilli culture [14]. All 269 cases were reviewed for microbiology data as well; while most cases were caused by *M. fortuitum*, zero cases involved CRMC.

2.2. Clarithromycin resistant Mycobacterium chelonae

A second PubMed and Embase search was completed to review the literature for any cases of CRMC in any site of infection (Table 2). We included studies that contained some clinical description of the patients from whom the isolate was cultured. We identified 9 reports with 12 patients where CRMC was identified. Only one patient, a patient with a scleral buckle infection had CRMC identified on initial culture without a described history of pre-exposure to macrolides [33]. Four more patients are described from a cystic fibrosis NTM registry and were likely to have had prior macrolide exposure, but this was not confirmed [34]. Of the remaining 7 patients, 6 received long-term systemic corticosteroid therapy and developed CRMC infections after initially being treated with a clarithromycin-based regimen (usually monotherapy) [35–40]. The last patient who was not immunosuppressed, developed a CRMC prosthetic hip infection after failing an initial clarithromycin-based regimen [41].

CRMC is a rare entity. Rodriguez *et al.* demonstrated that antimicrobial resistance in *M. chelonae* developed in the presence of subinhibitory concentrations of clarithromycin in 2007 in a laboratory environment [42]. Consequently, most reports on clarithromycin resistant *M. chelonae* have been in the setting of macrolide exposure and/or monotherapy in disseminated infection (Table 2). Macrolide resistance in *M. abscessus* ssp. *abscessus* is a well described entity, and it is usually due to the inducible *erm*(41) gene, which is not present in *M. chelonae* [43]. In contrast, *M. chelonae* clarithromycin resistance is usually mediated by a single point mutation at position 2058 or 2059 of the 23S rRNA gene [36,44]. Unfortunately, genetic sequencing data was not performed on our patient's isolate to confirm the mechanism of resistance.

3. Conclusions

Rapidly growing NTM infections after breast surgery have been reported widely in the literature, often in the context of outbreaks associated with specific centers, surgeons, or contaminated equipment. Several series have specifically been reported in patients returning to the U.S. after pursuing medical tourism in the Dominican Republic [28,30,45,46]. Our case is notable for two distinct reasons: 1) surgical cure of NTM infection occurred without anti-mycobacterial therapy and 2) the demonstration of CRMC in a non-immunocompromised patient with localized disease and no previous macrolide exposure. To our knowledge, this is the first report of surgical cure of a confirmed NTM breast implant infection and the second report of CRMC identified in a patient who did not have previous macrolide exposure (first in breast infection).

Our case further highlights the challenges commonly encountered by clinicians treating NTM infections, including the longer time required for AST for mycobacteria, incl. NTMs, when compared to other routine AST in clinical laboratories, because of the time required for organism growth and the fact that AST is often performed by reference laboratories as a send-out test. Other challenges in treating NTM infections include common side effects of first line antibiotics necessitating construction of an alternative regimen, which is often difficult in the setting of drug resistant organisms with limited available oral options. While newer oral antibiotics such as bedaquiline and omadacycline are potentially promising for the treatment of NTM infections, evidence for use of these agents is sparse and access is often limited due to prohibitive costs. In our patient's case, an unexpected pregnancy enabled observation of the natural course of the disease after thorough debridement and removal of the implants. While surgical debridement and anti-mycobacterial therapy, typically with intravenous antibiotics for the initial phase of treatment, remains the standard of care for the majority of patients with post-surgical NTM infection given the risk of poor outcomes, our case shows that if anti-mycobacterial therapy cannot be provided, there is a chance of cure with surgical debridement alone. However, if this strategy is pursued, extremely close follow-up is warranted to mitigate the risk of a poor outcome that can occur if recurrence of infection is not promptly diagnosed and treated.

Ethical statement

The patient described in this case gave consent to the use of her de-identified information for this report.

Funding

There are no funding sources involved in the authorship of this article.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgement

The authors would like to acknowledge Megan McNichol, MLS, AHIP from Beth Israel Deaconess Medical Center Knowledge Services for assistance with the literature review.

Appendix

PubMed and Embase search strategy for surgical cure of NTM breast implant infection:

("Nontuberculous Mycobacteria"[MeSH Term] OR "Nontuberculous Mycobacteria"[tiab] OR "Mycobacterium Infections, Nontuberculous" [MeSH Term] OR "Mycobacterium Infections" [MeSH Term] OR "Mycobacterium chelonae"[MeSH Term] OR "Mycobacterium chelonae"[tiab]) AND ("breast"[MeSH Term] OR "breast"[tiab] AND "augmentation"[tiab] OR "breast implants"[tiab] OR "breast implants"[MeSH Term] OR "breast implants/adverse effects"[MeSH Term] OR "breast augmentation"[tiab] OR "breast augmentation surgery"[tiab] OR "Mammaplasty"[MeSH Term] OR "Breast/surgery"[MeSH Term]) AND ("Surgery"[tiab] OR "Surgical Wound Infection"[MeSH Term])

PubMed and Embase search strategy for Clarithromycin Resistant Mycobacterium Chelonae infections:

"clarithromycin"[tiab] OR "Clarithromycin"[Mesh] OR "macrolide "[tiab] OR "macrolides"[tiab] OR "Macrolides"[Mesh]) AND ("resistant"[tiab] OR "resistance"[tiab]) AND ("Mycobacterium Chelonae"[tiab] OR "Mycobacterium chelonae"[Mesh]) NOT ("breast implant infections" [tiab] OR"Breast Implants"[Mesh] OR "Breast Implant"[tiab]) AND English[lang]

The patient described in this case gave consent to the use of her pictures and her de-identified information for this report.

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