Two Cases of Necrotizing Fasciitis Due to *Acinetobacter baumannii*[†]

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Necrotizing fasciitis has conventionally been associated with the streptococci, and when it is caused by other organisms, it is most often the result of a polymicrobial infection. We report on two cases of fatal monomicrobial necrotizing fasciitis due to *Acinetobacter baumannii*, an unusual finding that may be an indication of enhanced virulence of the organism.

CASE REPORTS

Case 1. Patient X was a 21-year-old male with multiple medical problems, including end-stage renal disease secondary to systemic lupus erythematosus, thrombotic thrombocytopenic purpura, and mesenteric vasculitis. The patient had been treated with prolonged courses of systemic corticosteroids and intermittently with rituximab and had undergone multiple courses of treatment consisting of plasmapheresis, blood transfusions, and hemodialysis. In August 2007, he was admitted to the hospital for pulmonary edema and bright red blood from the rectum. At that time, blood cultures grew vancomycin-resistant *Enterococcus faecium* and *Candida albicans*. He was treated with daptomycin and fluconazole, and all blood cultures subsequently became negative. Days later, he tested positive for *Clostridium difficile*-associated colitis; therefore, metronidazole was added to his antibiotic regimen.

Despite embolization of the vessels of the terminal ileum, the patient continued to experience gastrointestinal bleeding. He therefore underwent an exploratory laparotomy, ileocecal resection, and ileostomy 3 weeks after admission. He appeared to be recovering until 5 days later, when he developed bacteremia caused by the gram-negative organisms *Klebsiella pneumoniae* and *Citrobacter freundii*, for which he was treated with imipenem and was continued on metronidazole.

Two weeks after the ileocectomy, at approximately 2 a.m., the patient noted pain along his left flank and thigh. Physical examination revealed faint erythema and tenderness. By 11 a.m. of the same day, there were marked increases in the levels of erythema, edema, and pain. The erythema extended beyond a marked line by greater than 2 cm in 1 h. It extended from flank to flank across the patient's back and thighs bilaterally. Bullae appeared on the skin over a similar distribution. An incision was performed at the bedside, and a frozen section revealed inflamed and necrotic fibroconnective tissue and muscle, with quantitative biopsy specimens revealing >100,000 gram-negative rods. The patient gave consent for and underwent an emergent debridement in the operating room for

suspected necrotizing fasciitis. Amikacin, vancomycin, and clindamycin were empirically added to his existing antibiotic regimen of imipenem and metronidazole, pending culture and susceptibility testing results.

During debridement, the necrotizing fasciitis was noted to be more extensive than was evident from the physical examination at the bedside. Necrotic tissue extended along the anterior abdominal wall (along the dehiscence surgical wound) and both flanks, cephalad to the scapula, and down to the knees bilaterally. Although it was eviscerated, the small bowel was viable. The flanks revealed thrombosed vessels and dead fat. The infection appeared to spare the penis and scrotum. Approximately 40% of the patient's total body surface area ultimately had to be debrided during the procedure. The tissue was debrided to an area where the margins appeared grossly uninfected and viable, and a second-look procedure was planned for the following day. Unfortunately, all of the tissue margins demonstrated gram-negative rods on Gram stain, and the patient was therefore taken back to the operating room the next day for further debridement. At that time, the infection extended to the perineum, lower extremities, deep intercostal muscles, and entire abdominal wall (Fig. 1a and b). Bilateral hip disarticulations were considered to help control the infection. However, the extent of disease was severe, and the patient was coagulopathic, requiring multiple units of blood and vasoactive agents for hemodynamic stability. Therefore, following extensive discussion with the family, the decision was made not to pursue further surgical debridement and to return the patient to the intensive care unit for continued hemodynamic resuscitation. At this point, the patient died, despite cardiopulmonary resuscitation efforts. He died within 36 h of first noting the pain in his thigh.

Numerous antemortem cultures of specimens of blood and muscle tissue (from the legs and intercostal muscle tissue) grew *Acinetobacter baumannii*. Cultures of blood of the patient had continued to grow *Klebsiella pneumoniae* and *Citrobacter freundii*, despite treatment; however, *A. baumannii* was the sole pathogen recovered from all tissue specimens submitted. The *A. baumannii* blood isolate was resistant to all antimicrobial agents tested (amikacin, ampicillin-sulbactam, ceftazidime, ceftriaxone, ciprofloxacin, gentamicin, imipenem, piperacillin-tazobactam, tobramycin, and trimethoprim-sulfamethoxazole), with zone sizes greater than 6 mm by disk

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FIG. 1. (a) Necrotic muscle tissue of patient X the day after the initial debridement; (b) grossly appearing viable muscle tissue of patient X following the second debridement; however, closer examination revealed extensive soft tissue infection that tracked to the bone.

diffusion. The wound isolates displayed resistance to all antimicrobials (with zone sizes greater than 6 mm) except amikacin (zone size, 26 mm), and they demonstrated intermediate susceptibility to tobramycin (zone size, 13 mm).

Case 2. Patient Y was a 47-year-old woman with a history of human immunodeficiency virus infection and end-stage renal disease who had undergone an exploratory laparotomy for ovarian torsion and the subsequent lysis of adhesions. She left the hospital against medical advice and was readmitted within 1 day with a diagnosis of surgical wound cellulitis. A culture of

her wound specimens grew coagulase-negative staphylococci. She was treated with amoxicillin-clavulanate. However, susceptibility testing showed the organism to be methicillin resistant, and the patient developed a pelvic abscess within 1 week. She was empirically treated with piperacillin-tazobactam, vancomycin, and clindamycin, as well as with percutaneous abdominal drainage. She did not improve; therefore, amikacin was added to her treatment regimen. During this time, culture and susceptibility testing results were pending, because the organism isolated from the blood was not identified by standard meth-

TABLE 1. Blood culture results and susceptibility testing profile of the isolates from patient Y determined by a reference laboratory

Antimicrobial tested	MIC ($\mu\text{g/ml}$) ^a		
	<i>Acinetobacter</i> strains 1 and 2	<i>Acinetobacter</i> strain 1	<i>Acinetobacter</i> strain 2
Amikacin	>32 (R)		
Ceftazidime	>16 (R)		
Gentamicin	>8 (R)		
TMP-SMX ^b	>2/38 (R)		
Imipenem	>2 (R)		
Ciprofloxacin	>2 (R)		
Piperacillin-tazobactam	>64/4 (R)		
Ticarcillin-clavulanate	>64/2 (R)		
Meropenem	>8 (R)		
Levofloxacin	>4 (R)		
Cefepime		16 (I)	>16 (R)
Tigecycline ^c		1	0.5
Colistin ^c		Not performed	≤ 2

^a The letters in parentheses indicate the susceptibilities of the strains: I, intermediate; R, resistant.

^b TMP-SMX, trimethoprim-sulfamethoxazole.

^c The susceptibility profile was not interpretable.

odologies and was submitted to a reference laboratory for identification (by DNA sequencing) (Table 1). Meanwhile, the patient underwent an exploratory laparotomy for resection of the abscess. The next day, she developed blisters on her right thigh, and the biopsy specimens revealed bacteria in the dermis and underlying fat, consistent with cellulitis caused by a gram-negative organism. During this time, her blood cultures showed the growth of *A. baumannii* isolates that displayed

sensitivity only to ampicillin-sulbactam. The patient's infection worsened, and she underwent excisional debridement of the wounds on her right lower extremity. The biopsy specimens from her right thigh and buttock revealed skin and soft tissue with bacteria, inflammation, and necrosis, consistent with necrotizing fasciitis. The sole organism isolated from the debrided tissue was *A. baumannii*, which, again, was sensitive only to ampicillin-sulbactam (Table 2). Within six days of debridement, the strains of *A. baumannii* growing from the patient's surgical wound and abdominal fluid exhibited resistance to all antimicrobial agents tested (blood cultures by this point showed no growth of the organism). Specimens of the patient's blood and Jackson-Pratt (JP) drain later grew *Pseudomonas aeruginosa*; however, as was the case for patient X, the cultures of her wound specimens grew *A. baumannii* as the sole pathogen. During this time, colistin was added to her treatment regimen, but the patient's wound never improved. She developed severe septic shock and died 18 days after the blisters were first noted on her thigh.

Acinetobacter baumannii is an increasingly prevalent and significant pathogen. It is often associated with immunocompromised and hospitalized patients. It has been described in cases of sepsis, wound infections, and pneumonia in these populations (1, 3, 4, 8, 13, 15). Although it is rare, *A. baumannii* has been described to be a cause of community-acquired bacteremic cellulitis and community-acquired pneumonia in previously healthy people as well (3, 4). Specifically, wound infections due to *A. baumannii* have been highlighted in association

TABLE 2. Culture results and antimicrobial susceptibility profiles for the isolates from patient Y obtained beginning on the day of excisional debridement

Date (mo/day/yr)	Site	Organism identified	Susceptibility result ^a
07/16/06	Right anterior thigh	<i>A. baumannii</i>	SAM, S (15); R to rest of panel (≥ 6)
07/22/06	Abdominal fluid	<i>A. baumannii</i> strain 1	Panresistant (≥ 6)
	Abdominal fluid	<i>A. baumannii</i> strain 2	Panresistant (≥ 6)
	Blood	NG ^b	
07/23/06	Abdominal wound and JP drain	<i>A. baumannii</i> strain 1	TOB, I (13); R to rest of panel (≥ 6)
	Abdominal wound and JP drain	<i>A. baumannii</i> strain 2	TOB, I (13); R to rest of panel (≥ 6)
	Blood	NG	
07/24/06	JP drain	<i>Pseudomonas aeruginosa</i>	ATM, I (19); CIP, LVX, R (6); S to rest of panel (≥ 16)
	Blood	NG	
07/30/06	JP drain	<i>Pseudomonas aeruginosa</i>	ATM (16); GEN (8), I; CIP (≥ 4); LVX (≥ 8), R; S to rest of panel (≤ 1 to 16, depending on antimicrobial)
	JP drain	<i>A. baumannii</i>	Pan-Resistant (≥ 6)
	Blood	<i>Pseudomonas aeruginosa</i>	ATM (16); GEN (8); AMK (32), I; CIP (≥ 4); LVX (≥ 8), R; S to rest of panel (≤ 1 to 8, depending on antimicrobial)
07/31/06	Blood	<i>Pseudomonas aeruginosa</i>	ATM (16), I; CIP (≥ 4); GEN (≥ 16), R; S to rest of panel (≤ 1 to 16, depending on antimicrobial)

^a The *A. baumannii* antimicrobial susceptibility test panel (by disk diffusion) consisted of amikacin (AMK), ampicillin-sulbactam (SAM), ceftazidime (CAZ), ceftriaxone (CRO), ciprofloxacin (CIP), gentamicin (GEN), imipenem (IPM), piperacillin-tazobactam (TZP), tobramycin (TOB), and trimethoprim-sulfamethoxazole (SXT). Zone sizes (in mm) are given in parentheses. The *Pseudomonas aeruginosa* antimicrobial susceptibility test panel (MICs [in $\mu\text{g/ml}$] were determined by use of the Vitek 2 system) consisted of amikacin, aztreonam (ATM), ceftazidime, ciprofloxacin, gentamicin, imipenem, levofloxacin (LVX), piperacillin-tazobactam, and tobramycin. S, susceptible; I, intermediate; R, resistant.

^b NG, no growth.

with severe trauma, as in cases of automobile accidents (2) and in American soldiers wounded in Iraq and Afghanistan (1).

A. baumannii has been reported in cases of polymicrobial necrotizing fasciitis (12). To our knowledge, there is only one published report of *A. baumannii* as the sole pathogen causing necrotizing fasciitis in a human. The retrospective review by Liu et al. (12) reported *A. baumannii* as a monomicrobial cause of necrotizing fasciitis in 2 of 87 cases. However, the same study found a single pathogen as the causative agent of necrotizing fasciitis in 67.8% of the cases studied, a finding that is inconsistent with those of most other studies, which have reported a polymicrobial cause in the majority of cases (6, 8, 10, 11, 19). Liu et al. acknowledged that their high rate of monomicrobial isolation might have been due to “relatively unsophisticated techniques for collection, transfer, or culture of anaerobic specimens,” but they did not explain this further (12). For the two cases reported here, specimens were collected using sterile techniques. By using known dilutions of the homogenized tissue in saline solution, the number of organisms was calculated per gram of tissue and was found to be less than 100,000, if no organisms were seen, or greater than 100,000, if any organisms were seen on the direct smear.

Routine procedures include rigorous attempts to isolate all potential organisms. Specimens are inoculated onto aerobic and anaerobic media. The colonies are checked for growth on a daily basis, and the growth of organisms is then checked phenotypically and, if indicated, by automated methods for a final identification. For both of the cases reported here, tissue specimens grew only one organism; this organism was identified by Gram stain as well as by growth on MacConkey agar medium as a gram-negative organism. Its coccobacillary phenotype, growth as small purple colonies on MacConkey agar, and negative oxidase reaction were suspicious for the presence of *Acinetobacter* species. Use of the Vitek 2 automated system (bioMérieux, Marcy l'Étoile, France) with the AST-GN 13 gram-negative card (bioMérieux) validated for use with the instrument identified the isolates as representing *Acinetobacter* species in our reported cases. Disk diffusion was used to determine the organism's susceptibility profile, according to Clinical and Laboratory Standards Institute (CLSI) guidelines.

Most studies find necrotizing fasciitis to be polymicrobial in approximately 70% of cases, with the most common microorganisms identified as being group A and B streptococci, the staphylococci, members of the family *Enterobacteriaceae*, and *Pseudomonas aeruginosa* (6, 8, 10, 11, 19). Necrotizing fasciitis is therefore, no longer known as “acute streptococcal gangrene,” the term originally used to describe the syndrome by Meleney in 1924 (14).

A. baumannii is becoming progressively more resistant to even the most-broad-spectrum antibiotics, and physicians are faced with limited therapeutic options (7, 17, 20). Polymyxins are increasingly being used as a last-resort treatment in infections caused by multidrug-resistant (MDR) *Acinetobacter baumannii* strains, which are an increasingly prevalent problem. MDR *A. baumannii* strains have been defined as *A. baumannii* strains resistant to three or more representatives of the following classes of antibiotics: fluoroquinolones (ciprofloxacin), extended-spectrum cephalosporins (ceftazidime and cefepime), β -lactam- β -lactamase inhibitor combinations (ampicillin-sul-

bactam), aminoglycosides (amikacin and tobramycin), and carbapenems (imipenem and meropenem) (7). It has been postulated that resistance to carbapenems may be, in itself, sufficient to define an isolate of *A. baumannii* as highly resistant (17). Success with the treatment of such cases with combined antimicrobial therapy has been reported, with rifampin (rifampicin) and colistin providing promising synergy in clinical experience (16). Tigecycline has also been shown to have good in vitro activity against *A. baumannii* (16). Unfortunately, there are currently no CLSI-approved breakpoints for the interpretation of the MICs for tigecycline or rifampin for *Acinetobacter* species, and laboratories have only recently begun to report susceptibility results for colistin. Furthermore, at the time of the two cases reported here, testing for susceptibility to colistin, tigecycline, and rifampin was not performed in our laboratory and there were no CLSI-published interpretative guidelines for the use of these drugs to treat infections caused by *Acinetobacter* strains. We now test our *Acinetobacter* isolates for susceptibility to colistin and tigecycline. Finally, resistance to the polymyxins has been reported, and there have even been reports of heteroresistance among strains of *A. baumannii* (16).

A number of naturally occurring and acquired virulence factors conferring antimicrobial resistance have been linked to MDR *A. baumannii* (7, 17). Hujer et al. studied a large number of MDR *A. baumannii* isolates from civilian and military patients and detected 16 unique resistance genes or gene families and four mobile genetic elements (7). In addition, that group found the first reported β -lactamase genes, *bla*_{OXA-58}-like and *bla*_{PER}-like genes, in U.S. MDR *A. baumannii* isolates, highlighting the complex genetic background of this organism. In their analysis, only 11 isolates (15%) were resistant to all five classes of antibiotics to which the isolates were tested for resistance, with 89% of the isolates being resistant to at least three classes of antibiotics (7). Seventeen of 19 isolates (90%) that were imipenem and/or meropenem resistant by disk diffusion possessed evidence for the presence of a *bla*_{OXA-23}-like or a *bla*_{OXA-58}-like gene (7). Finally, whole-genome sequencing of a resistant *A. baumannii* strain showed that *A. baumannii* was able to acquire new genetic material under antibiotic pressure (5).

Testing for virulence factors has primarily focused on antimicrobial resistance; however, virulence factors conferring increased pathogenicity are largely unknown. For example, it is known that representatives of *A. baylyi* have an extraordinary capacity to acquire foreign DNA; however, it is unknown how pervasive natural competence is among other *Acinetobacter* spp. (18). Genetic dynamism would confer a selective advantage over other organisms with more static genomes and might contribute to the virulence of *A. baumannii*. In addition, *A. baumannii* is thought to devote a considerable portion of its genome to pathogenesis, and Perez and colleagues (16) found that a large number of the genes in *A. baumannii* contain virulence islands. Unfortunately, little else is known about the organism's virulence factors as they relate to pathogenicity. Testing for virulence factors is an area of increasingly important research and will shed light on the means for differentiation of colonization from infection; the establishment of clonality in cases of outbreaks; the identification of environmental sources; and the

elimination of this very hardy, naturally competent, and ubiquitous organism (13).

The two patients described in this report were immunocompromised and had open portals of entry (lines, surgical wounds) for infection. Thus, one could argue that infection with any health care-associated pathogen, including *A. baumannii*, was not altogether unexpected (7). *A. baumannii* is ubiquitous and has been found in the sources of water used to clean wounds (13). In fact, *Acinetobacter* spp. are reportedly the most common gram-negative bacteria to colonize the skin of hospital personnel (1, 9). What was most unusual about the cases discussed here was the fact that *A. baumannii* was the sole organism isolated from the necrotic tissue in both patients and was isolated in multiple sets of cultures. Furthermore, even among immunocompromised patients, the pathogen does not usually cause necrotizing fasciitis but, rather, causes bloodstream, lung, or (milder) skin and soft tissue infections. As in our institution, there are immunocompromised patients in many academic and community centers around the United States, and yet, to our knowledge, the entity that we describe has not previously been reported. When *Acinetobacter* species have been cited as a cause of skin and soft tissue infections, it has been among returning soldiers and such citations are not associated with a great degree of mortality. In our cases, despite aggressive and timely therapy, mortality ensued. This is a critical distinction in our patients. Furthermore, the characteristics of the organisms and not just those of the patients alone could account for the differences between our patients and returning soldiers—it is possible that the *Acinetobacter* isolates from these patients may have had characteristics different from those of the isolates from soldiers returning from Iraq.

Finally, the fact that we had two patients displaying this distinctive presentation of necrotizing fasciitis within 1 year of each other may indicate the emergence of an *A. baumannii* strain with new virulence capabilities. An enhanced virulence capacity of the organism may also explain the rapid spread of *A. baumannii* in case 1. During debridement, an area of tissue that appeared grossly to be viable became necrotic within hours. We speculate that perhaps the acquisition of a virulence factor from other pathogens that the patients harbored, as well as their immunocompromised state, may have played a role in the rapidity and the severity of the disease. Unfortunately, it was not possible to demonstrate the possible relatedness between the isolates, as the first case was brought to the attention of the authors when the second case, which occurred 12 months later, was investigated. It is our hope that future molecular testing of *A. baumannii* strains will reveal virulence factors that may account for these extremely rapidly spreading strains of the organism.

In conclusion, we report on two cases of necrotizing fasciitis from which *A. baumannii* was isolated as the sole pathogen. As infection with MDR *A. baumannii* can be rapidly fatal in immunocompromised patients, as illustrated by our cases, we stress the importance of immediate reporting of suspected *A. baumannii* isolates in cases of necrotizing fasciitis. Physicians should be aware that this pathogen can trigger a novel clinical course and that these resistant organisms can be remarkably virulent, producing rapidly advancing lesions. At present, even experienced clinicians would not suspect *Acinetobacter* spp. as the primary etiologic

agent in cases of necrotizing fasciitis. They suspect the streptococci, gram-negative rods (other than *Acinetobacter* species), and mixed infections. As *Acinetobacter* species can cause severe disease in patients, timely routine polymicrobial antimicrobial coverage with prompt debridement in cases of necrotizing fasciitis may not be adequate. It is important for physicians to know the severity of disease that *Acinetobacter* species can cause so that they may consider empirical therapy with colistin (or some alternate drug[s] to combat MDR *A. baumannii*) quickly, especially if a gram-negative or a gram-variable (cocco)bacillus is the preliminary finding reported by the laboratory. Finally, in cases of necrotizing fasciitis, it is imperative that laboratories specify that the Gram stain result may indicate an organism that resembles an *Acinetobacter* species, as there may be no time to wait for final culture results. The laboratory can do this by reporting the presumptive Gram stain findings as specifically as possible, using terms like “pleiomorphic, gram-variable, or gram-negative coccobacillus, suspicious for *A. baumannii*” instead of “gram-negative (cocco)bacillus.”

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