ATYPICAL MYCOBACTERIAL EXIT-SITE INFECTION AND PERITONITIS IN PERITONEAL DIALYSIS PATIENTS ON PROPHYLACTIC EXIT-SITE GENTAMICIN CREAM

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We report 9 cases of exit-site infection and continuous ambulatory peritoneal dialysis peritonitis associated with atypical mycobacteria. All patients had been using topical gentamicin cream as prophylaxis for exit-site infection before the onset of these infections. Gentamicin cream is postulated to be a potential risk factor for atypical mycobacterial infection because of selective pressure on other micro-organisms. The microbiology of atypical mycobacteria and the treatment for atypical mycobacterial infections are discussed.

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Peritoneal dialysis (PD) is an effective renal replacement therapy for end-stage renal failure (ESRF) patients. However, one of the most important complications related to this therapy is infection. Catheter exit-site infection (ESI) is a major source of morbidity. It has been suggested that ESI increases the risk of catheter loss, of peritonitis from the same micro-organism, and of overall PD technique failure (1).

Various methods have been adopted to lower the rate of ESI, including application of topical antibiotics at the exit site. Mupirocin cream has been shown to be effective against gram positive bacteria, including methicillin-resistant *Staphylococcus aureus* and certain gram negative bacteria. However, it is less effective against most gram-negative bacilli and anaerobic bacteria (2). For example, the minimum inhibitory

concentration of mupirocin with respect to *Escherichia coli*, *Klebsiella pneumoniae*, *Enterobacter* species, and *Proteus mirabilis* is 64 µg/mL – 128 µg/mL. The minimum inhibitory concentration even reaches 1600 µg/mL – 6400 µg/mL with respect to *Pseudomonas aeruginosa* and *Morganella morganii*. As a result, other broader-spectrum antimicrobial agents have been studied in the hopes of overcoming the weakness of mupirocin.

Gentamicin cream has been shown not only to be effective against *P. aeruginosa* and other gram-negative bacteria, but also to be as effective as mupirocin cream in reducing *S. aureus* infection. Gentamicin cream has been recommended as the agent of choice for the prevention of ESI (3). However, concerns about its routine use include the emergence of resistance and the overgrowth of non-susceptible organisms.

The issue of rapid-growing mycobacterial ESIs after prolonged gentamicin application has been raised (4). Whether gentamicin cream should be used indiscriminately for ESI prevention is still unknown. We report 9 cases of ESI caused by atypical *Mycobacterium* that occurred at our center during 2007 – 2010, all after the use of prophylactic gentamicin cream (Table 1). Exit-site infection was defined as presence of purulent or bloody discharge that may be associated with erythema, tenderness, exuberant granulation tissue, or edema (5).

Ethics Committee approval was obtained for publication of human data.

CASE REPORTS

CASE 1

A 63-year-old woman with ESRF because of lupus nephritis had been on continuous ambulatory PD (CAPD) since 2006. She was started on prophylactic

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TABLE 1
Clinical Presentation, Culture and Sensitivities, and Treatment Outcome of Atypical Mycobacterial Infections in
Nine Patients on Continuous Ambulatory Peritoneal Dialysis

Variable	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9
Infection type	ESI	ESI	Peritonitis	ESI	ESI	ESI	ESI	Peritonitis	ESI
Date	26 Apr 2007	26 Jul 2007	29 Jul 2007	21 Sep 2007	3 Oct 2007	3 Dec 2007	1 Sep 2008	8 Jan 2010	23 Aug 2010
Last-dose antibiotic	Cefazolin	Vancomycin	Ceftazidime plus ciprofloxacin	On vancomycin	Cloxacillin	Ampicillin/ clavulanate	Vancomycin	No antibiotics before infection	Cefepime
Interval from last antibiotic to infection (months)	4.5	0.5	5.5		23	6.5	14.5		6
Duration of topical gentamicin before infection (months)	8	9	21	5.5	10.5	11	30	1	24
Mycobacterial species	<i>M. fortuitum</i>	<i>M. abscessus</i>	<i>M. abscessus</i>	<i>M. chelonae</i>	<i>M. abscessus</i>	<i>M. abscessus</i>	<i>M. fortuitum</i>	<i>M. abscessus</i>	<i>M. abscessus</i>
Test	Hsp, Gh	Hsp, Hf	Hsp, Gf	Hsp, HiX2 16s	Hsp, Hf	Hsp, Hf	Hsp, Gc	Hsp, Gf	Hsp, Gf
Sensitivity profile									
Amikacin	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive
Cefoxitin	Sensitive	Insensitive	Insensitive	Resistant	Insensitive	Insensitive	Insensitive	Sensitive	Sensitive
Clarithromycin	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive	Sensitive
Doxycycline	Insensitive	Resistant	Resistant	Sensitive	Resistant	Resistant	Resistant	Resistant	Resistant
Levofloxacin	Sensitive	Resistant	Resistant	Insensitive	Resistant	Resistant	Sensitive	Resistant	Resistant
Moxifloxacin								Sensitive	
Linezolid				Sensitive	Sensitive	Sensitive		Sensitive	
Treatment duration (weeks)									
IP amikacin	20	8	8	12	14	8	2	4	
IV cefoxitin			28				6		
P0 clarithromycin		28		8	14	42	18		11
P0 doxycycline				8					
P0 levofloxacin	20	4							
P0 moxifloxacin								20	
IV meropenem		2	4						
P0 azithromycin								6	
Outcome	CAPD uninterrupted	Tenckhoff catheter reinserted 5 months later	Switched to HD	CAPD uninterrupted	CAPD uninterrupted	CAPD uninterrupted	Tenckhoff catheter reinserted 16 months later	Tenckhoff catheter reinserted 9 months later	Switched to HD

ESI = exit-site infection; Hsp = heat shock protein analysis; IP = intraperitoneal; IV = intravenous; P0 = oral; CAPD = continuous ambulatory peritoneal dialysis; HD = hemodialysis.

exit-site gentamicin cream (0.1%, produced by Perrigo Company, Allegan, MI, USA) from the commencement of CAPD. She had been taking low-dose prednisolone (5 mg daily) for residual lupus activity. In November 2006, she experienced 1 episode of CAPD peritonitis from *Streptococcus viridans*, and she was treated with intraperitoneal (IP) cefazolin for 2 weeks.

Five months later, this patient presented with her first episode of ESI. Bacterial culture grew *M. fortuitum*. She was given IP amikacin and oral levofloxacin for 20 weeks. She responded well to the treatment, and CAPD was continued without the need for removal of the Tenckhoff catheter.

CASE 2

An 81-year-old woman with immunoglobulin A nephropathy, diabetes mellitus, and hypertension had been on CAPD since March 2006. She was started on prophylactic gentamicin in December 2006. She experienced an episode of methicillin-resistant coagulase-negative *Staphylococcus* ESI in 2007 and was treated with vancomycin for 2 weeks. However, the ESI was refractory to treatment and a subsequent culture yielded *M. abscessus*. She was treated with intravenous meropenem, oral clarithromycin, oral levofloxacin, and IP amikacin. The Tenckhoff catheter was removed 2 weeks later in view of persistent purulent discharge; the catheter was reinserted 5 months later. Since then, she has remained on CAPD uneventfully.

CASE 3

A 39-year-old woman with crescentic mesangio-proliferative glomerulonephritis had been on CAPD therapy since 2002. She had recurrent *P. aeruginosa* ESI during 2004 – 2007, requiring prolonged courses of antibiotic therapy. She was started on prophylactic gentamicin cream in January 2005.

This patient presented with peritonitis and ESI in July 2007 and was empirically treated with IP cefazolin and gentamicin. Bacterial culture of the exit-site swab and peritoneal fluid grew *M. abscessus*. She was treated with clarithromycin, amikacin, and meropenem. Her Tenckhoff catheter was removed because of poor clinical response. To avoid ototoxicity, amikacin was replaced with cefoxitin after 8 weeks. Cefoxitin was continued for 28 weeks because of an intra-abdominal collection that required drainage. Her CAPD treatment was discontinued, and she was switched to long-term hemodialysis.

CASE 4

A 68-year-old man with renal failure from unknown causes had been on CAPD since 1996. He had recurrent bacterial ESIs and was started on prophylactic gentamicin cream in May 2007. He developed an episode of methicillin-resistant coagulase-negative *Staphylococcus* ESI 2 months later. Despite prolonged treatment with IP vancomycin and oral rifampicin, and local debridement twice, discharge from the exit site persisted. Four weeks after the initial ESI presentation, the exit-site swab grew *M. chelonae*. The ESI was controlled with oral clarithromycin and doxycycline, together with IP amikacin, for 12 weeks. This patient continued CAPD uneventfully till he succumbed to *E. coli* peritonitis 9 months later.

CASE 5

A 58-year-old man with diabetic nephropathy had been on CAPD since July 2003. He experienced 1 episode of culture-negative CAPD peritonitis 3 months after commencement of CAPD. He was put on prophylactic topical gentamicin cream in November 2006. He presented with ESI in October 2007, and the culture grew *M. abscessus*. He was started on amikacin and clarithromycin, but the amikacin was discontinued after 4 weeks because of ototoxicity. The patient refused further antibiotic treatment or removal of Tenckhoff catheter despite persistent discharge from the exit site. He continued on CAPD and the exit site continued to grow the same atypical mycobacterial organism till he died 16 months later from peritonitis. The culture of the peritoneal effluent grew *M. abscessus*.

CASE 6

An 82-year-old woman with microscopic polyarteritis had been on CAPD since March 2006. She had recurrent urinary tract infection and was treated with multiple courses of ampicillin-clavulanic acid. She was started with prophylactic gentamicin cream in October 2006 and presented with an ESI 14 months later. Bacterial culture of the exit-site swab grew *M. abscessus*. She was treated with IP amikacin and oral clarithromycin for 8 weeks, followed by clarithromycin alone for 34 weeks more. She responded well to the treatment, and CAPD was continued without interruption.

CASE 7

A 62-year-old man with immunoglobulin A nephropathy had been on CAPD since 1999. He had history of

CAPD peritonitis attributable to *Acinetobacter* species in 2004 and *Moraxella* species in 2006. Prophylactic gentamicin cream was started in March 2006.

This patient presented with tunnel tract infection and ESI in 2008, and the exit-site swab grew *S. aureus* and *Corynebacterium jeikeium*. He was treated with vancomycin and rifampicin, but did not respond clinically. Four weeks after the initial presentation, a repeat exit-site swab grew *M. fortuitum*. The patient was treated with IP amikacin and oral clarithromycin for 2 weeks, followed by clarithromycin alone for 10 weeks, and finally with intravenous cefoxitin and clarithromycin for 6 weeks. The Tenckhoff catheter was removed on day 9 for refractory infection and was reinserted 16 months later.

CASE 8

A 45-year-old man with obstructive uropathy started on CAPD in 2009 and had started topical gentamicin cream as ESI prophylaxis 2 months after commencement of CAPD. He developed refractory peritonitis 1 month afterward. Effluent culture grew *M. abscessus*, and the Tenckhoff catheter was removed on day 18. The exit site was normal immediately after removal of the Tenckhoff catheter, but after a few days, was noted to discharge pus. The pus also grew *M. abscessus*. The patient was treated with 4 weeks of amikacin plus moxifloxacin, followed by moxifloxacin alone for 16 weeks more. The infection was controlled, and the patient was restarted on CAPD after a period of temporary hemodialysis for 9 months.

CASE 9

A 56-year-old man with diabetic nephropathy had been on CAPD since 2008. He also had a history of hypertension, hyperlipidemia, gout, and atrial fibrillation. He had 5 episodes of CAPD peritonitis (twice with *Klebsiella*, once each with *Strep. bovis* and *Strep. viridians*, and once culture-negative). He was started on gentamicin cream as ESI prophylaxis soon after commencement of CAPD. In August 2010, he had a tunnel tract infection presenting with erythema, swelling, and discharge of pus from the exit site. The exit-site swab culture later grew *M. abscessus*. He was treated with intravenous meropenem for 5 weeks, together with oral clarithromycin. His Tenckhoff catheter was removed, and he was permanently switched to hemodialysis.

DISCUSSION

Exit-site infection is one of the major catheter-related infective complications of PD (6). If left untreated,

ESI may evolve into peritonitis and catheter loss. The incidence of ESI ranges from 0.05 to 1.02 episodes per patient-year, because a uniform definition is lacking (7). On the other hand, *S. aureus* is consistently identified as the most common organism isolated in ESI.

To reduce the incidence of ESI, various methods have been developed. Systemic therapies include prophylactic oral trimethoprim-sulfamethoxazole, cyclic rifampin, and a combination of cyclical oral rifampin with daily mupirocin ointment applied to the exit site (8). However, the problem of antibiotic resistance and the adverse effects of systemic antibiotics preclude these preventive measures from becoming routine (8).

There is evidence that local application of mupirocin cream at the catheter exit site is an effective and well-tolerated method for prevention of *S. aureus* ESI (9). Because the relevant study did not prove that mupirocin cream is effective against other micro-organisms, broader-spectrum topical antibiotics such as gentamicin cream have been studied. Bernardini *et al.* (3) showed that exit-site gentamicin cream could reduce gram-negative catheter infections (including those with *P. aeruginosa*) and peritonitis (particularly with gram-negative organisms) by 35%. Gentamicin cream was also as effective as mupirocin cream in preventing *S. aureus* infections, an encouraging result. However, the use of gentamicin cream may not be without side effects. The present case series demonstrates a temporal relationship between use of gentamicin cream and the development of atypical mycobacterial infection.

Our center adopted routine use of topical gentamicin as ESI prophylaxis in 2006. However, only about 50% of patients received the cream solely because of patient preference. From 2006 to 2010, 332 patients used the gentamicin cream, and 331 patients did not. All 9 episodes of atypical mycobacterial ESI or peritonitis occurring during that time were associated with use of gentamicin cream. A Fisher exact test showed a statistically significant association between atypical mycobacterial infection and gentamicin use ($p = 0.0037$, Table 2, Figure 1). From 1996 to 2006, before the practice

TABLE 2
Contingency Table Analyzing the Association Between Topical Gentamicin Use and Presence of Atypical Mycobacterial Infection, 2006 – 2010

Topical gentamicin	Atypical mycobacterial infection	
	Present	Absent
Yes (<i>n</i> patients)	9	323
No (<i>n</i> patients)	0	331

Figure 1 — The incidence of atypical mycobacterial infection in patients on continuous ambulatory peritoneal dialysis using and not using topical gentamicin cream (1996 – 2010).

of gentamicin cream application, about 540 patients were on CAPD at our center, but only 1 documented case of atypical mycobacterial ESI occurred. No change had been made in our empiric treatment protocol, nor in the methodology for handling the microbiologic cultures during the study period. A similar finding was reported by Tse *et al.* (4) in 2007. In their case series, a cluster of 5 consecutive PD patients developed atypical mycobacterial ESIs; all the ESIs occurred after gentamicin prophylaxis was implemented.

Gentamicin cream is a wide-spectrum antibiotic preparation for topical administration. Susceptible bacteria include *Streptococcus*, *S. aureus* (both coagulase-positive and -negative), and gram-negative bacteria (*P. aeruginosa*, *Aerobacter aerogenes*, *E. coli*, *Proteus vulgaris*, and *K. pneumoniae*). The use of gentamicin cream is well known to permit overgrowth of non-susceptible microorganisms, of which atypical mycobacteria can be one. It has been hypothesized that selective pressure against other bacteria favors the growth of atypical mycobacteria and causes ESI in patients using topical gentamicin cream. The use of broad-spectrum antibiotics before episodes of atypical *Mycobacterium* ESIs may also be considered a source of selective pressure. However, the antibiotics were given more than 6 months before the ESI episodes in the 5 cases in our series. Although we cannot rule out the contribution of systemic antibiotics to the development of these atypical *Mycobacterium* infections, we believe the role of those antibiotics was not that important. In our case series, some of the gentamicin cream used by patients were sent for mycobacterial culture and yielded no growth, making the possibility of direct inoculation of the bacteria unlikely.

Nontuberculous, or atypical, mycobacteria (NTM) can be found in the natural environment—for example, in soil, dust, rivers, and streams (10). These organisms are even found in hemodialysis fluid, pharmaceutical

preparations, and disinfectant solution (11). They are classified into four groups based on their culture characteristics (12). Group I organisms are photochromogens that will produce pigment only when exposed to light. Examples include *M. kansasii* and *M. marinum*. Group II are scotochromogens, which will produce pigment in both light and dark condition. *M. rhodesiae* and *M. goodii* are some examples. Group III are non-chromogens, which do not produce pigments, and *M. avium* complex belongs to this group. Group IV are rapidly growing mycobacteria that can give rise to mature growth after a few days. Examples include *M. chelonae*, *M. abscessus*, and *M. fortuitum*. In the present study, confirmation of the subtype of atypical mycobacteria was obtained by polymerase chain reaction restriction fragment length polymorphism analysis of the *hsp65* gene. The sizes of the restriction fragments produced by the primer enzyme, *Sau96I*, and the second restriction enzyme, *CfoI*, were different for the various subspecies of atypical mycobacteria (13).

Peritonitis related to atypical mycobacteria is a rare event, accounting for fewer than 1% of all peritonitis episodes in Hong Kong (14). Most of the atypical mycobacterial ESIs or CAPD-related peritonitis episodes are caused by group IV organisms, and occasionally by group II organisms (15,16). Infections caused by the other groups are even rarer. Predisposing factors include infection with HIV, recurrent bacterial peritonitis with multiple courses of broad-spectrum antibiotics, poor residual renal function, and inadequate dialysis (17).

The usual clinical presentations of atypical mycobacterial infection are unresolved ESI and culture-negative ESI. The episodes are sometimes mistaken for diphtheroid ESI, because the atypical mycobacteria resemble gram-positive rods under light microscopy. However, the organisms can be readily differentiated by the bright red color they take on with Ziehl–Neelsen stain; diphtheroid organisms will not show that color.

Optimal therapy for atypical mycobacterial ESI is not yet defined. A prolonged course (in terms of months) of antibiotics is usually required. Dual antibiotics are usually given to prevent development of resistance with monotherapy. For *M. fortuitum*, a combination of oral quinolones (ciprofloxacin or levofloxacin), tetracyclines (doxycycline or minocycline), macrolides (clarithromycin or azithromycin), or trimethoprim–sulfamethoxazole can be given empirically while awaiting sensitivity results (18). For *M. chelonae*, clarithromycin is usually given in locales in which most *M. chelonae* are sensitive to that agent. Doxycycline or ciprofloxacin, with a sensitivity of around 25%, can be added. For *M. abscessus*, the empiric antibiotics of choice are clarithromycin plus either amikacin or high-dose cefoxitin.

In our series, all isolates were sensitive to amikacin and clarithromycin. Clarithromycin was not used in 2 cases (patients 3 and 8) because of adverse drug reactions. Most of the isolates were intermediately to fully sensitive to cefoxitin, but that drug has become unavailable in the local market for the past couple of years. Among the quinolones, levofloxacin was not impressively useful, but moxifloxacin was used in one case (8) in which the isolate was resistant to levofloxacin.

Antibiotics should be modified according to sensitivity tests. Removal of the Tenckhoff catheter is often required in refractory cases. Case reports in addition to ours have demonstrated successful treatment of atypical mycobacterial infections without the removal of the Tenckhoff catheter. Shaving of the external cuff was performed in some of the reported cases (19,20).

CONCLUSIONS

We report a series of atypical mycobacterial ESIs and peritonitis episodes after a variable duration of use of topical gentamicin cream. Gentamicin cream can be theoretically be considered a risk factor for this type of infection, given its ability to promote the growth of atypical mycobacteria by suppressing other microorganisms. Clinical studies are thus warranted to look for alternative prophylactic measures to alleviate the potential problems associated with the prolonged use of a single antibacterial agent. Optimal treatment for atypical mycobacterial ESI is not yet defined, but the prolonged use of dual appropriate antibiotics is usually required. Tenckhoff catheter removal is not mandatory, but might be required in refractory cases.

DISCLOSURES

The authors have no financial conflicts of interest to declare.

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