**CASE REPORT**

**Cutaneous atypical mycobacterial infection with *Mycobacterium fortuitum* arising after endovenous radiofrequency ablation**

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**Key words:** atypical mycobacteria; cutaneous infection; infection; *Mycobacterium fortuitum*; rapidly growing mycobacteria; venous ablation.

**INTRODUCTION**

Rapidly growing mycobacteria (RGM) are defined as nontuberculous mycobacteria that show growth within 7 days. RGM are widely distributed in the environment, and several studies have identified RGM in tap water. The ability of RGM to form biofilm serves to increase virulence and augment their resistance to disinfection. In immunocompetent patients, cutaneous infection occurs at sites of piercing trauma. We present an unusual clinical variant of a cutaneous RGM infection caused by *Mycobacterium fortuitum* in an elderly man who presented with cutaneous infection at the access location of his cosmetic endovascular radiofrequency venous ablation.

**CASE REPORT**

A 76-year-old man underwent cosmetic endovenous radiofrequency ablation to varicose veins of the left distal thigh and proximal lower leg. Two weeks after the procedure, a papule appeared on the left medial thigh. His primary care provider prescribed a trial of topical clobetasol; however, over the next 3 weeks, the lesion persisted with the appearance of 2 additional lesions. At that point, the patient was referred to the dermatology department, where he presented with cutaneous infection at the access location of his cosmetic endovascular radiofrequency venous ablation.

**Abbreviation used:**

RGM: rapidly growing mycobacteria

in a sporotrichoid pattern. Shave biopsy of the papule on the left medial calf found a necrotic base with seropurulent discharge; the specimen was sent for aerobic, mycobacterial, and fungal cultures and for histopathologic examination. He was prescribed a 10-day course of doxycycline. At the 2-week follow-up, the lesions showed no change, and the patient was started on oral clarithromycin because of concern for atypical mycobacteriosis. At the 3-week follow-up, acid-fast bacteria culture with DNA microarray confirmed the presence of *M. fortuitum*.

Microscopic examination of the tissue found dermal necrosis and acute inflammation with organisms visualized on Fite stain (Fig 2). Fungal culture showed no growth at 1 month. At the 5-week follow-up, the lesions showed mild clinical improvement. At this time, the sensitivity results became available and showed resistance to clarithromycin and sensitivity to doxycycline, amikacin, ciprofloxacin, and minocycline. Although moderate clinical improvement occurred while the patient was taking clarithromycin, the therapeutic regimen was bolstered with the addition of minocycline to clarithromycin to address concerns for inducible macrolide resistance using macrolide monotherapy. This regimen was continued for 6 additional weeks.

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Marked clinical improvement was observed the following week, and the lesions resolved after 6 weeks of the combined antibiotic regimen. There were no recurrences at follow-up visits for more than 1 year (Fig 1, B).

**DISCUSSION**

Although about 70 RGM species are recognized, approximately 90% of human infections are caused by *Mycobacterium abscessus*, *Mycobacterium chelonae*, and *M. fortuitum*.1 These 3 species are also commonly isolated in skin and soft tissue infections. Infections with *M. fortuitum* and *M. abscessus* do not show predilection for host immune status; however, *M. chelonae* is more likely to occur in immunosuppressed individuals, commonly patients on systemic corticosteroids.2

The clinical presentation of cutaneous RGM infection varies, with immunocompetent patients typically presenting with localized infection, whereas immunosuppressed patients may present with widespread cutaneous lesions or signs of systemic infection.3 A presumptive clinical diagnosis of an atypical mycobacterial infection is confirmed with culture and subsequent molecular identification. Sensitivity studies guide treatment of this infection.

Although macrolide activity studies in *M. fortuitum* find that 80% of isolates are susceptible to clarithromycin (minimum inhibitory concentration ≤ 4 μg/mL),4 there is concern for inducible resistance to macrolide therapy with the detection of a novel gene *erm(39)* in this species.5 This gene encodes a methylase that adds 1 or 2 methyl groups to the 23S rRNA, preventing macrolides from binding to the bacterial ribosome. Clinicians should proceed with caution when treating infections caused by *M. fortuitum* and *M. abscessus* with macrolide monotherapy, as inducible resistance genes have been discovered in these 2 species; however, *M. chelonae* isolates do not exhibit the inducible resistance gene.6

Health care–associated atypical mycobacterial infections are nearly always caused by RGM and are well studied in the literature.2 Outbreaks of RGM have occurred after surgeries and cosmetic procedures, but sporadic cases are also observed.7 Tap water is a known source of atypical mycobacteria and is a common cause of postprocedural infections when surgical instruments are incorrectly cleaned with municipal water or when sterile or clean fields are contaminated with tap water.2 Certain strains of acid-fast bacteria are known to be resistant to standard antiseptic preparations, and resistance patterns can vary depending on the strain.8,9 Thus, iatrogenic infection can theoretically occur despite the use of appropriate aseptic technique. Reports of atypical mycobacterial contamination within antiseptic solutions have been described.10

The patient presented here received ablative therapy to the great saphenous vein at the level of the knee for treatment of varicosities on the left
medial leg. Although aseptic technique is essential to reduce the likelihood of iatrogenic infection, additional possible causes include inadequate disinfection of procedure devices and contamination from tap water sources. We report a rare clinical presentation of a cutaneous postsurgical atypical mycobacterial infection in the setting of an aseptic procedure. Further studies of locally isolated wild-type RGM species are warranted given the discrepancies in resistance that have been described.

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