Multidrug-Resistant Acinetobacter Extremity Infections in Soldiers

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War wound infection and osteomyelitis caused by multidrug-resistant (MDR) *Acinetobacter* species have been prevalent during the 2003–2005 military operations in Iraq. Twenty-three soldiers wounded in Iraq and subsequently admitted to our facility from March 2003 to May 2004 had wound cultures positive for *Acinetobacter baumannii* complex. Eighteen had osteomyelitis, 2 burn infection, and 3 deep wound infection. Primary therapy for these infections was directed antimicrobial agents for an average of 6 weeks. All soldiers initially improved, regardless of the specific type of therapy. Patients were followed up to 23 months after completing therapy, and none had recurrent infection with *Acinetobacter* species. Despite the drug resistance that infecting organisms demonstrated in this series, a regimen of carefully selected extended antimicrobial-drug therapy appears effective for osteomyelitis caused by MDR *Acinetobacter* spp.

Casualty statistics from the 2003–2005 military operations in Iraq show an increase in the ratio of wounded to fatal casualties compared to previous operations in the Persian Gulf, Vietnam, and Korea (1). This relative increase of wounded casualties has led to an increased incidence of war wound infection and osteomyelitis, especially caused by multidrug-resistant (MDR) *Acinetobacter* species. The incidence of bacteremia at military medical facilities caused by *Acinetobacter baumannii* has also increased (2). The current incidence of infection with *Acinetobacter* should not be surprising. These organisms were the most frequently recovered gram-negative isolate from war wounds and the second most frequent bacterium causing bloodstream infection in US Marines with extremity wounds during the Vietnam War (3). In nonconflict environments, *Acinetobacter* species are rarely responsible for community-acquired infections. In the hospital setting, *Acinetobacter* species are an important cause of nosocomial infection, yet these infections were rarely encountered in our facility until we began observing them in soldiers with infected wounds. Nosocomial infections caused by *Acinetobacter* species include pneumonia, meningitis, bloodstream, urinary tract, surgical wound, and soft tissue infections (4). Such infections are challenging to treat because of extensive antimicrobial drug resistance. Osteomyelitis caused by *Acinetobacter* occurs, but it is less frequently reported and had not been identified in our facility during the 14 months before March 2003. Optimal therapy for osteomyelitis caused by these organisms is not well defined because of limited available data. This case series reviews 1 military medical center’s experience with these infections, including species identified, antimicrobial drug–susceptibility patterns, antimicrobial drug therapy, and clinical outcomes.

Methods

Case reports were compiled from active-duty soldiers admitted to Brooke Army Medical Center (BAMC) in San Antonio, Texas. This tertiary military medical center serves a population of active-duty and retired soldiers and their dependents along with a limited number of civilian trauma patients admitted from the local area. The hospital was operating at an average capacity of 175 beds during the study period. This facility also houses the US Army’s Institute of Surgical Research, which treats both active-duty and civilian trauma patients with burn injuries. Data collection for this case series was completed under a study protocol approved by BAMC’s Department of Clinical Investigation Institutional Review Board.

Identification of Patients

All wound, sputum, urine, and blood culture results completed at our hospital from March 1, 2003, to May 31, 2004, were reviewed. Those patients who had *Acinetobacter*-positive cultures were then compared to all...
active-duty soldiers admitted to our facility. A soldier was
considered for inclusion if he had an Acinetobacter-posi-
tive culture and had been deployed to Iraq or Afghanistan
and had an admission diagnosis of injury (ICD codes
800.0–900.0). Similarly, hospital admission and laboratory
data were reviewed for the 14 months before the study
period to define the incidence of Acinetobacter infection in
hospitalized, active-duty soldiers before the onset of mili-
tary action in Iraq.

Case Definitions

Patients with either Acinetobacter contiguous focus
osteomyelitis or wound infection are included in this series.
Cases were defined as osteomyelitis if bone tissue collect-
ed during surgical procedures (primarily open debride-
ments but also including placement of external or internal
fixators or bone grafting) was positive for Acinetobacter
spp. on routine culture (5,6). In addition, patients with open
fractures or exposed bone with gross findings of infection
(purulence, necrotic tissue, or environmental contamination
with exposed bone), clinical evidence of infection (temper-
ature >38°C, leukocyte count >12,000/µL), and Acinetobacter
spp. identified from culture of deep wound tissue obtained intraoperatively, excluding bone, were also
defined as having osteomyelitis (7). Cases were defined as
wound infection if similar deep wound cultures were posi-
tive for Acinetobacter spp. with gross findings and clinical
evidence of infection but no exposed bone and no fracture.
Colonization with Acinetobacter was defined as a positive
culture for Acinetobacter without gross findings or clinical
evidence for infection.

The Acinetobacter isolate was defined as MDR if it was
resistant to ≥3 classes of antimicrobial agents as tested by
automated antimicrobial drug–susceptibility testing (Vitek,
bioMérieux, Hazelwood, MO, USA) (8). On occasion, iso-
lates were further evaluated with disk diffusion antimicro-
bial testing for susceptibilities to alternate antimicrobial
drugs, such as colistin, or to confirm automated suscepti-
bility results. Confirmatory disk diffusion susceptibility
testing was completed only for those isolates that were
resistant to all antimicrobial agents by automated testing or
if only 1 antimicrobial drug was listed as susceptible. Disk
diffusion testing was performed in accordance with
Clinical and Laboratory Standards Institute (formerly
NCCLS) guidelines (9). Colistin susceptibility was
assumed if the zone of inhibition was ≥14 mm (10).

Patients were evaluated for recurrence of infection. Many patients underwent subsequent reconstructive sur-
geries, and the bone tissue was sent for culture. Definitions
of recurrent infection followed the previously described
criteria for the case definitions with the following addi-
tions: recurrent infection was defined as having Acinetobacter
spp. isolated at the original site of infection
after completing an antimicrobial drug treatment course
for the initial infection; secondary infection was defined as
infection with a different organism at the same site as the
initial Acinetobacter infection.

Data Collection

Both electronic and paper charts of all patients who met
case definition criteria were retrospectively reviewed for
demographic, diagnostic, and treatment data. Laboratory
results were reviewed for Acinetobacter species isolated
and antimicrobial drug susceptibilities. Patients were also
interviewed either in person or by telephone to confirm
mechanism of injury, length of antimicrobial drug treat-
ment course, recurrence of infection, subsequent hospital
admissions, and clinical outcome of the sustained injury
and infection (resolved, continuing convalescence, or
amputation). Follow-up was defined as the time from com-
pleting the initial antimicrobial treatment course to the date
of the study interview.

Results

Case Inclusion Criteria

From March 1, 2003, to May 31, 2004, a total of 24,114
cultures (blood, urine, wound, sputum) were completed in
our hospital. Of these, 145 (0.6%) were positive for
Acinetobacter spp. During the same period, 237 active-
duty patients were admitted to our facility with the admis-
sion diagnosis of injury (Figure). Of these admitted
soldiers, 151 (64%) had been deployed to OIF/OEF. Cultures of blood, wound, sputum, urine, or skin were
obtained for 84 of these patients; 48 (32% of admitted
deployed soldiers) were positive for Acinetobacter spp. Of
these, 30 (63%) represented clinical infection; the remain-
ing 18 represented colonization with Acinetobacter. Of
those patients with cultures that represented clinical infec-
tion, 23 met the case definition for Acinetobacter
osteomyelitis (Table 1) or Acinetobacter wound infection
(Table 2). During the 14 months before the study period,
only 2 active-duty soldiers, of 326 admitted to our facility,
had any Acinetobacter infection. The incidence of
Acinetobacter infection during the study period represents
a significant increase when compared to the control period
(p < 0.01 by 2-tailed Fisher exact test).

Demographics

All patients included in this series had been transferred
to BAMC through the military airmobile medical evacua-
tion system. All, excluding one, were evacuated through,
and admitted for at least 1 day to, Landstuhl Army Medical
Center in Landstuhl, Germany; 3 patients were admitted to
a second US Army medical center before admission to
BAMC. The median time from injury to admission at
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Multidrug-resistant Acinetobacter Infections
BAMC was 6 days (range 2–36 days, Table 3). The median time from injury to identification of infection was also 6 days (range 3–12 days).

Acinetobacter infection was initially identified at BAMC in 15 of the 23 patients; the remainder were identified at a previous medical center. None were initially diagnosed prior to evacuation from Iraq or Afghanistan. The median age of the patients was 26 years (range 20–48), and all but 2 were men. Patients were generally stable on admission to BAMC and did not require admission to an intensive care unit.

Microbiologic Data

Patients with Acinetobacter osteomyelitis primarily had bone tissue collected during surgical procedures that was culture-positive for A. calcoaceticus-baumannii complex. This was the only species and organism identified in all initial tissue cultures. Ten patients had deep wound cultures, excluding bone tissue, that were positive for A. calcoaceticus-baumannii complex. Five (patient numbers 4, 9, 10, 11, and 15, Table 1) had open fractures with environmental contamination and signs of infection that met the case definition of osteomyelitis. The remaining 5 (Table 2) did not meet criteria for diagnosis of osteomyelitis and were diagnosed with wound infection. Two of these patients had burn injuries. Cultures of debrided soft tissue in these 2 patients were positive for Acinetobacter within the first 8 days of hospitalization, and pathologic evaluation of tissue demonstrated invasive infection. Patient no. 22 had a soft tissue wound culture positive on hospital day 5 (postinjury day 9); patient no. 23 had a soft tissue wound culture positive on hospital day 8 (postinjury day 10).

Antimicrobial Drug–susceptibility Data

Thirty-eight cultures from the 23 patients reported in this study were positive for Acinetobacter spp. (Table 4). Twenty-nine isolates were MDR, as tested by automated susceptibility testing. All but 4 of the MDR isolates were susceptible to imipenem, and no imipenem resistance developed in the 15 patients who received this drug during therapy. Three of these 4 isolates were susceptible only to amikacin. Of the 25 imipenem-susceptible MDR Acinetobacter isolates, 10 demonstrated resistance to all other tested antimicrobial agents. Other isolates were susceptible to only 1 other antimicrobial agent: 7 were also susceptible to amikacin, 3 to ampicillin/sulbactam, 2 to tobramycin, and 1 to trimethoprim/sulfamethoxazole. Nine isolates were not MDR. These isolates were susceptible to ≥3 classes of the tested antimicrobial agents. Three MDR isolates were tested for susceptibility to colistin; all 3 were susceptible by disk diffusion testing. One was susceptible only to imipenem, 1 to amikacin alone, and 1 to both amikacin and ceftazidime.

Therapy

Antimicrobial drug treatment of these infections was based on susceptibility testing, and all patients with osteomyelitis underwent multiple surgical debridements of necrotic bone. Ten of the patients with osteomyelitis were treated with dual antimicrobial agents, 7 with monotherapy, and 1 with surgical debridement alone. Only patients with osteomyelitis received dual antimicrobial drug therapy. Of the 10 treated with dual therapy, 5 had MDR Acinetobacter spp. and 5 had non-MDR Acinetobacter spp. isolated. The primary combination of antimicrobial agents was imipenem (500 mg every 6 h) in combination with high-dose amikacin (15–20 mg/kg daily). In a few instances, when imipenem was not active against the isolated organism, ampicillin/sulbactam or ceftazidime was used if either was active against the particular isolate (Table 1). Of the 7 treated with monotherapy, 5 had MDR Acinetobacter isolated. All patients with wound infection received monotherapy based on antimicrobial drug–susceptibility testing results.

Follow-up

The follow-up period was 1–23 months (mean 9 months). During this time, no Acinetobacter infections recurred at any site, including the bloodstream. Seven secondary infections occurred, 6 in those with an initial diagnosis of osteomyelitis and 1 with wound infection. Four occurred in patients with MDR Acinetobacter (3 with osteomyelitis and 1 with wound infection). These secondary infections primarily involved other resistant nosocomial pathogens (see expanded online Tables 1 and 2, available at http://www.cdc.gov/ncidod/eid/vol11no08/05-0103.htm).
During the 14 months before March 2003, only 2 active-duty soldiers had Acinetobacter infection. A soft tissue infection with Acinetobacter developed in 1 soldier with a history of bullous pemphigoid. Bacteremia with Acinetobacter developed in the other soldier, who had a history of Ewing sarcoma. The latter Acinetobacter isolate was not a MDR organism and was treated with imipenem (500 mg parenterally) for 14 days.

**Discussion**

The 23 cases observed during the study period represent a significant increase in the incidence of clinical infection with Acinetobacter in our facility. Similarly, the rate of blood, wound, or urine cultures positive for Acinetobacter species increased 3-fold during the study period as compared to the control time period (data not shown). This increase and the influx of severe extremity infection due to MDR Acinetobacter species posed considerable challenges. The foremost was determining appropriate therapy for osteomyelitis caused by MDR Acinetobacter species without institutional or historical experience to guide us. In addition, increasing prevalence of this MDR gram-negative organism in our facility mandated new infection control procedures to limit nosocomial spread. Finally, the occurrence of Acinetobacter wound infection was somewhat unexpected, and initially the reservoir for infection was unclear and generated much debate. Recent investigation by the military medical and research community suggests that these are nosocomial infections; however, their exact source remains unclear.

Most Acinetobacter infections reported in the literature reflect nosocomial Acinetobacter, as hospitalized patients
are at increased risk because of severe illness or disability, extremes of age, and relative states of immunocompromise (4). *Acinetobacter* species can cause infection in any organ system, including bacteremia, pneumonia, endocarditis, meningitis, urinary tract infection, intraabdominal abscess, osteomyelitis, soft tissue infection, and surgical site infections (11). Data collected from a review of sentinel hospitals in the United States demonstrated that 1.5% of all nosocomial bloodstream infections were due to *Acinetobacter* species (12). Crude death rates associated with nosocomial *Acinetobacter* infection are 19%–54% (12–15). The difficulty in treating these infections is not due to any excessive virulence of the organism per se but rather to its antimicrobial drug resistance. Many nosocomial isolates are resistant to ≥3 classes of antimicrobial agents, which classifies them as MDR organisms (8,12). A common susceptibility pattern in this case series was resistance to all antimicrobial agents except imipenem and amikacin.

When these patients were first evaluated, data to guide therapeutic decisions were limited. Previous reported experience with osteomyelitis caused by *Acinetobacter* species is scant. It has been described after a hamster bite in an 8-year-old boy (16) and in a patient who previously had an artillery fragment injury that caused an open fracture of the right femur (17). Other reviews have described osteomyelitis as a sequela of infection with *Acinetobacter* species but did not report details of therapy or follow up (4,11). Patients in our case series primarily received extended dual antimicrobial–drug therapy based on

### Table 2. *Acinetobacter* wound infection*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Wound infection location</th>
<th>Mechanism of injury</th>
<th>MDR† isolate</th>
<th>Bacteremia</th>
<th>Parenteral drug therapy</th>
<th>Follow-up, wk‡</th>
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<td>Left thigh wound</td>
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<td>Scalp wound</td>
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<td>Imipenem 500 mg every 6 h for 14 d</td>
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†MDR, multidrug-resistant; RPG, rocket-propelled grenade.
‡Length of follow up after completion of antimicrobial drug therapy.

### Table 3. Patient demographics*

<table>
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<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Time (d) from injury to BAMC admission</th>
<th>Diagnosis of infection</th>
<th>No. MC admissions before BAMC admission</th>
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*BAMC, Brooke Army Medical Center; MC, medical center; N, no; Y, yes; NA, not available.*
susceptibility patterns of the recovered organisms. Combination therapy has been shown to decrease the risk for development of more highly resistant organisms, which has been reported when single agents are used alone (18). While on this antimicrobial regimen, patients demonstrated clinical improvement with marked reduction of inflammatory markers. Many of these patients had internal stabilizing hardware placed into the infected area at the time of diagnosis of infection. This hardware remained in place at the completion of parenteral therapy. In these situations, when the causative organism was susceptible to oral antimicrobial agents, oral suppressive therapy was continued as long as the stabilizing hardware remained in place. In most cases, however, because of extended antimicrobial drug resistance, no oral agents maintained activity at the completion of parenteral therapy. In these situations, when the causative organism was susceptible to oral antimicrobial agents, oral suppressive therapy was continued as long as the stabilizing hardware remained in place. In most cases, however, because of extended antimicrobial drug resistance, no oral agents maintained activity against the Acinetobacter isolate. Once those infected with MDR isolates demonstrated clinical improvement and normalization of inflammatory markers, antimicrobial drug therapy was discontinued without continuing long-term suppressive therapy (see expanded online Tables 1 and 2, available at http://www.cdc.gov/ncidod/eid/vol11no08/05-0103.htm).

During the follow-up period, no recurrent episodes of Acinetobacter osteomyelitis have occurred. The relative brevity of follow-up is a limitation of this study. The ultimate outcome for these patients will not be known for many years, as they have increased risk for recurrent infection throughout their lifetime. In addition, Acinetobacter organisms do not possess substantial inherent virulence. None of the patients in this series failed therapy, and none died because of Acinetobacter infection. Such is not the case in outbreaks among immunocompromised or intensive care patients, in whom Acinetobacter infection leads to increased mortality (12–15). The successful outcomes in this case series may be a reflection of the youth and general good health of the soldiers infected.

MDR Acinetobacter is an important nosocomial pathogen with multiple recent outbreaks reported (18–22). It has the capacity to survive in dry environments (23,24), which increases the risk for nosocomial transmission. The increasing prevalence of MDR Acinetobacter in our facility led to new infection control procedures. Currently, all injured soldiers admitted to our facility returning from OIF/OEF are placed in contact isolation. Screening cultures of the axilla, groin, and any open wound are completed to assess for colonization with MDR Acinetobacter, which was identified in 18 of 151 admitted soldiers during the study period (Figure). If all cultures taken on admission are negative, the soldier is then removed from contact isolation. Soldiers with wound infection or osteomyelitis caused by MDR Acinetobacter are kept in contact isolation for the duration of hospitalization. Implementation of these types of infection control procedures has limited nosocomial spread in previously reported outbreaks (18,20,22), which is the goal of our current policy, in addition to controlling the continuing reservoir of this organism.

As previously noted, we initially suspected that colonized soldiers themselves were the reservoir for MDR Acinetobacter, and that this colonization was obtained from the environment. This hypothesis was based on 2 facts. First, these organisms are ubiquitous in the environment (4,25), and inoculation of these organisms into war wounds during traumatic blast, shrapnel, or projectile injuries seemed to be plausible. Second, Acinetobacter spp. had previously been described as common pathogens in war wounds (3), supporting the initial hypothesis. However, these infections are apparently similar to recently reported nosocomial MDR Acinetobacter infections. Investigation into the cause of these infections is ongoing, but the source is unlikely to be environmental. Multiple follow-up soil samples have not yielded Acinetobacter, yet it has been recovered from environmental cultures within field medical facilities. The final outcome of this investigation is pending further analysis.

Data from this case series demonstrate that highly resistant Acinetobacter infection, including osteomyelitis, can be successfully treated with appropriate surgical debridement, directed antimicrobial drug therapy, and careful follow-up. Our patients responded to this multifaceted approach, although their final outcome will not be determined for several years. These patients continue to be followed for recurrence of MDR Acinetobacter infection. Clearly, guided therapy based on antimicrobial drug susceptibility leads to suppression of recurrent infection up to 23 months. Most of the patients in this series did not receive extended continuation therapy with oral antimicrobial agents; whether such therapy would provide added benefit is unclear. However, few antimicrobial drug options are currently available, with none soon to be

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<th>Antimicrobial drug</th>
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<tr>
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<td>Trimethoprim/sulfamethoxazole</td>
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*Colistin susceptibility evaluated in 3 multidrug-resistant isolates.
released, to treat infections caused by resistant gram-negative organisms. Increasing prevalence of these types of infections highlights the necessity for newer antimicrobial agents with activity against these organisms.

Dr. Davis is a fellow in infectious disease at BAMC, Ft. Sam Houston, Texas. His primary research interest is nosocomial methicillin-resistant Staphylococcus aureus infections.

References


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