Short Communication

Post-Laparoscopic Wound Infection Caused by Scotochromogenic Nontuberculous Mycobacterium

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SUMMARY: Nontuberculous mycobacteria are known to produce soft-tissue infections following surgical procedures. We report a non-healing surgical wound infection caused by a scotochromogenic nontuberculous mycobacterium, possibly Mycobacterium flavesceus, which was earlier thought to be saprophytic. This organism was isolated from the wound discharge at the site of incision following a laparoscopic cholecystectomy; the finding suggests that M. flavesceus is a clinically important pathogen in cases of surgical wound infections.

Nontuberculous mycobacteria (NTM) have been isolated from various sites, viz., sites of soft-tissue infections, minor trauma, and surgery, using various procedures (1). Mycobacterium fortuitum and Mycobacterium chelonae frequently cause such infections (1). However, Mycobacterium flavesceus, a scotochromogenic, has rarely been reported to cause such infections (2,3). This organism was earlier thought to be saprophytic, and its isolation from samples has always been attributed to either colonization or contamination in the laboratory. Isolation of M. flavesceus from a non-healing surgical wound prompted this present study.

A 40-year-old woman underwent laparoscopic cholecystectomy for symptomatic cholelithiasis at a community hospital. Six weeks later, she developed fever (39.3°C) and discharging sinuses at the epigastric and hypochondrial port sites, and hence, presented to the outpatient surgery department at the Post Graduate Institute of Medical Education and Research, Chandigarh, India. Sinus exploration at both these sites failed to reveal any foreign body. The sinuses were curetted, and the discharge was cultured on blood agar (Hi Media, Mumbai, India) and MacConkey agar (Hi Media) to observe the growth of aerobic and anaerobic bacteria. The plates were incubated at 37°C overnight and for 48 h for anaerobic organisms. The patient was then empirically administered a combination of amoxicillin and clavulanic acid following which she showed some improvement in symptoms. The fever (39.3°C) persisted for approximately 8 days, after which it subsided. However, the culture did not show growth of any microorganism. Gram staining, Ziehl-Neelsen staining, and fungal staining of the sinus discharge did not reveal any microorganism. Fine-needle aspirate (3 ml) from the right abdominal swelling revealed a resolving inflammatory pathological condition. Twelve weeks later, she presented with occasional mild fever (38.3°C), which persisted for up to 3 months. She developed nodularity with mild pain at one of the port sites. Histopathological examination of the lesion showed granulomatous inflammation with necrosis and Langhans' type giant cells. Initial laboratory studies showed normal blood count (88 × 109/l); with 70% neutrophils and 32% lymphocytes; the findings of urinalysis and serum biochemical analysis were also found to be normal. Radiographic examination of the chest, abdomen, and pelvis did not reveal any abnormal findings. Erythrocyte sedimentation rate (ESR) was 124 mm/h and C-reactive protein (CRP) level was 7.8 mg/dl. The tuberculin skin test showed highly positive results with an induration of 20 × 20 mm. She was then administered antitubercular therapy (ATT) with the four-drug regimen (isoniazid, rifampicin, streptomycin, and pyrazinamide), but she did not respond to 4 months of therapy.

After the discontinuation of ATT for 1 month, she developed an abscess with multiple sinuses at the right hypochondrial port site. She showed elevated CRP levels (9.4 mg/dl) and ESR (140 mm/h) and persistent low-grade fever. Debridement and drainage was performed, and the collected pus sample was tested for culture sensitivity. The pus samples were cultured on blood agar, MacConkey agar, Lowenstein-Jensen medium, Mycobacteria Growth Indicator Tube (MGIT 960), and Sabouraud’s dextrose agar for detecting aerobic and anaerobic organisms, i.e., mycobacteria and fungi, respectively. Staining procedures including Gram staining and acid-fast staining were performed to detect fungi. Acid-fast staining by the Ziehl-Neelsen method showed short and stout acid-fast bacilli (AFB), and the culture showed the growth of smooth yellow colonies with 2+ growth after 2 weeks of incubation at 37°C on Lowenstein-Jensen medium. Growth was observed on the MGIT 960 on the 4th day, and testing with para-nitro benzoic acid (PNB) confirmed that the growing organism was a nontuberculous mycobacterium (NTM). The organism was identified as M. flavesceus on the basis of the following findings: the colonies were scotochro-

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mogenic (grow when kept in the dark), were resistant to inhibition by PNB, and tested positive for 68°C catalase, nitrate reductase, Tween 80 degradation, and 5% NaCl tolerance (4). Susceptibility testing of these isolates was performed using the Etest method (AB Biodisk, Solna, Sweden) (5). The organism was found to be sensitive to amikacin (minimum inhibitory concentration [MIC], 0.25 μg/l), ofloxacin (MIC, 0.002 μg/l), ciprofloxacin (MIC, 0.25 μg/l), kanamycin (MIC, 0.03 μg/l), cefoxitin (MIC, 0.12 μg/l), ethambutol (ETM) (MIC, 16 μg/l), rifampicin (RIF) (MIC, > 256 μg/l), isoniazid (INH) (MIC, 8 μg/l), and streptomycin (STM) (MIC, > 256 μg/l). Thus, the isolate was resistant to primary antitubercular drugs (INH, RIF, STM, and ETM) and sensitive to all other drugs including ofloxacin and amikacin. Specific treatment for Mycobacterium flavescens, i.e., using ofloxacin and amikacin, was initiated after 8 months of initial operation. The course of treatment with ofloxacin was continued for 6 months. The patient’s condition was normal, during the 6-month follow-up, and recurrence of signs of wound infection was not observed. She had gained weight, and did not have any fever. The CRP levels normalized during the first week of specific antitubercular therapy, and the ESR normalized after 14 days.

Our patient’s infection was most likely caused by scotochromogenic NTM, possibly Mycobacterium flavescens, because AFB were seen in the stained pus obtained from the abscess, and pus culture showed growth of numerous colonies (with 2+ growth) of mycobacteria, which were biochemically identified as Mycobacterium flavescens. All other aerobic and anaerobic cultures were sterile, and multiple samples (pus/discharge) from different sites (discharging sinuses at the right hypochondrial port site) showed the same results, i.e., growth of scotochromogenic NTM possibly Mycobacterium flavescens. Furthermore, the patient’s condition improved relatively quickly with appropriate therapy. All these evidences clearly suggest that scotochromogenic NTM possibly Mycobacterium flavescens were indeed the cause of the patient’s prolonged illness.

M. flavescens is a known environmental contaminant in water, and its colonization in humans has only rarely been associated with disease. However, few studies have shown that this organism is the cause of muscle abscesses, postsurgical abscesses, and infections following penetrative trauma (2,3). Gluteal abscesses caused by Mycobacterium flavescens infection following intramuscular injection have also been reported (2). Fischer et al. (3) have reported a case of postsurgical infection caused by Mycobacterium flavescens after spinal surgery in a child. In the present study, we could not confirm the source of infection; however, we found that the infection in our patient fits the pattern of infection potentially resulting from the introduction of mycobacteria during surgery because the infection was present at the site of laparoscopic surgery. We could not determine the best duration of treatment for the patient, but found that this patient responded very well to a long course of treatment.

In conclusion, this study shows that scotochromogenic NTM (M. flavescens) is a clinically important pathogen in cases of surgical wound infections. Thus, mycobacterial infections must be considered in wounds that show delayed healing and do not respond to antibiotics used for acute pyogenic infections. High degree of suspicion, specific identification, and susceptibility testing will enable timely administration of appropriate antimicrobial therapy.

Conflict of interest None to declare.

REFERENCES