SKIN

BRIEF ARTICLE

A 14-year-old Girl with Atrophic Plaques on the Arm: Case Report

Hannah R. Riva, MD,[1] Mary E. Cavanagh, MD,[2] Ayezel Munoz Gonzalez, MD[2]

[1] Paul L. Foster School of Medicine, Texas Tech University Health Sciences Center, El Paso, TX, USA
[2] Department of Dermatology, University of Texas Medical Branch, Galveston, TX, USA

ABSTRACT

As the incidence of cutaneous infections due to nontuberculous mycobacteria continues to rise, clinicians should be aware of the various ways in which such infections may present. This case seeks to highlight an unusual clinical presentation of a co-occurring epidermal inclusion cyst and atypical mycobacterial infection. A 14-year-old female presented to dermatology clinic with a three-month history of a cutaneous lesion on the right posterior arm. She reported a sudden onset of burning pain in the area when she first noticed the lesion. There was no known history of insect bites or prior trauma to the area. Examination revealed two adjacent atrophic plaques with surrounding erythema. Histopathology demonstrated dermal scar with suppurative granulomatous inflammation concerning for atypiScal cutaneous infection. Subsequent excisional biopsy demonstrated a ruptured epidermal inclusion cyst. Bacterial and fungal cultures were negative, whereas acid-fast bacilli culture was positive for *Mycobacterium abscessus*. Due to antibiotic resistance among various nontuberculous mycobacteria strains, it is important to acquire culture sensitivities to adjust antibiotic treatment as needed based on sensitivity results.

INTRODUCTION

Atypical mycobacteria, also called environmental mycobacteria or nontuberculous mycobacteria (NTM), are a diverse group of ubiquitous, acid-fast organisms widespread in the environment. In addition to pulmonary disease, lymphadenitis, and disseminated disease, skin and soft tissue infections (SSTIs) are another manifestation of NTM infection. NTM infections are on the rise both in incidence and prevalence worldwide, particularly in developed countries with declining incidence rates of *M. tuberculosis*. Particularly, SSTIs caused by NTM are also increasing in incidence and prevalence worldwide.[1,2] The rise in NTM SSTIs has been initially hypothesized to correspond with an increase in individuals who are immunosuppressed, however, NTM SSTIs are not limited to the immunosuppressed state and have been documented occurring in numerous healthy individuals. Exact incidence of NTM SSTIs has yet to be determined.

CASE REPORT

A 14-year-old female with no significant past medical history presented to our clinic with a three-month history of a cutaneous lesion on the right posterior arm. She first noticed the lesion after developing a sudden onset of burning pain in the area. There was no known

history of insect bites, trauma to the area, or drainage from the lesion. No pertinent travel history or environmental exposures were reported. Prior treatments included topical clindamycin and mupirocin, which were ineffective. Examination revealed two adjacent atrophic plaques with surrounding erythema (**Figure 1A**). On dermoscopy, there were large, looped vessels with background yellow-white globules (**Figure 1B**). Punch biopsy was performed. No treatment was given at the first visit as the lesion was not initially clinically suspected to be infectious. Histopathology demonstrated dermal scar with suppurative granulomatous inflammation. A Verhoeff-Van Gieson (VVG) elastin stain was obtained to confirm clinical and histopathologic evidence of scar, and VVG stain was indeed decreased, consistent with scar formation. Due to the presence of suppurative granulomatous inflammation on hematoxylin and eosin (H&E), which was suggestive of an infectious etiology, infectious stains—Period acid-Schiff (PAS), Gram, Auramine-rhodamine, and Fite—were obtained; these stains resulted negative for microorganisms. Upon follow-up examination four weeks later, clinical and dermoscopic appearance (**Figure 2A** and **Figure 2B**, respectively) was relatively unchanged; tissue culture and excisional biopsy were performed. Histopathology of the excisional biopsy demonstrated dermal epithelial cyst lining with loose lamellated keratin contents and surrounding granulomatous inflammation, consistent with a ruptured epidermal inclusion cyst (**Figure 3**). Period acid-Schiff (PAS), Gram, and Fite stains were again negative. Bacterial and fungal tissue cultures were negative, whereas acid-fast bacilli (AFB) tissue culture was positive for *Mycobacterium abscessus*. Diagnosis was consistent with ruptured epidermal inclusion cyst co-occurring with an atypical mycobacterial (*Mycobacterium abscessus*) infection. In addition to complete excision, the patient was treated with a 3-month course of oral azithromycin. Susceptibility testing demonstrated sensitivity to macrolides and resistance to tetracyclines. The patient was seen for follow-up nine months later and was noted to have complete clinical resolution.

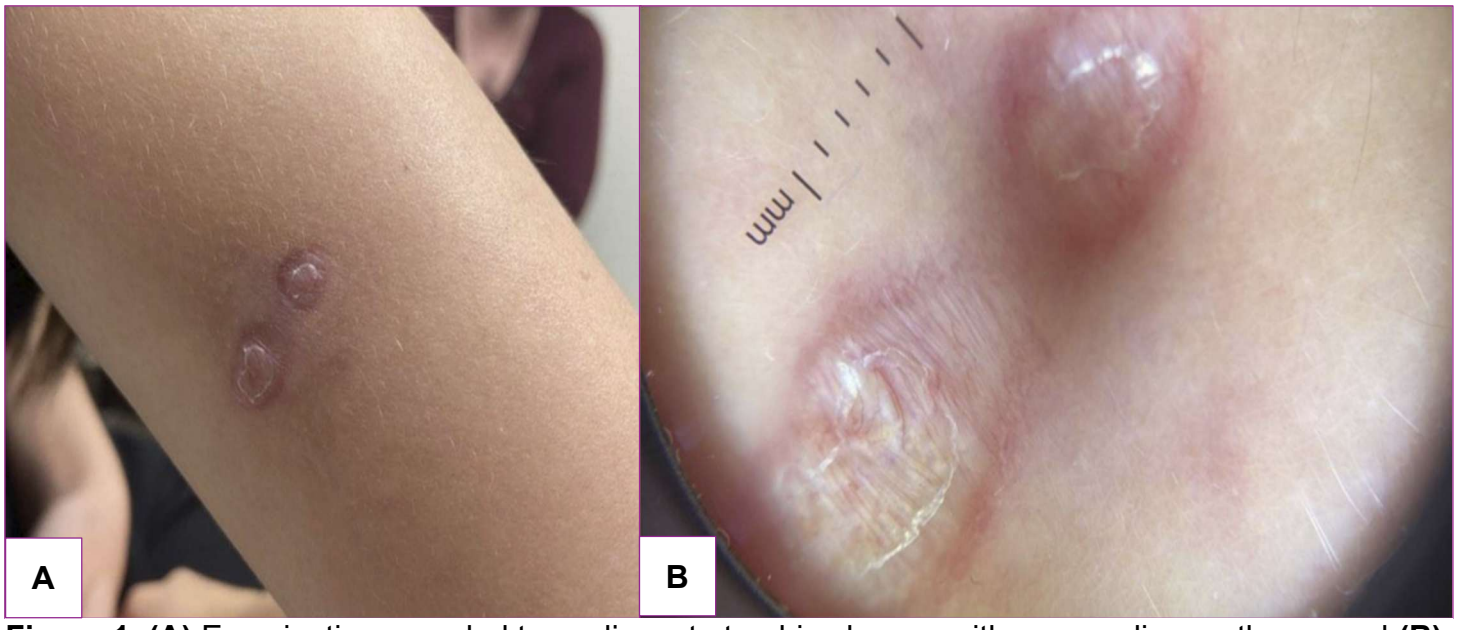

Figure 1. (A) Examination revealed two adjacent atrophic plaques with surrounding erythema and **(B)** On dermoscopy, there were large, looped vessels with background yellow-white globules

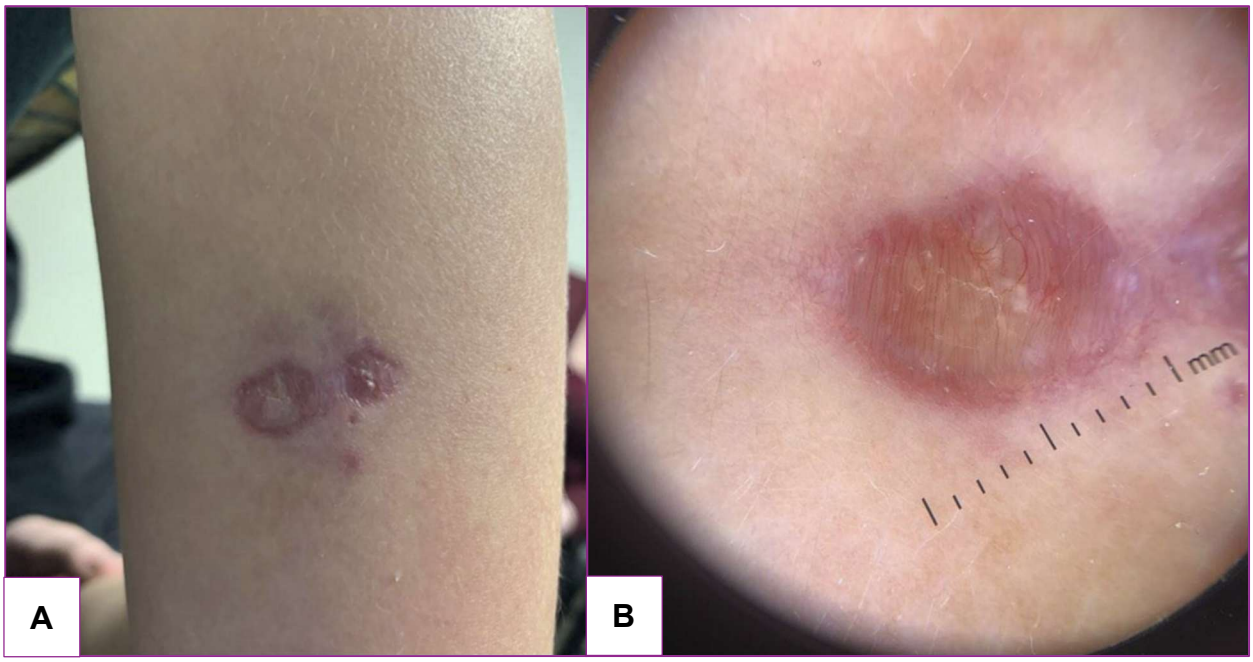

Figure 2. Upon follow-up examination four weeks later, **(A)** clinical and **(B)** dermoscopic appearances were relatively unchanged

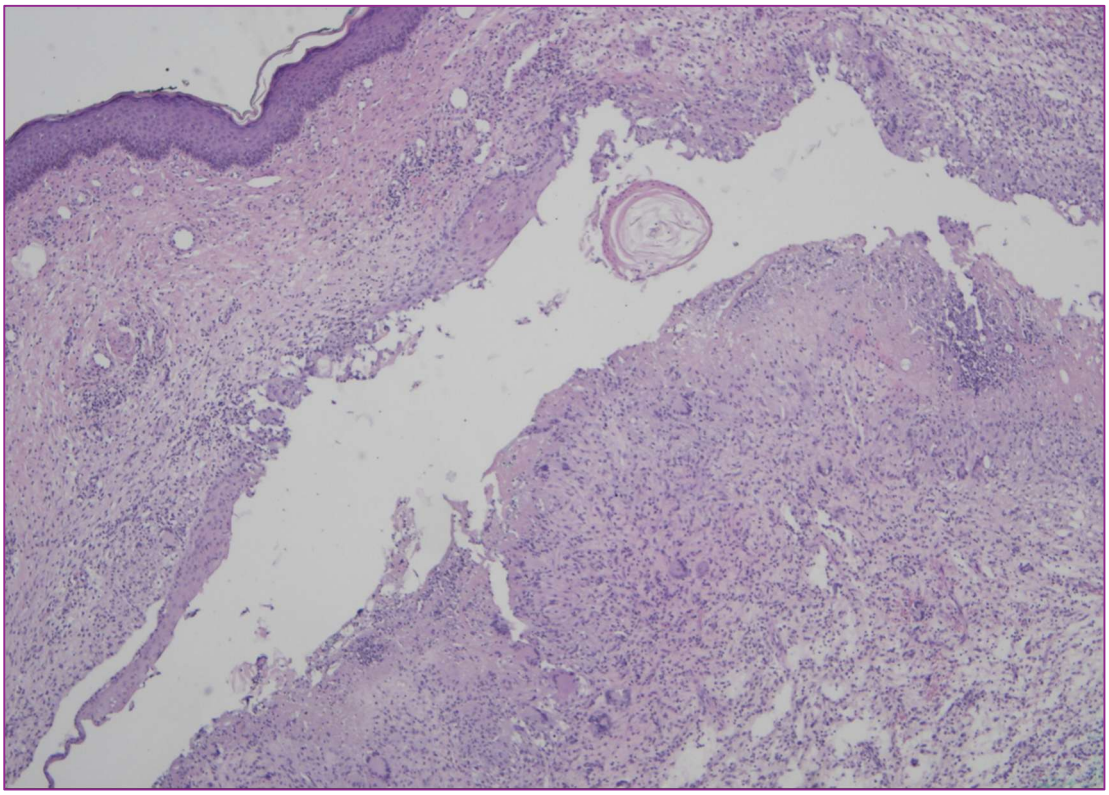

Figure 3. Histopathology of the excisional biopsy (hematoxylin & eosin stain) demonstrated dermal epithelial cyst lining with loose lamellated keratin contents and surrounding granulomatous inflammation, consistent with a ruptured epidermal inclusion cyst

DISCUSSION

Mycobacterium chelonae is an NTM classified as a rapidly growing mycobacterium (RGM). *M. chelonae* is divided into six groups: *Mycobacterium fortuitum group, M.chelonae/M. abscessus* complex, *Mycobacterium smegmatis*, *Mycobacterium mucogenicum* group, *Mycobacterium mageritense/Mycobacterium wolinskyi*, and the pigmented RGM.

M. chelonae and *M. abscessus* were considered the same entity until 1992; they are differentiated by their intergenic sequence.[3] Of note, *Mycobacteroides abscessus* is the new term for *Mycobacterium abscessus*.[4] *M. abscessus* is a rod-shaped acid-fast bacillus that is ubiquitous in the environment, like most NTM species, and has been found in soil, natural and drinking water sources, sewage water, and decaying vegetation; in addition to environmental sources, there are reports of *M. abscessus* being spread by transmission from infected humans.[2,5] In addition to high multi-drug resistance, *M. abscessus* is associated with biofilm formation and resistance to high temperatures and disinfectants.

Cutaneous NTM infections typically develop after direct inoculation of the skin through traumatic injury, surgery, or cosmetic procedures. A recent pediatric case highlights a cutaneous NTM infection arising at the site of an accidental firearm injury on the foot.[6] Another noteworthy case series in the literature includes an outbreak of 12 children who developed NTM cutaneous infection of the hands and/or feet from an improperly maintained wading pool.[7] It was later discovered that pool maintenance did not fully comply with state regulations, and *M. abscessus* was isolated from pool equipment.

Often the exact source of NTM infection may not be elicited by history alone. Presentation of cutaneous NTM can be highly variable and may include cellulitis, abscesses, nodules (with sporotrichoid spread), ulcers, vasculitis, draining sinus tracts, or other non-specific skin lesions.[8,9] A recent case series of cutaneous NTM infections from our institution, the University of Texas Medical Branch, found that the odds of inclusion of NTM in the initial differential diagnosis was 45 times higher if the patient was seen by dermatology compared with other specialties ($p<0.001$), yet the diagnosis was considered still in only 61% of initial dermatology encounters.[10] Chronic cutaneous lesions with a history of trauma, surgical procedures, cosmetic interventions, or environmental exposures such as aquatic environments should raise suspicion for NTM skin infection, however, these may not be endorsed in the clinical history, as in our patient. Cutaneous NTM infections should be considered in the differential diagnosis particularly in chronic cutaneous lesions that do not resolve with antibiotic therapy and when bacterial cultures are negative.[11]

Treatment of cutaneous disease is dependent upon the species, wherein members of the *M. abscessus* complex tend to have susceptibility to certain macrolides, amikacin, imipenem, and cefoxitin.[5] Regarding antibiotic treatment selection, the aforementioned case series also found that all 78 cases of NTM infections showed at least partial susceptibility to clarithromycin, however, some rapidly growing mycobacteria (RGM) species, including *M. abscessus*, had an erythromycin resistance methylase (*erm*) gene that confers macrolide resistance.[10] Thus, due to antibiotic resistance among

various NTM strains, it is important to acquire culture sensitivities to adjust antibiotic treatment as needed based on sensitivity results.

This case seeks to highlight an unusual clinical presentation of a co-occurring epidermal inclusion cyst and a primary atypical mycobacterial infection. As the incidence of cutaneous infections due to NTM continues to rise, clinicians should be aware of the various ways in which such infections may present, especially in the pediatric population where this diagnosis may also be encountered.

Conflict of Interest Disclosures: None

Funding: None

Corresponding Author:
Hannah R. Riva, MD
512-897-4095
Email: Hriva0915@gmail.com

References:

1. Wentworth AB, Drage LA, Wengenack NL, Wilson JW, Lohse CM. Increased incidence of cutaneous nontuberculous mycobacterial infection, 1980 to 2009: a population-based study. *Mayo Clin Proc*. 2013;88(1):38-45.
2. To K, Cao R, Yegiazaryan A, Owens J, Venketaraman V. General overview of nontuberculous mycobacteria opportunistic pathogens: Mycobacterium avium and Mycobacterium abscessus. *J Clin Med*. 2020;9(8).
3. Akram SM, Rathish B, Saleh D. Mycobacterium chelonae infection. *StatPearls*. Treasure Island (FL): StatPearls Publishing Copyright © 2023, StatPearls Publishing LLC.; 2023.
4. Gupta RS, Lo B, Son J. Phylogenomics and comparative genomic studies robustly support division of the genus Mycobacterium into an emended genus Mycobacterium and four novel genera. *Front Microbiol.* 2018;9:67.
5. Boudehen YM, Kremer L. Mycobacterium abscessus. *Trends Microbiol*. 2021;29(10):951-952.
6. Berkhout A, Curtis N, Gwee A, Harris C, Burgner D. Mycobacterium abscessus soft tissue infection: Review of published cases and challenges in treatment. *Pediatr Infect Dis J*. 2020;39(7):e130-e132.
7. Carter KK, Lundgren I, Correll S, et al. First United States outbreak of Mycobacterium abscessus hand and foot disease among children associated with a wading pool. *J Pediatric Infect Dis Soc*. 2019;8(4):291-296.
8. Maruma F, Carter R, Moosa F, Makuru H, Asmal T. Acute symmetric peripheral gangrene following a long-standing Mycobacterium malmoense infection: A very rare kinship. *JAAD Case Rep.* Vol 6. United States 2020.
9. Weinberg M, Bag-Ozbek A, Chen D, Yao Q. Small vessel vasculitis secondary to Mycobacterium chelonae. *Rheumatol Int*. 2021;41(9):1691-1697.
10. Philips RC, Hoyer PE, White SM, et al. Cutaneous nontuberculous mycobacteria infections: A retrospective case series of 78 patients from the Texas Gulf Coast region. *J Am Acad Dermatol*. 2019;81(3):730-739.
11. Lamb RC, Dawn G. Cutaneous non-tuberculous mycobacterial infections. *Int J Dermatol*. 2014;53(10):1197-1204. doi:10.1111/ijd.12528