About 20 species of rapidly growing mycobacteria species that are capable of infecting human beings and causing bloodstream infections have been identified. Many more of these species are being discovered worldwide, especially in resource-poor settings. These microorganisms have been known to cause outbreaks and pseudo-outbreaks. Although rapidly growing mycobacteria are not highly virulent or life threatening, they have a high predisposition to create biofilms and to colonise and infect intravascular catheters. Early detection and identification of specific species can help to estimate predictable antimicrobial susceptibility patterns. However, because susceptibility data originate from developed countries, studies in resource-poor settings urgently need to be done. The best outcome of cure without recurrence depends on a combination of at least 4 weeks of treatment with two or more active antimicrobial agents, plus removal of the intravascular catheter. We review and discuss the epidemiology, pathogenesis, diagnosis, management, and outcomes of rapidly growing mycobacterial bloodstream infections.

**Introduction**

Non-tuberculous mycobacteria are an important cause of human diseases and infections. Runyon classified these organisms as rapidly growing mycobacteria if they can produce mature colonies on agar plates within 7 days, whereas slowly growing mycobacteria need several more days to grow. Although a national disease surveillance system, such as that which exists for *Mycobacterium tuberculosis* in developed countries, has not been put into practice, infections caused by rapidly growing mycobacteria have been increasingly reported during the past few years. These rapidly growing organisms have been associated with a wide range of clinical syndromes in both immunocompetent and immunocompromised hosts, ranging from mild diseases, such as skin and soft tissue infections, to more severe disorders, including osteomyelitis, and lymph node, respiratory tract, bloodstream, and disseminated infections.

The ability of rapidly growing mycobacteria to form a protective biofilm layer has important implications for their successful survival in the environment. This ability is also a notable contributory factor in the pathogenesis of catheter-related bloodstream infections, which are the most common form of rapidly growing mycobacterial health-care-associated diseases. Differences in species’ patterns of susceptibility and resistance to first-line antituberculous drugs create challenges in the treatment approach to these organisms. Nonetheless, with knowledge of the most frequently implicated rapidly growing mycobacteria bloodstream pathogens, some common susceptibility patterns can be used to guide appropriate empirical treatment choices during the wait for full identification and susceptibility results. We review and discuss the epidemiology, pathogenesis, diagnosis, management, and outcomes of rapidly growing mycobacterial bloodstream infections.

**Microbiology and epidemiology**

More than 50 species of rapidly growing mycobacteria have been identified, more than a third of which are opportunistic human pathogens. The most frequently encountered rapidly growing mycobacteria that cause human infections are *Mycobacterium abscessus*, *Mycobacterium chelonae*, *Mycobacterium fortuitum* (complex), *Mycobacterium mucogenicum*, and *Mycobacterium neoaurum*. These organisms are ubiquitous in nature and can be found in soil, dust, rocks, bioaerosols, and water. Rapidly growing mycobacteria have been detected in harsh environmental conditions, such as low pH, extreme temperatures, and low nutrient concentrations. These microorganisms are usually resistant to standard disinfectants, such as chlorine, organomercurials, and alkaline glutaraldehydes. Some rapidly growing mycobacteria species can grow at temperatures well above or below the physiological range—e.g., *Mycobacterium thermoresistibile* grows optimally at 42–52°C—whereas others, such as *M. abscessus*, *M. fortuitum*, and *M. mucogenicum*, can do so at much lower temperatures. The clinical isolation of rapidly growing mycobacteria usually shows seasonal peaks in the summer and autumn, after warm temperatures and abundant rainfall. As environmental organisms, rapidly growing myobacteria can survive these rigorous conditions, mainly by creation of a biofilm. This protective layer has frequently been found in aquatic habitats such as water distribution systems and tap water. In these scenarios, when the rapidly growing mycobacterium has been incorporated into the biofilm, the microorganism can dislodge and disperse itself throughout the environment. The ability of these organisms to live in water pipes and tolerate extreme environments makes them an ideal candidate for nosocomial propagation.

In both developed and developing countries, as medical conditions that compromise the immune system become more widespread, the prevalence of non-tuberculous mycobacterial diseases is expected to increase. Unavoidable health consequences of ongoing and unending natural and human disasters, including social unrest, ethnic conflict, war, earthquake, and floods, seem to be worsening the situation substantially. Improvement in isolation and identification techniques in tertiary care resource-rich settings has increased awareness of rapidly growing mycobacterial infections. Furthermore, as the
costs of phenotypic and molecular diagnostic technology continue to decrease, and the number and quality of national and regional reference laboratories increase, the incidence of rapidly growing mycobacterial diseases will continue to rise. Hence, unsurprisingly, several new rapidly growing mycobacteria species are being identified worldwide, especially in resource-poor countries.  

Some of these microorganisms are usually found in a specific geographical location and are characterised by a particular antimicrobial susceptibility profile, which have important diagnostic and therapeutic implications.  

Outbreaks and pseudo-outbreaks caused by technical, instrumental, or laboratory contamination have been described extensively; therefore, recovery of rapidly growing mycobacteria from sterile and even non-sterile sites should always be questioned. Nonetheless, despite the possibility of contamination, recovery of rapidly growing mycobacteria from the bloodstream should be considered a true pathogen, until proven otherwise; thus, treatment should be provided, especially for immunocompromised patients. The panel lists the species of rapidly growing mycobacteria that cause bloodstream infections.  

**Biofilm formation and pathogenesis**  

Unlike other obligate human pathogenic mycobacterial organisms such as *M tuberculosis* and *Mycobacterium leprae*, the natural reservoirs of rapidly growing mycobacteria are aquatic and terrestrial environments. As environmental organisms, they usually exist in biofilms. Costerton and colleagues defined biofilms as matrix-enclosed bacterial populations that adhere to each other or to surfaces or interfaces, or both. Conversely, Zambrano and colleagues referred to biofilms as an extracellular matrix or layer that contains exopolysaccharides, proteins, and even nucleic acids in which organisms embed themselves and aggregate. Mycobacterial organisms are unique because they cannot produce exopolysaccharides, but produce mainly glycopeptidolipids and fatty acids; thus, mycobacterial biofilms have also been considered as a greasy way to hold microbial aggregates together on surfaces.  

During the past two decades, investigators of several studies have established that almost all species of rapidly growing mycobacteria can form biofilm, which enhances their ability to cause catheter-related bloodstream infections. In 1991, we described 15 cases of catheter-related bloodstream infections caused by *M fortuitum* and *M chelonae* and showed, with electron microscopy, that *M chelonae* can form biofilm and adhere to the surface of the central venous catheter. Subsequently, Hall-Stoodley and colleagues showed, with the modified Robbins device, that *M fortuitum* and *M chelonae* can form dense biofilms within 48 h on the surfaces of silastic discs. Esteban and colleagues showed that all rapidly growing mycobacteria species can form biofilm. However, *M abscessus* causes faster biofilm growth than did other rapidly growing mycobacteria, which is believed to contribute to the pathogenicity of this more virulent organism.  

Martin-De Hijas and colleagues correlated biofilm development with pathogenicity and clinical significance by testing 167 clinical strains of rapidly growing mycobacteria. Strains were deemed to be pathogenic and clinically significant if they were isolated from biopsy specimens, purulent exudates, and blood or bone marrow cultures. Biofilm formation was detected in 122 (73·2%) strains and involved all species tested. In the clinically significant strains, biofilm development was detected most commonly in *M abscessus* (87·5%) and was significantly associated with pathogenicity and clinical significance (p<0·001). Additionally, clinical significance and pathogenicity were significantly associated with sliding motility (p=0·0037). Furthermore, the microcolony structure of rapidly growing mycobacterial biofilm was described further by Williams and colleagues to include pillars of various shapes and extensive cording.  

The ability of rapidly growing mycobacteria to form dense biofilms not only explains their roles in the pathogenesis of catheter-related bloodstream infections, but also the need to remove the central venous catheter. Rapidly growing mycobacteria embedded in dense biofilm become resistant to antimicrobial treatment; hence, appropriate and active agents, given orally or intravenously, often do not eradicate these organisms from the catheter surface to which they adhere, necessitating removal of the central venous catheter. Hence, the organism’s ability to form dense biofilm contributes to its resistance to antimicrobial agents. Other pathogenic factors are the unique structure of the mycobacterial cell wall, which
serves as a permeation barrier; the ability of some mycobacteria to survive phagocytic cells; the rapid mutation of the target molecules; and the density of these organisms. 17-21

Definitions and characteristics
Central line-associated bloodstream infections are defined by the US Centers for Disease Control and Prevention as follows: at least one positive blood culture with a recognized pathogen (such as rapidly growing mycobacteria); an indwelling intravascular catheter (such as a central venous catheter) that has been in place for at least 48 h before the onset of bloodstream infection; signs and symptoms of infection (such as body temperature greater than 38°C, chills or hypotension); and neither the bloodstream infection nor the signs and symptoms of infection can be attributed to an infection at another site (eg, pneumonias or soft-tissue infections). 17 Therefore, most bloodstream infections caused by rapidly growing mycobacteria are central-line-associated bloodstream infections. El Helou and colleagues reviewed 111 patients with rapidly growing mycobacterial infections and concurrent central venous catheters; all patients fulfilled the criteria for central-line-associated bloodstream infections, with no other apparent infections at other sites.

The Infectious Diseases Society of America (IDSA) established more stringent criteria for catheter-related bloodstream infections. In addition to the criteria already listed, quantitative microbiological assessment is needed to ascertain that the central venous catheter is the source of the infection with the following criteria: a positive semiquantitative culture of the catheter segment, showing the same organism isolated from the bloodstream (in this case, rapidly growing mycobacteria); simultaneous quantitative blood cultures; or differential time to positivity. 70 However, several limitations hinder the diagnosis of catheter-related bloodstream infections caused by rapidly growing mycobacteria. First, cultivation of rapidly growing mycobacteria and their identification within 72 h is difficult. Second, catheter segments with no bacterial growth within 48-72 h are usually considered to be negative; thus, these catheters are discarded. Third, quantitative blood cultures and differential time to positivity are often not done in most medical centres. Therefore, the IDSA criteria for catheter-related bloodstream infections are difficult to fulfill and verify when bloodstream infection with rapidly growing mycobacteria is involved.

Other bloodstream infections, such as rapidly growing mycobacterial infections in cardiac implantable electronic devices and native and prosthetic valves, have also been described. 84-87 Because no specific definitions for rapidly growing mycobacteria exist, these complex intravascular infections are usually confirmed by extrapolation of data from other guidelines. 80,81 Infections related to a cardiac implantable electronic device can present as a range of syndromes, the most common of which is a generator pocket site infection. Local signs for infection associated with positive cultures for rapidly growing mycobacteria, obtained from intraoperative fluid collections, or the explanted cardiac implantable electronic device, are usually sufficient to confirm the diagnosis. Although a positive culture for rapidly growing mycobacteria recovered from purulent discharge from the pocket site suggests a true infectious process, the possibility of contamination should be considered, especially if the cardiac implantable electronic device has eroded through the skin. Cardiac implantable electronic device endocarditis and native and prosthetic valve endocarditis can be confirmed by use of the modified Duke’s criteria. 90-92 If a transthoracic echocardiogram does not indicate device-related endocarditis, the clinician should proceed with a more sensitive transoesophageal echocardiogram, especially if blood cultures are persistently positive. 90-92

Diagnosis of rapidly growing mycobacterial infections
In our clinical experience, rapidly growing mycobacterial bloodstream infections are usually not as virulent or aggressive as are other Gram-positive or Gram-negative bacterial bloodstream infections. These rapidly growing mycobacterial bloodstream infections are often associated with systemic symptoms, such as fever and chills, and local manifestations of infection, such as erythema, warmth, pain, tenderness, and drainage at the intravascular exit site. 9 Other, less commonly encountered rapidly growing mycobacterial infections, such as those cultured from cardiac implantable electronic devices, and native and prosthetic valves, usually present as a subacute infectious process. 92-94,95 These intravascular infections can progress into a myocardial abscess, which can further complicate the treatment course of such infections. 92-94 Although the mortality rate for uncomplicated rapidly growing mycobacterial bloodstream infections might be low, the mortality rate for cardiac implantable electronic device or valvular infections can be as high as 25%. 13,12,14,79

The main risk factors for bloodstream infection include immunosuppression, extended duration of catheter placement, and previous antimicrobial treatment. 84,85

When rapidly growing mycobacteria are identified from routine blood culture, they should always be regarded as true pathogens. However, because these organisms are frequently found in the environment, any breach in sterility could contaminate the blood, intravascular device, or tissue specimen. Several preventive measures can be used to reduce contamination, such as: avoidance of contact between the intravascular catheters and tap water; avoidance of multidose injection vials; not preparing ice from tap water in the operating room; and collection of samples in sterile, leak-proof, disposable, labelled, laboratory-approved containers. 13,12 These specimens can also be contaminated at any step within the laboratory, especially with tests that are manually labour intensive. Although transport media and preservatives are not usually recommended, refrigeration of samples at 4°C is preferred.
to decrease bacterial overgrowth, especially if transportation to the laboratory is delayed by more than 1 h.¹

Successful cultivation and recognition of rapidly growing mycobacteria in the blood might affect clinical care, in terms of timely empirical antimycobacterial treatment and intravascular catheter removal. Of equal importance is the rapid and accurate species identification that is used not only to refine clinical care but also to establish the pathogenicity and biological behaviour of the organism. Rapidly growing mycobacteria can be readily cultivated in routine blood cultures. Existing continuously monitored blood culture systems, such as Bactec (BD Diagnostic Systems, Sparks, MD, USA), Versa-Trek (Trek Diagnostic Systems, Cleveland, OH, USA), and BacT/Alert (BioMerieux, Durham, NC, USA), all work well. In our experience, quantitative culture using the Isolator method (Wampole Laboratories, Princeton, NJ, USA) by lysis and centrifugation of the blood yields especially good results. A blood sample (10 mL) is drawn into the isolator tube, and the leucocytes that contain intracellular bacteria are lysed and spun down to the bottom to be plated onto chocolate and sheep blood agar plates,⁶ both of which support growth of rapidly growing mycobacteria. Typically, after an incubation of 3–4 days, colonies, from a few up to more than 1000, appear on the plates. The first sign of rapidly growing mycobacteria is the Gram stain appearance of clusters of beaded Gram-positive rods (figure 1A). This finding will lead to use of a special stain to confirm mycobacteria; on a quick Kinyoun stain, rapidly growing mycobacteria appear as red bacilli on a blue background from the counterstain (figure 1B).

Traditional biochemical tests are unable to distinguish reliably among the many species of rapidly growing mycobacteria. For example, Conville and Wittebsky²⁹ reported that carbon (sugar) use tests are unable to identify M. mucogenicum. Biochemical tests are also more subjective than the newer methods (discussed below) in terms of judging reactions, and are time-consuming because they need an incubation period of 1–2 weeks to undertake or repeat.⁴⁶ These technical issues, combined with infrequent isolation of rapidly growing mycobacteria, mean that most hospital laboratories have to use reference laboratories to identify rapidly growing mycobacteria. Thus, developing countries might need to find an able laboratory or collaborator in a developed country for this purpose.

In the next few years, as new molecular methods become available, many more species will probably be identified. However, these new species might not be new microorganisms, but rather species that are technologically easier to identify. In the past two decades, these technological advances have enabled faster and more accurate identification of rapidly growing mycobacteria. Analyses of mycobacterial mycolic acids by high-performance liquid chromatography are available in most reference laboratories.⁶⁻⁶⁶ The recent widespread use of PCR and gene sequencing offers the most accurate identification of rapidly growing mycobacteria species.

Frequently analysed genes include the 16S rRNA gene, hsp65, rpoB, and others; all of these genes are highly conserved throughout bacterial evolution, but are variable enough to allow species identification.²⁵⁻⁵² Analyses of these genes are especially useful for the diagnosis and identification of mycobacteria because of the slow growth of these organisms and the scarcity of study devices in clinical laboratories. In our experience and that of several other research groups, sequencing of the first 500–600 base pairs of the 16S gene offers the most accurate, rapid, and cost-effective method to identify mycobacteria, including rapidly growing mycobacteria.³¹⁻⁷⁷ This method entails one PCR and one sequencing reaction to identify a rapidly growing mycobacterial species and usually takes 1–3 days after culture recovery, dependent on practical logistics. Figure 2 shows the preferred method for development of an identification flowchart for rapidly growing mycobacteria.

**Antimicrobial susceptibility**

A common feature of all rapidly growing mycobacteria is their resistance to first-line antituberculous agents. Antimicrobial susceptibility tests against rapidly growing mycobacteria can be done reliably with the broth dilution method, and the Clinical and Laboratory Standards Institute (CLSI) has established interpretation criteria for these organisms.⁹⁸ Antimicrobial agents that are active against these organisms include aminoglycosides, macrolides, fluoroquinolones, tetracyclines, carbapenems, cefoxitin, co-trimoxazole (trimethoprim–sulfamethoxazole), and linezolid.⁵²⁻⁹⁸ However, susceptibility patterns vary greatly between species (table).¹,⁴,⁶⁻⁷,¹۱,¹۵,⁴⁵,⁵۸,¹۰۱

*M. mucogenicum*, the most commonly identified rapidly growing mycobacterial bloodstream isolate, is usually susceptible to these antimicrobial drugs.¹,⁴,⁴⁵ In a study in

![Figure 1A](image1a.png)  
(A) Gram stain.  
(B) Kinyoun stain.
which *M. mucogenicum* was isolated in 45 (39%) of 116 patients, all isolates were susceptible to amikacin, clarithromycin, imipenem, and co-trimoxazole, whereas susceptibilities to cefoxitin and ciprofloxacin were 93% and 86%, respectively. M. fortuitum is usually susceptible to amikacin, ciprofloxacin, imipenem, and co-trimoxazole. However, the proportion of strains susceptible to clarithromycin is low compared with that of other rapidly growing mycobacterial species.81,83 Furthermore, studies have shown that *M. fortuitum* and related species, such as *Mycobacterium smegmatis*, contain inducible erythromycin methylase genes (*erm* gene), which can confer resistance to macrolides.80,83 Thus, even when these isolates are susceptible to macrolides, clarithromycin should be used with caution, especially when given as monotherapy.

*M. abscessus* is deemed to be one of the most virulent and resistant species of rapidly growing mycobacteria.44 It is usually resistant to imipenem, ciprofloxacin, co-trimoxazole, linezolid, and doxycycline, but is susceptible to amikacin and clarithromycin.44,53,102 Its susceptibility to cefoxitin varies, ranging from 7% to 75%.44,64-66 *M. abscessus* can also possess an inducible *erm* gene.20-26 Resistance to macrolides can also be caused by a mutation in the *rrl* gene, which encodes the 23S rRNA peptidyltransferase.103-105 Furthermore, mutations in the *rrs* gene can also affect the 16S rRNA, causing resistance to aminoglycosides.106,107 Therefore, empirical antimicrobial combination treatment might be needed to prevent antimicrobial resistance and clinical failure.

*M. chelonae* and *M. abscessus* have similar phenotypes and antimicrobial susceptibility profiles. *M. chelonae* is usually susceptible to amikacin, clarithromycin, and linezolid, and resistant to quinolone, cefoxitin, and co-trimoxazole. Susceptibility rates to doxycycline and imipenem are roughly 30% and 40%, respectively.11,44,101,102 Although *erm* methylase genes have been detected in *M. chelonae*, they are not associated with high minimum inhibitory concentration values to macrolides.85

*M. neoaurum* is usually susceptible to amikacin, clarithromycin, and linezolid, and resistant to quinolone, cefoxitin, and co-trimoxazole.11,44,101 Brown-Elliott and colleagues reported that after incubation of *M. neoaurum* with clarithromycin for 14 days, susceptibility to this drug decreased from 47% to 8%. These findings suggest induced *erm* gene expression or other mechanisms of clarithromycin resistance.

**Figure 2: Steps and timings for the identification of rapidly growing mycobacteria (RGM) by 16S rRNA gene sequencing**

![Figure 2](image-url)

**Table: Susceptibility results for rapidly growing mycobacteria bloodstream isolates by species and antimicrobials**

<table>
<thead>
<tr>
<th>Mycobacterium species</th>
<th>Amikacin</th>
<th>Cefoxitin</th>
<th>Ciprofloxacin</th>
<th>Clarithromycin</th>
<th>Doxycycline</th>
<th>Imipenem</th>
<th>Linezolid</th>
<th>Minocycline</th>
<th>Co-trimoxazole</th>
<th>Tobramycin</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>M. mucogenicum</em></td>
<td>100%</td>
<td>100%</td>
<td>90-100%</td>
<td>78-100%</td>
<td>80-100%</td>
<td>95-100%</td>
<td>80-100%</td>
<td>90-100%</td>
<td>95-100%</td>
<td>95-100%</td>
</tr>
<tr>
<td><em>M. fortuitum</em></td>
<td>100%</td>
<td>100%</td>
<td>75-100%</td>
<td>5-67%</td>
<td>66-100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>5-67%</td>
<td>100%</td>
</tr>
<tr>
<td><em>M. abscessus</em></td>
<td>86-88%</td>
<td>19-50%</td>
<td>0-7%</td>
<td>0-55%</td>
<td>100%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>M. chelonae</em></td>
<td>100%</td>
<td>100%</td>
<td>0-7%</td>
<td>0-55%</td>
<td>100%</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Susceptibilities were established by broth microdilution with use of the recommended guidelines from the Clinical and Laboratory Standards Institute (formerly National Committee for Clinical Laboratory Standards) for rapidly growing mycobacteria.14

**Therapeutic management**

During the past few decades, investigators of several studies of rapidly growing mycobacterial bloodstream infections have assessed patients’ characteristics, risk factors, and antimicrobial susceptibilities.1,3,4,11,45 However, no randomised controlled studies have been done to provide adequate recommendations about antimicrobial agents, treatment duration, or the importance of removal of the intravascular medical device.

**Monotherapy versus combination antimicrobial treatment**

A common challenge with rapidly growing mycobacteria bloodstream infections is the decision of whether to use a single or combination antimicrobial regimen. Many arguments tend to favour a combination approach. First, once rapidly growing mycobacteria colonies appear on the culture media, identification of the species with 16S ribosomal PCR analysis usually takes at least 3 additional days.16 Therefore, because of the noticeable differences in susceptibility patterns between species and among strains of the same species, no single antimicrobial agent (except amikacin) is active against all species.14,15 A second issue is the development of resistance, such as the acquisition of inducible *erm* methylase genes that confer macrolide resistance.106-107 Several other targeted mutations or efflux pumps cause resistance to quinolones and macrolides.107-109 Of note, the presence of these resistance mechanisms is not always associated with a higher
resistance profile. Third, the antimicrobial minimum inhibitory concentrations of these antimycobacterial drugs do not always represent in-vivo activity. Therefore, until the results of further studies are available, combination antimicrobial treatment seems to be reasonable.

**Empirical and targeted antimicrobial treatment**

Once a rapidly growing mycobacterium has been identified, effective empirical antimicrobial treatment should address the most commonly isolated rapidly growing mycobacteria: *M mucogenicum*, *M fortuitum* (complex), *M abscessus*, *M chelonae*, and *M neoaurum*. Generally, almost all species are susceptible to amikacin. *M abscessus* is usually macrolide susceptible but quinolone resistant. By contrast, *M fortuitum* and *M neoaurum*, which are usually quinolone susceptible, are resistant to macrolides, whereas *M mucogenicum* is often susceptible to quinolones and macrolides. Therefore, an initial antmycobacterial combination should ideally include intravenous amikacin, a quinolone, and a macrolide. After the isolate has been identified and the susceptibility panel made available, the original empirical regimen should be modified accordingly. The antimicrobial regimen should consist of at least two active drugs.1 If the patient is considered haemodynamically unstable and there are no side-effects to or contraindications for amikacin, continuation with this drug, plus a quinolone or macrolide, is reasonable.

**Antimicrobial treatment duration**

The ideal duration of treatment for pulmonary, skin, and bloodstream rapidly growing mycobacterial infection is controversial. For lower respiratory tract infections, antibiotic treatment for up to 12 months might be recommended, whereas at least 6 months of treatment is suggested for osteomyelitis.1 The duration of treatment for uncomplicated rapidly growing mycobacterial bloodstream infection differs between studies; nonetheless, at least 4 weeks of a combination antimicrobial regimen is associated with resolution of the infectious process.1

**Management of intravascular foreign medical devices**

Rapidly growing mycobacterial bloodstream infections almost always occur in patients with indwelling intravascular catheters and are mostly central-line-associated bloodstream infections.18,31-36 Infections of intracardiac vascular devices and prosthetic valves have also been reported.12,37-79 The ability of rapidly growing mycobacteria to form biofilms that adhere to the surface of these devices plays a major part in these infections. Findings from many studies have shown a benefit with removal of intravascular devices.12,37-39,75-79,81 A recent study at our institution showed that removal of the intravascular catheter was associated with a significant decrease in the rate of bloodstream relapse (p=0.007).7 Therefore, in addition to provision of adequate antmycobacterial treatment, removal of the intravascular foreign medical device is crucial (figure 3).

**Management in resource-poor settings**

Because the medical treatment of rapidly growing mycobacterial infections is difficult and prolonged treatment is usually needed, the cost of care in resource-poor countries can become prohibitive, especially when intravenous amikacin, oral macrolides, and quinolones are incorporated into the antimicrobial regimen. Rapidly growing mycobacteria species that have been identified in developing countries can also have a specific antimicrobial resistance profile, probably because of geographical distribution, population antimicrobial exposure, and methodological issues.12,13,19-21 Therefore, to provide adequate antimicrobial recommendations, and to understand the complexities of rapidly growing mycobacteria antimicrobial resistance, studies in resource-poor countries are urgently needed.1

**Outcome**

Overall, rapidly growing mycobacterial bloodstream infections are associated with a good clinical outcome.

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**Figure 3: Proposed treatment algorithm for rapidly growing mycobacterial bloodstream infections**

[Diagram showing the proposed treatment algorithm for rapidly growing mycobacterial bloodstream infections]
The use of appropriate antimicrobials and removal of the intravascular catheter leads to resolution of the infectious process in more than 90% of cases. Relapses have been associated with the intravascular catheter not being removed. Compared with other bacterial bloodstream infections, the mortality rate associated with rapidly growing mycobacterial bloodstream infections is usually low.

Conclusions

Many rapidly growing mycobacterial species have been identified in human beings, causing a wide range of infectious syndromes. Although these bloodstream infections are not highly virulent or life threatening, they have a high predisposition to create a biofilm, and to colonise and infect intravascular catheters. Until proven otherwise, these organisms should be regarded as true pathogens. Early identification at the species level can provide predictable antimicrobial susceptibility patterns. The best chance of cure is obtained with a combination of at least two active antimicrobials given for a minimum of 4 weeks, plus removal of the intravascular catheter.

Contributors

GEH and GMV, as first coauthors, contributed equally to drafting and revision of the Review with input from all authors. All authors approved the final version.

Conflicts of interest

IIR has served on the speaker’s bureau for Cook, has received grant funding from Cook, and has received royalties related to patents licensed to Cook in which he is an inventor or co-inventor (of coated intravascular catheters). All other authors declare that they have no conflicts of interest.

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Mycobacterium abscessus: a review of the literature.


Review


