

Surgical Site Infection due To *Mycobacterium mageritense* and Literature Review

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Abstract

Surgical site infection due to *Mycobacterium mageritense* is reported in an 85-year-old Japanese woman. In this patient, a mesh patch was inserted to close a ventral hernia and surgical site infection occurred 7 days postoperatively. The isolate was confirmed to be *Mycobacterium mageritense* by analysis of the 16S rRNA and *rpoB* genes. The infection was successfully treated with negative pressure wound therapy and long-term antibiotics without removal of the mesh patch.

Keywords: *Mycobacterium mageritense*; Surgical site infection; 16S rRNA; *rpoB*; Negative pressure wound therapy

Introduction

Mycobacterium mageritense is a rapidly growing nonpigmented mycobacterium that most closely resembles the *M. fortuitum* third biovariant complex [1]. There have been few reports about skin or soft tissue infections, especially surgical site infection, due to *M. mageritense* [2-5]. Here we report the first case of successful treatment of surgical site infection due to *M. mageritense* without removing the implanted mesh patch.

Case Report

An 85-year-old woman was admitted to our hospital for surgery to control a symptomatic incisional hernia. In June 2006, she had undergone extended resection and transplantation of a rectus abdominis musculocutaneous flap for Paget's disease of the vulva. However, a postoperative ventral hernia developed along with cicatricial stenosis of her anal region. In October 2006, a second operation was performed for the ventral hernia and it was found that the hernial orifice was too large to close, so it was reinforced with mesh. Subsequently, we recommended re-operation with a mesh patch, but the patient declined. After her symptoms gradually became worse and control of excretion was impaired, she eventually agreed to surgery.

In October 2015, hernia repair was performed with a mesh patch. On postoperative day 7, the abdominal wall became tense and the wound discharge changed from serous to yellowish-brown and purulent. A swab of the wound site was taken to test for microorganisms. Then the wound was opened widely and we performed irrigation and debridement, followed by negative pressure wound therapy (NPWT). An acid-fast bacillus was detected after only 2 days of culture of the wound swab on blood agar at 35°C under

aerobic conditions and was identified as *Mycobacterium abscessus* by the DNA-DNA hybridization method (Kyokuto, Japan).

We also performed drug susceptibility testing and found resistance to clarithromycin, which suggested that other rapidly growing mycobacteria should be considered. Therefore, we performed analysis of the 16S ribosomal RNA (16S rRNA) and *rpoB* genes by amplifying sequences of 1,391 bp and 723 bp, respectively. According to the Ez-taxon database (<http://www.ezbiocloud.net/eztaxon>), the isolate showed 100% concordance of its 16S rRNA sequence with that of *Mycobacterium mageritense* (DSM 44476^T) and 99.57% concordance with *Mycobacterium peregrinum* (ATCC 14467^T). According to the BLAST database (<http://blast.ddbj.nig.ac.jp/blastn?lang=ja>), the *rpoB* sequence of the isolate showed 100% agreement with that of *M. mageritense* (ATCC 700351). Thus, we concluded that the isolate was *M. mageritense*.

NPWT was continued with wound irrigation at every dressing change and intravenous antimicrobial therapy was provided with a combination of levofloxacin and imipenem/cilastatin for 6 weeks. Signs of infection improved and granulation of the wound occurred gradually, after which we switched her antimicrobial therapy to oral minocycline and levofloxacin for an additional 3 months.

Discussion

M. mageritense was first described by Domenech et al. [1]. Similar to other rapidly growing mycobacteria like *Mycobacterium fortuitum* and *M. abscessus*, this microorganism is commonly linked to human disease. Similar with other rapidly growing mycobacteria, this organism is ubiquitous and also isolated from most municipal water supplies. In our case, infection might be established through water. There have been few reports describing skin or soft tissue infections due to *M. mageritense*. Table 1 summarizes the reported cases of skin and soft tissue infection [2-6].

Unlike other rapidly growing mycobacteria, *M. mageritense* is resistant to clarithromycin [2]. In our patient, the isolate was resistant

to clarithromycin but susceptible to fluoroquinolones, imipenem, and minocycline.

Age	Sex	Past history	Surgical procedure	antibiotics	Duration of ABx	Duration for heal	Reference number
37	Female	Liposuction (surgical wound infection)	Yes	CFPX, DOXY	6 months	6 months	2
25	Male	Compound fracture of the knee(wound infection)	Yes	AMK, IPM	ND	ND	2
43	Female	No	No	ST, LVFX	3 months	3 months	3
56	Female	No	No	GFLX	2 months	2 months	3
48	Male	Wound after trauma in tsunami survivor	ND	ND	ND	12months	4
66	Male	Gastrectomy (surgical wound infection)	Yes	MLFX, CAM	4days	At least 2 months	2 5
52	Female	No	Yes	CAM, LVFX	6 months	Less than 6 months	6

CFPX: Ciprofloxacin, DOXY: Doxycycline, AMK: Amikacin, IPM: Imipenem, ST: Sulfamethoxazole-trimethoprim, LVFX: Levofloxacin, GFLX: Gatifloxacin, MFLX: Moxifloxacin, CAM: Clarithromycin

Table 1: Past reports of skin and soft tissue infection due to *Mycobacterium mageritense*.

This case suggests that if a rapidly growing mycobacterium is isolated which shows resistance to clarithromycin, it is necessary to perform a gene assay instead of DNA-DNA hybridization. Adekambi et al. suggested that *rpoB* is more suitable than 16S rRNA for diagnosis of rapid growth mycobacterias [7]. And for the identification of the isolate in our case, 16S rRNA was also useful. Thus, we recommend to use *rpoB* gene analysis together with 16S rRNA for identifying rapid growth mycobacteria. Also, even if the strain is initially susceptible to macrolides, resistance to these drugs is easily induced by endogenous resistance genes, so it is thought that the use of macrolides for *M. mageritense* infection should be restricted [8]. Thus, it may be necessary to repeat drug susceptibility testing, particularly at institutions that cannot perform gene assays.

The treatment of *M. mageritense* infection has not been established and it varied in the cases reported to date (Table 1). Because infection was refractory, antibiotic therapy was performed for several months in the previous patients and was combined with drainage and surgical treatment.

Unlike the reported cases, our patient's wound infection improved with repeated irrigation and NPWT plus antimicrobial therapy, and we did not remove the mesh patch taking her overall status and her request for no further surgery into consideration. When infection of a foreign body occurs, it often becomes a reservoir for bacteria and leads to treatment failure. Kannaiyan et al. reported a series of surgical site infections caused by rapidly growing mycobacteria [9], including four cases with a mesh. And the mesh was removed in all patients.

NPWT is a method of reducing edema by removal of intercellular lymph fluid, and it promotes wound healing through this mechanical effect as well as reducing the bacterial burden by suction drainage [10]. With our combined approach to treatment, it soon became impossible to isolate *M. mageritense* from the wound, but complete granulation required several months. In conclusion, removal of the mesh patch may have promoted more rapid healing and is recommended when the

patients' condition permits it if surgical site infection due to rapid growth mycobacteria is detected, but combination therapy with NPWT and long-term antibiotic therapy could be the alternative strategy when the patients' condition does not permit.

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