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REVIEW

Pseudomonas putida war wound infection in a US Marine: A case report and review of the literature

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Summary US service members are returning from the war in Iraq and Afghanistan with wound infections due to uncommon bacteria. *Pseudomonas putida*, a member of the fluorescent group of pseudomonads, primarily causes infection in immunosuppressed hosts and patients with invasive medical devices. *P. putida* has been implicated in outbreaks often traced to a contaminated fluid and is a rare cause of clinical infection. However, it should be considered a pathogen when isolated from pure culture. The objective of this article is to present a case report of a *P. putida* war wound infection, review previous *P. putida* infections, and provide a concise review of the epidemiology, risk factors, and management of infections due to this organism.
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Introduction

More than 28,000 United States service members have been wounded during the conflict in Iraq.[1] A large number of

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troops have developed war wound infections secondary to *Acinetobacter species* (sp.).[2,3] Aronson et al., in their recent review of infections among deployed personnel, point out that the nature of present day conflicts is changing the microbiology of wound infections.[4] The findings of Petersen et al. suggest that, in Iraq, Gram-negative organisms are the most common cause of war wound infections associated with injuries from high velocity projectiles, blunt force, or blast injury.[2] We present a case of a traumatic wound infection secondary to *Pseudomonas putida* and a literature review regarding the clinical characteristics and management of infections due to this organism.

Case

A 24-year-old previously healthy male United States Marine sustained blast injuries from an improvised explosive device (IED) while on foot patrol in Iraq. Injuries included right elbow traumatic arthrotomy (without fracture), bilateral open tibia/fibula fractures with compartment syndrome as well as left open talus and calcaneus fractures. Within one hour of injury, the patient underwent initial, emergent, surgical debridement overseas, including skeletal stabilization and bilateral leg fasciotomies. Extensive lower extremity injuries necessitated bilateral transtibial amputations on the day of the explosion. While en route to definitive care, several, serial debridements of extremity injuries were performed at Al Asad Airfield, Iraq, and Landstuhl Regional Medical Center, Germany. Two days after injury, the patient was admitted to National Naval Medical Center, Bethesda, MD, and 10 days later developed leukocytosis, fever of 102.0 °F, and purulent drainage from the right leg stump (Fig. 1). Laboratory evaluation revealed leukocytosis (13.5 K/uL: 79% neutrophils), thrombocytosis (720 K/uL), and an elevated C-reactive protein (2.194 mg/dL). The patient was taken to the operating room and incision and drainage of the right leg wound was performed. Intraoperative wound culture grew a non-lactose fermenting, indole-negative, oxidase-positive, Gram-negative bacillus identified as *P. putida* (Phoenix® Version: 5.15A; Beckton, Dickinson, and Company, Sparks, MD, USA). The organism was resistant to aztreonam and sensitive to fluoroquinolones, carbapenems, cephalosporins, and aminoglycosides. Blood and wound cultures for anaerobes, fungi and mycobacteria were all negative. Treatment with oral levofloxacin was initiated at 500 mg daily. Eight days after initial debridement, repeat incision and drainage of a right leg stump fluid collection again grew *P. putida*. Antibiotic regimen was changed to intravenous meropenem for 14 days. Eleven serial debridements of the right leg stump were required prior to definitive wound closure (Fig. 2). There were no further complications and the patient was discharged to physical rehabilitation without recurrence of infection.

Discussion

To our knowledge, this is the first reported case of a war-related wound infection due to *P. putida*. Petersen et al. reported that *Pseudomonas* species were the isolated source of infection in 14% of injured personnel in Iraq.[2] However, none of those isolates were *P. putida* (PC). Several other reports of isolates from the war in Iraq and Afghanistan also do not report this organism.[5,6]

P. putida is a member of the fluorescent group of pseudomonads. It is a non-lactose fermenting and oxidase-positive Gram-negative bacillus. It produces pyoverdin, a yellow–green pigment that fluoresces under UV light.[7] This organism is commonly found in the environment, including soil, prefers moist environments, and is often implicated in nosocomial infections by colonizing moist, inanimate hospital surfaces as well as patient mucous membranes and gains access to sterile body compartments through invasive medical devices or traumatic mucocutaneous defects.[7,8]

We performed a MEDLINE search from 1954 to 2007 of the English literature using the search terms: ''*Pseudomonas putida*'', and reviewed 154 papers containing 230 cases of interest (Table 1).[8–38] Historically, *P. putida* was thought to be nonpathogenic for man; however, over the last three decades, it has been recognized as a cause of true infection in the appropriate clinical setting.[34] For our review, cases were classified as true infection if they either involved bacteremia or were reported as such by the authors of the source papers. Of the 230 cases compiled for this review, 70% (160) represented true infections. A majority of the reported cases were bacteremias (39%), urinary tract infections (38%) or

Figure 1 Posterior flap failure of transtibial amputation.

Figure 2 Transtibial amputation revision following serial debridement procedures.

Table 1 *Pseudomonas putida* cases

Patient #	Author/year	Underlying conditions	Case	Diagnostics	Treatment
1—4	Von Graevenitz/1971	Rectal, sinus cancer	Bacteremia, UTI	Blood, urine culture	Various antimicrobials
5	Madhavan/1973	Intravenous drug abuse	Septic arthritis (sacroiliac joint)	Bone culture	Kanamycin, carbenicillin
6	Taylor/1984	Unk	Bacteremia[a]	Blood culture	Unk
7	Tabor/1984	Unk	Bacteremia[a]	Blood culture	Unk
8—13	MMWR/1986	Bone marrow transplant recipients, neonates	Bone marrow contamination (post-harvest), bacteremia[b]	Bone marrow, blood culture	Unk
14—28	Anaissie/1987	Leukemia, lung cancer, osteosarcoma	Bacteremia, pneumonia	Blood, sputum culture	Various antimicrobials
29	Macfarlane/1991	AML/neutropenia	Septic arthritis/bacteremia	Joint fluid, blood culture	Various antimicrobials
30	Kristensen/1995	Psychoneurosis anxiosa, perforated appendix	Bacteremia (self-induced)	Blood cultures	None, device removal only
31—85	Yang/1996	Various comorbidities	UTI, pneumonia, bacteremia, wound infection, meningitis, peritonitis	Unk	Various antimicrobials
86—91	Martino/1996	Lymphoma, AL, myeloma	Bacteremia	Blood culture	None, device removal only
92—114	Zafar/1998	Unk	Pseudo-UTI[c]	Urine culture	No treatment indicated
115	Fujita/1998	Bronchiectasis	Pneumonia	Sputum culture	Ceftazidime, minocycline
116	Ladhani/1998	Neonate	Dermatitis, meningitis, bacteremia	Blood, CSF cultures	Ceftazidime
117	Chiu/1998	Craniopharyngioma, panhypopituitarism, hypothermia	Bacteremia	Blood culture	Ceftazidime, amikacin
118—145	Weng/1999	Neurosurgical procedure	UTI	Urine culture	Unk
146—155	Romney/2000	Visited Ear, Nose, Throat clinic	Contaminated laboratory specimens[d]	Tissue culture	No treatment indicated
156	Spelman/2000	CAD, status-post CABG	Bacteremia	Blood culture	Unk
157—159	Szeto/2001	End-stage renal disease	Dialysis-associated peritonitis	Peritoneal dialysis effluent culture	Unk
160—184	Lombardi/2002	Unk	UTI, pneumonia, otitis, wound infection	Urine culture	Unk
185—188	Torii/2003	Unk	Bacteremia[e]	Blood culture	Unk
189	Docquier/2003	Unk	UTI	Urine culture	Unk
190—207	Bouallegue/2004	Neonates	Bacteremia[f]	Blood/umbilical catheter culture	Appropriate antimicrobial agent
208—214	Korcova/2005	Immunocompromised children	Bacteremia	Blood culture	Various antimicrobials
215	Chen/2005	None	Lower extremity cellulitis, bacteremia	Blood culture	Ceftazidime
216—220	Horii/2005	Unk	Acute, repeated or chronic UTI	Urine culture	Various antimicrobials

221—222	Perz/2005	Neonates	Bacteremia[b]	Blood culture	Ampicillin, gentamicin, device removal
223	Lew/2005	End-stage renal disease	Dialysis-associated peritonitis	Peritoneal dialysis effluent culture	Ceftazidime (intraperitoneal) Ciprofloxacin (oral)
224	Ying-Chen/2006	Myopia & astigmatism with use of orthokeratology contact lens	Infectious keratitis	Culture of cornea, contact lens, lens solution	Topical ciprofloxacin
225	Dervisoglu/2007	Peritoneal dialysis	Dialysis-associated peritonitis	Peritoneal dialysis effluent culture	Ceftazidime/gentamicin (intraperitoneal)
226—228	Aumeran/2007	Retinoblastoma, hepatoblastoma, AML graft	Bacteremia[f]	Blood culture	Unk
229—230	Almuzara/2007	Urethral catheter	UTI, pneumonia	Urine, tracheal aspirate culture	Unk
231	Current case	Blast injury	Post-traumatic wound infection	Wound culture	Meropenem

Abbreviations: AML = Acute myelogenous leukemia; AL = acute leukemia; UTI = urinary tract infection; CSF = cerebrospinal fluid; Unk = unknown or not reported; CAD = coronary artery disease; CABG = coronary artery bypass graft.

[a] Associated with contaminated blood products and topical anaesthetic agents.
[b] Associated with contaminated heparin sodium used to preserve bone marrow and to flush umbilical artery catheters.
[c] Associated with contaminated urine collection kit.
[d] Associated with contaminated anti-fog product used in clinic examination room.
[e] Associated with contaminated glass syringes.
[f] Associated with contaminated antiseptic solution.

pneumonias (13%).[8–14,16–21,23,24,26,27,29,31–36] Infections of the skin and soft tissue (4%), peritoneum (4%), central nervous system (2%), ear (2%), joints (1%), and cornea (1%) are uncommon but have been reported.[14,15,21–25,30,36,37] Of the 160 infections, 62% were nosocomially acquired. The majority of nosocomially acquired infections were either medical device-related (40%) or involved infusion of contaminated solutions leading to bacteremia (25%); infections involving stored blood products, topical anaesthetic agents, heparin flushes, and contact lens solution have been reported.[9–13,15,20,22,26,27,30–36] One case of auto-infection has been reported and involved injection of plant water utilizing a central venous catheter.[20] Underlying immunosuppression was reported in 25% of the infections.[8,10,11,13,15,22,24,26,30,34,36] Based on our review of the literature, it is unclear if the underlying immunosuppression alone is a risk factor to infection with *P. putida* since a majority (93%) of immunosuppressed individuals also had invasive medical devices, which may have been their predominant risk factor.[10,11,15,22,24,26,30,34,36] Further, even in this critically ill population, there were seven cases in which patients spontaneously cleared the organism with removal of the device.[10,26] Lending support to medical device-related risk is a report from one study of immunosuppressed individuals which noted an increase in *P. putida* septicaemia after an institution-wide increase in the use of indwelling central venous catheters.[10] Skin and soft tissue infections with *P. putida* are rare, with only seven previously published reports.[14,21,23,36] Of these seven skin and soft tissue infections, three were traumatic wounds that were cured with surgical debridement and antibiotic therapy, one was a neonate with a scalded skin-like syndrome with *P. putida* isolated from CSF and blood, two had wound infections without details, and one had presumed *P. putida* cellulitis which progressed to bacteremia. It is important to note that at least two of these skin infections were presumptive diagnoses. Of the 70 cases that were not classified as true infections, 69% were due to contamination of tissue and culture specimens.[13,28,36,38] For example, 10 of these cases were associated with tissue specimens that were collected in an outpatient clinic and contaminated by an anti-fog solution used to clean the examination room, and 23 cases were associated with contaminated urine collection kits.[28,38]

P. putida infection is also associated with outbreaks and has generally been attributed to a water source such as detergent-disinfectants diluted with tap water, showerheads, improperly sterilized glass syringes, and pharmacy compounded heparin flushes.[11,27,33] While it is uncertain where our patient acquired the infection, the failure to identify any other infections due to *P. putida* during and after his hospitalization at the US (our) facility makes a point source outbreak unlikely. Due to the time course of the presentation, it is possible that this organism might have been acquired at a field hospital, similar to the mechanism proposed by Scott et al.[3]

Based on the information gleaned from our literature review, we cannot suggest specific antibiotic regimens or duration of therapy for *P. putida* infection. Empiric therapy is similar to that of other *P.* species and should take into account the degree of illness in the patient. As with other bacterial infections, treatment should be based on available antimicrobial sensitivities combined with adequate surgical debridement when they represent primary soft tissue infections. Strains of *P. putida* resistant to fluoroquinolones, carbapenems, and aminoglycosides have been reported. Horii et al. reported on *P. putida* urinary isolates demonstrating that amino acid mutations in the quinolone resistance-determining regions may confer high-level resistance to fluoroquinolones.[18] Isolates with the IMP-type metallo-β-lactamase gene conferring carbapenem resistance were also seen in these isolates.[18] Mendes et al. reported an isolate from Latin America that acquired an aminoglycoside resistance gene that conferred phenotypic resistance to all clinically available aminoglycosides.[39] Our isolate was sensitive to levofloxacin, MIC 1.5 μg/mL (Etest®, AB Biodisk, Solna, Sweden), but our patient failed early therapy with this agent. The Clinical and Laboratory Standards Institute levofloxacin breakpoint for non-enterobacteriaceae is MIC $\leq$ 2 μg/mL.[40] Interestingly, using Monte Carlo simulations, Zelenitsky et al. demonstrated probability of ciprofloxacin cure rates as low as 27% when used in standard doses to treat *P. aeruginosa* infection.[41] Additionally, Kiser et al., again using Monte Carlo simulations, were unable to attain satisfactory probability of target attainment (PTA) with high-dose levofloxacin (750 mg daily) in Gram-negative isolates with MICs of 1 μg/mL.[42] Therefore, based on pharmacodynamic studies, fluoroquinolone monotherapy might be more prone to failure in the management of recalcitrant Gram-negative infections outside the urinary tract and other agents might be more effective.

Conclusion

Our case report and review illustrate several features about the epidemiology and management of infections due to *P. putida*. Based on the proportion of true infections in our review (70%), providers should not attribute isolation of this organism to insignificant culture contamination. Most infections are nosocomially acquired and the use of medical devices appears to be a risk factor for infection with this organism. Previous reports have suggested that immunosuppressed hosts appear to be at an increased risk of infection with this organism. Based on our review, it is not entirely clear that immunosuppression in the absence of medical devices is a significant risk factor.

P. putida is easily treated with drugs effective against other *P.* species. However, it has the ability to acquire antimicrobial resistance and providers should suspect this if first-line drug therapy fails. When skin and soft tissue structures are involved, adequate surgical irrigation and debridement is paramount to achieve cure as evidenced by the 11 serial debridements required in our patient and previous reports of surgical debridement paired with antibiotic therapy.[36] We believe our patient failed initial therapy due to inadequate irrigation and debridement and not true antibiotic failure. However, for reasons discussed above, it is also possible that we did not achieve adequate antibiotic concentration at the wound using standard doses of fluoroquinolone monotherapy. When choosing an antimicrobial agent for infections due to *P.* species, providers should consider pharmacodynamics as well as MIC in order to optimize therapy. Finally, a clustering of cases should mandate considerations for an outbreak investigation.

Conflict of interest

There are no conflicts of interest to report. The authors have received no financial support for this manuscript.

Disclaimer

The views expressed in this manuscript are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, nor the US Government.

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