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CASE REPORT

Native-valve endocarditis caused by *Mycobacterium chelonae*, misidentified as polymicrobial gram-positive bacillus infection

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Abstract *Mycobacterium chelonae*, a species of rapidly growing mycobacteria, may grow in routine blood culture media and stain as gram-positive bacilli, which may cause diagnostic confusion. A patient with native-valve endocarditis caused by *M. chelonae*, which was misidentified as various gram-positive bacilli, is presented.

Keywords Gram-positive bacilli · *Mycobacterium chelonae* · Native-valve endocarditis · Nontuberculous mycobacteria · Rapidly growing mycobacteria

Introduction

Mycobacterium chelonae is a rapidly growing mycobacteria (RGM) and a rare cause of endocarditis. In contrast to slow-growing mycobacteria, RGM may grow in routine blood culture media [1] and stain as gram-positive bacilli [2]. Although well known by experts, the gram positivity is not widely recognized by many physicians and technicians. Unless mycobacterial disease is suspected and acid-fast staining is applied, mycobacteria can be confused with other gram-positive bacilli [3, 4], which can lead to critical delay in diagnosis and treatment. We report a case of native-valve endocarditis caused by *M. chelonae* that was initially misidentified as multiple kinds of gram-positive bacilli.

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Case report

A 57-year-old man presented with fever and chest and abdominal pain. He was an intravenous drug user and had been diagnosed with cirrhosis from hepatitis C and alcoholism 10 years earlier. His past medical history was notable for splenic marginal-zone B-cell lymphoma that was successfully treated with rituximab and radiation therapy 7 years earlier. He had been diagnosed with *Staphylococcus aureus* endocarditis of the mitral valve 4 years before presentation. A patent foramen ovale and Chiari network were noted by the echocardiogram at that time. Soon after hospitalization, he developed bleeding esophageal varices that required vasopressors and intubation. Blood cultures on admission grew methicillin-resistant *S. aureus* that was treated with intravenous vancomycin. Subsequent blood cultures were negative, and the echocardiogram did not demonstrate any vegetation but did confirm previously noted moderate mitral regurgitation. He was successfully extubated on hospital day 7 and did well until hospital day 15 when he developed dyspnea, which was attributed to fluid overload and successfully treated. Blood cultures at this time were positive for *Rhodococcus equi* and *Brevibacterium*. The identifications were made using a biochemical panel for gram-positive bacteria, RapID system (Remel, Lenexa, KS, USA). He was treated with piperacillin–tazobactam. On hospital day 25, he developed pneumonia, and blood cultures grew extended-spectrum beta-lactamase-positive *Klebsiella pneumoniae* as well as *Corynebacterium* species and unidentified gram-positive bacilli. The piperacillin–tazobactam was switched to imipenem, and his central line was removed. Repeated blood cultures were negative. His condition subsequently improved and was discharged home.

One month later, he returned to the hospital complaining of worsening dyspnea. His electrocardiogram showed a

prolonged PR interval of 290 ms. A transesophageal echocardiogram showed severe mitral regurgitation and a 2 × 3 cm abscess along the inferior and septal walls, posterior to the mitral annulus. The abscess communicated with the left ventricle, and a fistula extended into the left atrium. He was started on imipenem, vancomycin, and anidulafungin. Blood cultures grew gram-positive bacilli, and anidulafungin was discontinued. Because the biochemical panel failed to identify the gram-positive organism, the sample was sent to a reference laboratory, Quest Diagnostics (Madison, NJ, USA). The technician there noticed the characteristic appearance of the mycobacterium in the Gram stain and confirmed it with an acid-fast stain. By hospital day 17, subsequent biochemical and chromatographic studies identified the gram-positive bacilli as *M. chelonae*. Gram-positive bacilli previously reported as *Rhodococcus*, *Brevibacterium*, and *Corynebacterium* from the previous hospitalization were also sent to reference laboratories, and all were identified as *M. chelonae* with the Sherlock Mycobacterial ID System (MIDI, Newark, DE, USA), which is a mycobaterial identification system based on mycolic acid analysis by high-performance liquid chromatography, by hospital day 25. Antibiotics were switched to clarithromycin, amikacin, and cefoxitin according to the susceptibility results. Unfortunately, the patient developed variceal bleeding and died shortly thereafter.

Discussion

This case shows how multiple positive blood cultures in an immunocompromised person actively injecting street drugs can lead clinicians astray if mycobacteria are not considered when culture results showed gram-positive bacilli. In our case, the diagnosis of *M. chelonae* infection was delayed because it was not considered and acid-fast staining was not initially performed.

Mycobacterium chelonae are RGM defined as a subgroup of mycobacteria that form colonies on subculture within 7 days [1]. These environmental bacteria can be found worldwide in soil, dust, rocks, bioaerosols, and water [1, 5]. Although usually nonpathogenic, they can cause symptomatic diseases [1]. Skin, bone, and soft tissue disease associated with trauma or surgery are the most common presentations [1], but endocarditis has also been reported. Microscopically these bacteria appear as slender, poorly stained, beaded gram-positive bacilli [2]. *M. chelonae*, similar to other rapid growers, can grow in the usual blood culture media, although these characteristics depend on the Gram staining technique, blood culture media, and culture conditions, such as temperature [3, 4, 6–8].

The difficulty in suspecting and identifying RGM has been shown in several studies. The Quality Control Center Switzerland mailed out *M. fortuitum* specimens, another RGM, labeled as "pus from an abscess, to be investigated for potentially pathogenic bacteria" to their participating laboratories [3]. Only 13 of the 50 participants (26 %) correctly identified it as "rapidly growing mycobacterium" or "*M. fortuitum*" based on Ziehl–Neelsen stains and biochemical tests. Twenty-three participants (46 %) diagnosed *Nocardia* sp. and 4 (8 %) participants diagnosed *Rhodococcus* sp. Another 8 participants reached incorrect or insufficient diagnosis as gram-positive rods, *Actinomyces*, *Streptococcus*, or *Corynebacterium*. All laboratories that did not use acid-fast stains failed to identify the organism correctly. Although all laboratories used Gram stains and biochemical studies, only 31 (62 %) used acid-fast stains, and 6 laboratories that used classical acid-fast stains incorrectly diagnosed *Nocardia*. Williamson and colleagues [4] also reported a fatal case of *Mycobacterium abscessus* endocarditis that was misidentified as *Corynebacterium* spp. using Gram stain morphology and growth characteristics.

In our case, the organisms in the blood cultures from his initial hospitalization were reported as *Rhodococcus*, *Brevibacterium*, *Corynebacterium*, and unidentified gram-positive bacilli. Our microbiology laboratory failed to recognize the morphological characteristics of mycobacteria in the Gram stain and used an inappropriate biochemical panel designed for gram-positive bacilli.

The case we report was also unusual in that *M. chelonae* causing native-valve endocarditis is extremely rare. Endocarditis caused by RGM usually occurs on prosthetic valves or indwelling devices sporadically or as part of outbreaks caused by contaminated valves or surgical equipment [9]. In the rare case reports of native-valve infections with RGM, the majority of patients have had a medical condition that predisposed them to infections and a port of entry to the blood stream, such as intravenous "street" drug use and hemodialysis [4, 10–19]. Many of the patients had damaged valves. Some cases had no identifiable predisposing conditions [10, 15]. Our patient carried multiple predisposing medical conditions including alcoholism, cirrhosis, hematological malignancy, and damaged valves, as well as multiple sources of potential bacteremia, including IV drug abuse, variceal bleed, endotracheal intubation, and central venous catheter placement. The disease course was subacute with a duration of weeks to months. Clinical presentations were usually systemic illness with fever and malaise. Blood cultures were generally positive, and the echocardiogram confirmed valvular involvement. Left-sided valves are involved more often than right sided. When reported, antibiotic susceptibility patterns often demonstrated resistance to multiple classes

of antibiotics. Multidrug therapy has been recommended because of the tendency of developing resistance during long-term treatment [1, 20]. Given that it is generally difficult to eradicate mycobacterial infection, surgical debridement was often recommended [21]. However, just as in our patient, often surgery could not be performed because of the poor general medical condition of these patients. Despite these interventions, the prognosis was generally poor. Most of the reported cases were fatal.

In conclusion, even though gram-positive bacilli and mycobacteria are uncommon causes of endocarditis, they are more likely in the setting of immunocompromised hosts with open access to the blood stream. Rapidly growing mycobacteria (RGM) are gram-positive bacilli that may grow in standard blood culture media and should be considered as potential causative organisms. Gram-positive bacilli cultured from high-risk patients should undergo acid-fast staining and mycobacterial culture.

Conflict of interest None.

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