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Review Article

Cutaneous non-tuberculous mycobacterial infections: An update

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ABSTRACT

Non-tuberculous mycobacteria (NTM) are increasingly recognized as causes of skin and soft-tissue infections. They include rapid-growing and slow-growing species. Hospital outbreaks related to contaminated water and in association with surgical and cosmetic procedures have been described. Infections are also associated with immunosuppression. NTM infections have a wide spectrum of clinical manifestations, though *Mycobacterium marinum* and *Mycobacterium ulcerans* manifest characteristic lesions – swimming pool granuloma and Buruli ulcer, respectively. NTM infection should be suspected when the skin infection (especially those following trauma or invasive procedure or in a patient with immunosuppression) does not respond to antibiotics. NTM are acid fast, but will be negative on cartridge based nucleic acid amplification tests for *Mycobacterium tuberculosis*. Diagnosis is confirmed by polymerase chain reaction test which is the gold standard. NTM show variable susceptibility to antimicrobials and no clear treatment guidelines are available. Surgical treatment may also be needed in some cases.

Keywords: Non-tuberculous mycobacteria, Atypical mycobacteria, Mycobacteria, Skin

INTRODUCTION

The term "Non-tuberculous mycobacteria" (NTM) refers to the group of all mycobacterial species other than *Mycobacterium tuberculosis (M. tuberculosis)* complex and *M. leprae*.[1] NTM are also referred to as atypical mycobacteria, environmental mycobacteria or mycobacteria other than tuberculosis. They are free-living, slender, non-motile, and acid-fast bacilli, that are ubiquitous in the environment, and have been isolated from water, soil, food products, plants, and domestic and wild animals including fish.[2,3] NTM are facultative, intracellular agents in humans and may be present as colonizers or as pathogens.

Skin and soft tissue infections (SSTI) caused by NTM are difficult to diagnose due to their wide spectrum of clinical presentations, as well as non-specific histopathology findings. Tissue culture and polymerase chain reaction (PCR) assays may be needed for diagnosis. Treatment is challenging and requires multiple antibiotic combinations and surgical intervention depending on the causative organism.[3]

NTM are traditionally classified based on the rate of growth *in vitro* as rapid-growing mycobacteria (RGM) and slow-growing mycobacteria (SGM).[4] They are further classified based on pigment production following exposure to light.[5] RGM grow rapidly in culture and hence can be isolated within 7 days.[4,5]

For some RGM species including *M. fortuitum, M. abscessus,* and *M. chelonae,* SSTIs are the most common presentation. Of the SGM species, *Mycobacterium avium* complex (MAC: *M. avium*, *M. intracellulare, M. chimaera), M. ulcerans* (Buruli ulcer), *M. marinum* (fish-tank granuloma)*, M. haemophilum*, and a few other rarer species have been implicated in SSTI and are being increasingly reported in recent years.[2] While these are the most common agents, virtually any species of NTM can cause cutaneous disease. NTM infection should be considered in all patients with treatment resistant SSTI.[2]

EPIDEMIOLOGY AND MODES OF TRANSMISSION

NTM are generally acquired through environmental exposure and there had been no reports of human-to-human transmission, until a case of potential *M. abscessus* transmission between cystic fibrosis patients was reported in 2013.[6]

Skin infection by NTM typically occurs following minor trauma, especially accidental inoculation such as by needles, wood splints, or fish spines. They are also reported to occur by accidental contamination of surgical or open wounds – and have been associated with nosocomial outbreaks following surgical or cosmetic procedures. Contaminated water has been identified as the source in many of these outbreaks.

Mycobacterial species are frequently found in hospital environment because of their biofilm forming capacity, making them highly resistant to standard decontamination techniques.[7]

Being relatively resistant to standard disinfectants such as chlorhexidine, glutaraldehyde, alcohol, and formaldehyde, RGM species are more commonly associated with nosocomial outbreaks.[3,8] Rarely, infection may spread hematogenously in immunocompromised hosts.[7]

The incidence of NTM infection has increased dramatically over the past several years. The incidence of cutaneous NTM infections in 2000–2009 was nearly threefold higher than in 1980–1999. This is due to the increase in procedures such as tattooing, mesotherapy, liposuction, dermal fillers, and body piercing.[9] Clustered cases have been reported from non-hospital settings such as tattoo parlors, nail salons, fish markets, and acupuncture centers.[7] Improved detection by rapid and reliable molecular methods has also contributed to the increased recognition and identification of NTM.

RISK FACTORS

Immunocompromised status due to human immunodeficiency virus infection, organ transplantation, and biological therapy using tumor necrosis factor-α (TNF-α) inhibitors predisposes to disseminated NTM infection. Individuals with Mendelian susceptibility to mycobacterial disease have selective susceptibility to clinical disease caused by environmental mycobacteria.[10]

CUTANEOUS MANIFESTATIONS

SSTI by NTM have highly variable manifestations, which can contribute to a delay in diagnosis. Clinically lesions may appear as papules, plaques, nodules, abscesses, sinuses, ulcers, panniculitis, folliculitis, and cellulitis. Infection may present in a sporotrichoid pattern, tracking along lymphatics from the site of inoculation, particularly in *M. marinum* infection. *M. ulcerans* and *M. marinum* produce characteristic clinical pictures (see below).[3,7,11] Reactive manifestations due to disseminated NTM have been described and include Sweet's syndrome, generalized pustulosis, erythema nodosum, and pustular psoriasis.[3]

Rapidly growing NTM species

The three primary SSTI-causing RGM species are *M. abscessus*, *M. chelonae, and M. fortuitum.* Contaminated water is a frequent source of infection. They are increasingly found in hospital settings following trauma, surgery, and cosmetic procedures. The clinical presentation is varied.

M. abscessus

Among the RGM species, *M. abscessus* is the major cause of skin infections. It was first described by Moore and Frerichs in 1953 in a woman with chronic osteoarthritis of knee, who developed a gluteal abscess.[12] *M. abscessus* is the most pathogenic and clinically challenging of RGM species. It frequently demonstrates a higher degree of antimicrobial resistance. The single most important factor that determines the course and prognosis of *M. abscessus* infection is the underlying immune status of the host.[13] Disseminated cutaneous infections have been reported in patients on immunosuppressive therapy.[14]

Multiple outbreaks following medical, surgical, and cosmetic procedures and acupuncture have been reported.[1] Forty-five surgical site infections due to *M. abscessus* among pediatric patients were reported in 1998 from New Delhi, India, in a single hospital.[15] *M. abscessus* was isolated from the tap water in the operating rooms, which was used for rinsing instruments. In 2004, the Centers for Disease Control and Prevention reported an outbreak of 24 cases of *M. abscessus* infection among patients who had undergone a variety of cosmetic procedures in The Dominican Republic.[16]

Cutaneous manifestations are non-specific. Initial presentation may be an abscess at the site of inoculation. Nodules, abscesses, and non-healing ulcers may be seen [Figure 1]. Sporotrichoid distribution of lesions has also been reported.[17] Disseminated disease presents with systemic symptoms and may be associated with red to violaceous, subcutaneous nodules, and lymphadenopathy.[18,19]

M. chelonae

M. chelonae can infect surgical and traumatic wounds, and cause injection site abscess, especially in diabetic patients on insulin. Reported infections with M. chelonae are on the rise.[20] Surgical site infections as well as wound infections are associated with tap water contamination – such an outbreak was reported from India in association with laparoscopic surgery [Figure 2].[21] Infection is also reported following acupuncture, tattooing, mesotherapy, and cosmetic procedures.

There are reports of non-healing leg ulcer after penetrating injury.[22] Fatal and disseminated chelonae infections have been rarely reported in immunosuppressed patients and following administration of biologicals such as adalimumab.[2,23]

Cutaneous manifestations include localized cellulitis, nodules, sinuses, abscesses, and ulcers.[1]

They are resistant to conventional anti-tuberculosis treatment (ATT), with varied susceptibility to other antimicrobials.

M. fortuitum

M. fortuitum was first isolated by Da Costa Cruz in 1938, from a patient with an injection site abscess.[24,25] In contrast to *M. chelonae* and *M. abscessus* infections, which commonly occur in immunocompromised hosts, infection due to *M. fortuitum* tends to occur mostly in otherwise healthy individuals of younger age group, and typically present with single lesion at the site of trauma.[1,26] In 2002, Winthrop *et al.* reported a community outbreak of *M. fortuitum* furunculosis following footbath at a nail salon in California. Shaving the legs before the procedure was identified as a possible risk factor.[27] Thus, mycobacterial infections should be considered and enquiry should be made regarding recent pedicures underwent by the patient when lower extremity, treatment-resistant furunculosis is encountered.[26]

Lesions can vary from painful nodules, abscesses, ulcers, draining sinuses, and tracts to cellulitis. *M. fortuitum* is a rare cause of dialysis catheter line infection.[28]

M. fortuitum is more susceptible to antibiotics, adjuvant surgical intervention is recommended as and when necessary.

Slow growing NTM species

M. marinum (M. balnei)

M. marinum was first isolated by Aronso in 1926; later in 1951, Norden and Linel identified it as the pathogen in a patient, who developed a granulomatous lesion after visiting a contaminated swimming pool.[29,30] It is an aerobic organism which grows best at a temperature of 30–32°C, and poorly at 37°C.

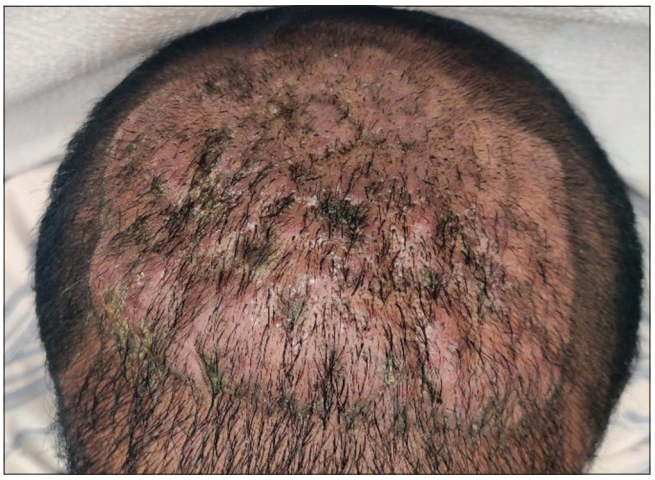


Figure 1: *Mycobacterium abscessus* infection on the scalp of a patient following hair transplantation presenting as multiple nodular lesions and abscesses – picture courtesy Dr. Anisha K Janardhanan, Consultant Dermatologist, Baby Memorial Hospital, Kozhikode.

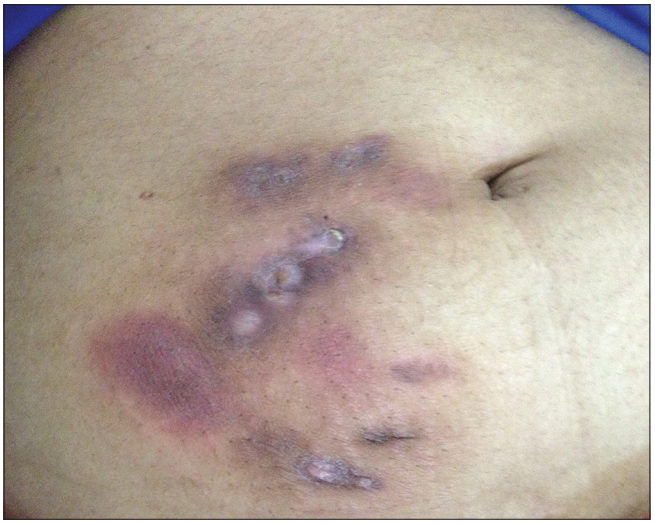


Figure 2: *Mycobacterium chelonae* infection presenting as nodules with discharging sinuses at the laproscopic port site on the abdomen – picture courtesy Dr Soumya Jagadeesan, Associate Professor, Dept of Dermatology, Amrita Institute of Medical sciences, Kochi.

M. marinum infection may be an occupational hazard, as in pet shop workers, or in fish fanciers with home aquariums as it is found both in fresh and salt water. It causes a characteristic skin infection termed *fish-tank or swimming pool granuloma*, which presents as solitary or multiple violaceous papulonodular lesions at the site of inoculation after 2–3 weeks, usually in the extremities. This may spread proximally in a sporotrichoid fashion due to lymphocutaneous spread or evolve to nodular, psoriasiform, or verrucous plaques which may later ulcerate [Figure 3]. Lymphadenopathy is rarely seen. Specific membrane lipids, called phenolic glycolipids, have been demonstrated to

recruit macrophages to the site of infection, which facilitates further spread.[7]

Spontaneous resolution can occur after many months, while deeper infections can lead to tenosynovitis ("fish tank finger"), bursitis, arthritis, and osteitis.[31,32]

There are reports of outbreak of infection due to NTM, probably *M. marinum* in Pacific islanders (locally known as spam disease), which manifest as verrucous and keloid plaques.[33,34]

M. marinum infection associated with TNF-α inhibitors was first reported in 1994, in a patient, who developed septic arthritis following etanercept therapy. Similar infections have been reported following infliximab and adalimumab.[35-39]

M. ulcerans

Infection with this species was first described in 1948 by MacCallum *et al.* from Australia in patients presenting with solitary ulcerative lesions in extremities, and the *Mycobacterium* was named "Bairnsdale bacillus," after the region, where most of the patients lived.[40] Later, it was renamed as *M. ulcerans*. Skin ulcers caused by *M. ulcerans* were reported from Buruli in Uganda in 1961, a region near the Nile River, and following several reports from the region, the disease was subsequently named Buruli ulcer (BU).[40]

M. ulcerans is the third most common cause of mycobacterial infection after tuberculosis and leprosy worldwide and BU is one of the skin-related neglected tropical diseases according to the WHO.[1]

Children and young adults are most commonly affected. Onset is typically as a subcutaneous nodule, which subsequently enlarges and ulcerates. It may later bore through the soft tissue, right up to the bone. Extremities are commonly affected, although head, neck, trunk, and genital region can be involved. Mycolactone, a highly diffusible and cytotoxic lipid has been identified as the pathogenic factor, and explains the distinct clinical presentation. Three major biological properties of mycolactone are cytotoxicity, immunosuppression, and analgesia, which correspond well to the characteristic features of the disease: extensive deep ulceration with thick yellowish necrotic tissue, undermining with limited inflammatory response and minimal or absent pain.[40-42]

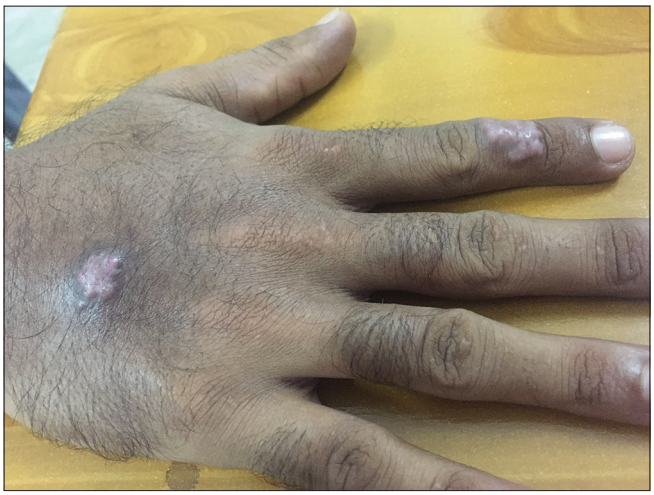


Figure 3: *Mycobacterium marinum* infection presenting as nodules on the hand – picture courtesy Dr Soumya Jagadeesan, Associate Professor, Department of Dermatology, Amrita Institute of Medical sciences, Kochi.

Antibiotic therapy is usually effective; however, patients with severe illness or delayed initiation of treatment may have permanent deformities. Without treatment, the disease resolves in some patients, while in others it ends in contractures that cause disfigurement, long-term disability, and social stigmatization, known as "bankruptcy wound".[41] Early detection and treatment is the only measure to prevent deleterious outcomes.[7,42]

MAC

This includes *M. avium, M. intracellulare, M. chimaera,* and other species. Primary cutaneous infections are rare. Skin lesions in disseminated disease have been described in immunocompromised patients, especially advanced AIDS. With the onset of the AIDS epidemic, MAC became more recognized as an opportunistic pathogen. Clinical manifestations include scaly papules, granulomatous plaques, subcutaneous nodules, pustules, verrucous ulcers, abscesses, and discharging sinuses.[3,43-45] A case of papulonecrotic tuberculid like presentation secondary to disseminated MAC infection in AIDS has been reported.[46]

Emerging pathogen: M. chimaera

M. chimaera was described by Tortoli *et al.* in 2004 as a colonizer of respiratory tract, in specimens from patients with chronic obstructive pulmonary disease and other lung diseases.[47] It is grouped under MAC. Since it shares features with *M. avium*, *M. intracellulare* and *M. scrofulaceum,* it was named *M. chimaera* – after chimera, a Greek mythological character composed of the parts of more than one animal.[47]

Clinical infections have a long and variable incubation period of up to 6 years. Outbreaks have been reported after open heart surgery. Cutaneous infection with *M. chimaera* is extremely rare. A case of cutaneous infection with *M. chimaera* was reported by Moutsoglou *et al.* in a patient with a non-healing leg ulcer.[48] George *et al.* reported a case of primary cutaneous infection with *M. chimaera* in a patient with diabetes mellitus that manifested with ulcerating nodules on the face [Figure 4].[49]

M. haemophilum

M. haemophilum is an aerobic fastidious organism, similar to *M. marinum* and *M. ulcerans*, and shows a predilection for the extremities, particularly over the joints. The name originates from the requirement of iron or hemin supplementation for growth.[50]

Infection generally occurs in immunocompromised hosts and may include erythematous to violaceous papules, plaques, or nodules which may progress to abscesses or deep-seated ulcers. It has certain similarities with *M. leprae*, including the presence of large quantities of docosanoic acid and analogous phenolic glycolipid. Immune reconstitution events similar to lepra reactions are described after initiation of anti-mycobacterial therapy.[44]

M. kansasii

M. kansasii is closely related antigenically to *M. tuberculosis*. Cutaneous infections may present as nodules, pustules, verrucous plaques, ulcers, and abscesses. The lesions may be arranged in a sporotrichoid pattern.[44] Similar to *M tuberculosis*, *M.kansasii* is present in nasopharyngeal secretions and can lead to periorificial cutaneous infection.

MICROBIOLOGICAL DIAGNOSIS

Tissue biopsy is the most sensitive means of obtaining a specimen.[3,44] Drainage material may also yield the organism. Specimens have to be initially analyzed with acid fast stain (e.g., Ziehl-Neelsen stain), cartridge-based nucleic acid amplification test (CB-NAAT - e.g., GeneXpert®) and culture. Culture is currently performed in liquid medium, in mycobacterial growth indicator tubes (MGIT – e.g., BACTEC®) which offer faster and more reliable detection compared to solid medium (Lowenstein-Jensen medium).[3,44]

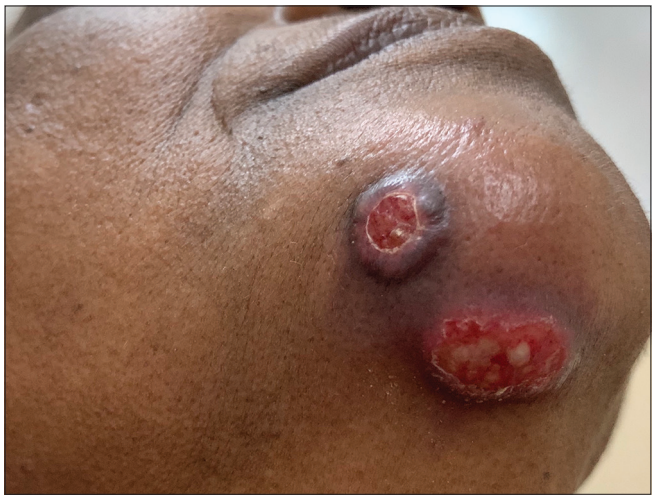
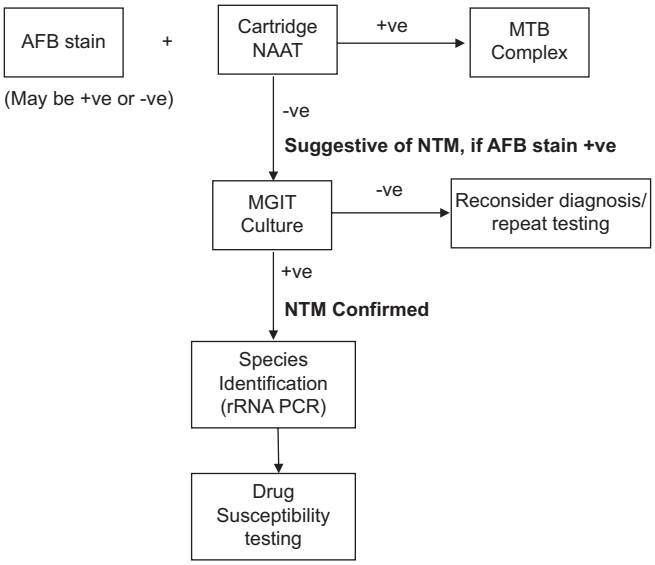


Figure 4: *Mycobacterium chimaera* infection presenting as ulcerating nodules on the face.

All NTM are acid fast, hence will stain positively; however, the sensitivity is low. CB-NAAT is specific for *M. tuberculosis complex,* and hence will be negative for NTM. This combination of positive acid-fast stain and negative CB-NAAT indicates the presence of NTM species.[3,44]

RGM species grow in culture within 7–10 days, while SGM species generally take more than 2 weeks. Once culture is positive, species identification is carried out on the growth using biochemical tests or PCR. Biochemical tests are hazardous and hence are not routinely done at present. 16S ribosomal ribonucleic acid (rRNA) gene analysis with PCR gives reliable identification of all known NTM species and is the standard. Some other targets for PCR are the *rpoB* gene (which has higher discriminatory power for RGM species) and *hsp65* gene.[51] Amplification of the target deoxyribonucleic acid followed by denaturing high-performance liquid chromatography is another promising approach. Matrix-assisted laser desorption ionization time-of-flight mass spectrometry is a novel non-PCR method, where different species are identified by their unique spectral fingerprint.[3,44,51]

The final step would be testing for drug susceptibility, which is recommended especially for RGM species, which are tested for susceptibility to macrolides, quinolones, tetracyclines, and imipenem using microbroth dilution testing [Flow Chart 1].[3,44]



Flow Chart 1: Microbiological diagnosis of non-tuberculous mycobacteria.
AFB: Acid fast bacillus, NAAT: Nucleic acid amplification test, MTB: *Mycobacterium tuberculosis*, NTM: Non-tuberculous mycobacteria, MGIT: Mycobacterial growth indicator tube, rRNA: ribosomal ribonucleic acid, PCR: Polymerase chain reaction.

Table 1: Non-tuberculous mycobacterial infection – treatment.

Organism	Preferred antimicrobials	Comments
RGM Species		Need susceptibility testing. 4–6 months therapy with at least two agents with *in vitro* activity.
M. fortuitum	Macrolides, quinolones, doxycycline, minocycline, amikacin, sulfonamides, cefoxitin, and imipenem	
M. chelonae	Tobramycin, clarithromycin, linezolid, imipenem, amikacin, clofazimine, doxycycline, and ciprofloxacin	Tobramycin more active than amikacin. Clarithromycin is the preferred second agent
M. abscessus	Macrolide and a parenteral agent such as amikacin, cefoxitin, imipenem or tigecycline, or combination of parenteral agents	Surgical resection often required
SGM Species		Delamanid and bedaquiline, tedizolid and avibactam are newer agents for SGM species
MAC	Macrolides: clarithromycin/azithromycin always in combination with ethambutol and a third agent – rifamycin (rifampin/rifabutin) or aminoglycoside (streptomycin/amikacin)	6–12 months treatment with at least 3 drugs recommended Excisional surgery or debridement often needed
M. haemophilum	Amikacin, clarithromycin, ciprofloxacin, rifampin, and rifabutin. Doxycycline and sulfonamides have shown variable susceptibility	All isolates resistant to ethambutol. A minimum of 6 months treatment is required.
M. marinum	Susceptible to rifampin, rifabutin, ethambutol, clarithromycin, sulfonamides, and trimethoprim-sulfamethoxazole; intermediately susceptible to streptomycin, doxycycline and minocycline.	Resistant to isoniazid and pyrazinamide. Usually two active agents given for 1–2 months after resolution of symptoms – 3–4 months in total.
M. ulcerans	Combination of rifampicin and clarithromycin is the recommended treatment.	Rifampin 10 mg/kg/day with clarithromycin 7.5 mg/kg twice daily for 8 weeks. Telacebec is a new anti-tuberculosis drug with potent activity aganist *M. ulcerans*. Surgical debridement combined with skin grafting is recommended.

RGM: Rapid-growing mycobacteria, *M. fortuitum: Mycobacterium fortuitum*, *M. chelonae: Mycobacterium chelonae*, *M. abscessus: Mycobacterium abscessus*, SGM: slow-growing mycobacteria, MAC: *Mycobacterium avium* complex, *M. haemophilum: Mycobacterium haemophilum*, *M. marinum: Mycobacterium marinum*, *M. ulcerans: Mycobacterium ulcerans*

HISTOPATHOLOGY

The histopathological features vary from an acute suppurative process with neutrophilic infiltration to typical granulomatous picture and macrophage infiltration. However, these are not specific to any species.[3] Combination of poorly formed granulomas and chronic suppuration should raise suspicion of NTM infection. Suppurative granuloma forming a central neutrophilic abscess has been described as a characteristic finding by Bartralot *et al*.[52] RGM species are more likely to cause suppurative inflammation and micro abscesses rather than granulomata. *M. ulcerans* infection is characterized by extensive necrosis and calcification, minimal inflammatory response, and occasional granulomas with large clusters of AFB.[53] *M. marinum* shows prominent epidermal changes such as acanthosis, pseudoepitheliomatous hyperplasia, and exocytosis.[54]

TREATMENT

Recommended treatment for the various NTM species is given in Table 1.[2,7,41,55-57]

In general, antibiotic susceptibility should be performed in all RGM species because of unpredictable resistance patterns.[3] Inducible macrolide resistance encoded by erm(41)/erm(39) gene is of particular concern.[2,7]

Newer agents

Delamanid and bedaquiline, two new anti-tuberculosis drugs, have activity against NTM, including MAC, *M. abscessus,* and *M. Ulcerans.* Tedizolid is emerging as a more tolerable alternative to linezolid. There are isolated reports of use of omadacycline in the treatment of cutaneous infections due to NTM.[55] Avibactam, a novel beta-lactamase inhibitor can restore susceptibility to beta-lactams in resistant *M. abscessus* strains.[7]

CONCLUSION

Skin infections caused by NTM are challenging in many ways, from non-specific presentations, difficulty in microbiological diagnosis, lack of well-defined treatment guidelines, and varying patterns of antimicrobial resistance. As a group, the incidence of NTM infections is on the rise; hence, it is important for the clinicians to have an in-depth understanding of the condition.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

Dr. Mamatha George is on the editorial Board of the journal.

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