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Case report

Bullous cellulitis caused by *Pseudomonas putida* in a patient with end-stage renal disease

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ABSTRACT

We present a case of bullous cellulitis in a 75-year-old male caused by *Pseudomonas putida* (*P. putida*) acquired from contact with contaminated water. Careful documentation of *P. putida* soft tissue infection is warranted given the rise in infections, marked antimicrobial resistance, and fatalities observed in a limited number of cases.

Introduction

Pseudomonas putida is a rod-shaped, flagellated, gram-negative bacterium found in most soil and water habitats. Due to its ability to metabolize organic pollutants such as toluene, it can thrive in areas rich in organic wastes [1,2]. Like other species of *Pseudomonas* known to cause soft tissue infections, the literature contains several cases of *P. putida* soft tissue infections and bacteremia resulting in a few reported fatal cases [3–5].

Bullous cellulitis is a typical presentation of soft tissue infection most often caused by *beta*-hemolytic *streptococci*, and less commonly by other bacteria such as *Serratia* [6]. *P. putida*, to our knowledge, is not known to cause bullous cellulitis, although the more studied *Pseudomonas aeruginosa* has been implicated in two case reports on bullous soft tissue and superficial skin infections [7,8]. In this report, we present a case of a 75-year-old male with history of end stage renal disease (ESRD) who presented with bullae in his right lower extremity (RLE) accompanied by associated swelling, erythema, and pain. Subsequent blood and wound cultures were positive for *P. putida* and he was successfully treated with targeted antimicrobials. We have discussed important features which, if present warrant prompt suspicion for *P. putida* infection thus guiding appropriate empiric antimicrobial coverage before definitive bacteria culture and sensitivity results are obtained.

Case report

A 75-year-old Caucasian male with history of ESRD presented with worsening erythema, swelling, and pain in his RLE for one month. He specifically denied exposure to lake water, well water, or hot tubs. After further prompting, he stated that his demise started after he attempted to clean a difficult-to-heal wound that had developed on the shin of his RLE. He used diluted Epsom salt in a “cleaned and sterilized” bucket that had earlier been used to carry materials for roof repair. On presentation, his pain was cramping/burning, 10/10 at worse and radiating up to his groin. He denied any numbness, tingling, or paresthesia, fevers, chills, or night sweats.

On initial exam, his vital signs were temperature of 37 °C, heart rate of 75 beats per minute, respirations of18/minute, blood pressure of 133/61 mmHg, and oxygen saturation of 96 % on room air. A circumferential erythema/swelling with irregular borders was noted on his RLE. The erythema extended just above the knee posteriorly. Two wound openings were noted on the shin area, one with minimal purulent discharge. Two plus pitting edema was also noted in his RLE. Toe movements were intact, as well as bilateral lower extremity sensation and strength. Pulses were palpable on both extremities, albeit less prominent on the RLE.

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Fig. 1. (**A**) Right lower extremity one day after treatment was initiated. Noticeable bullae noted. Black arrow indicates the original puncture wound which the patient attempted cleaning with Epsom salt. On presentation, the wound had mild purulent discharge which served as the sample for culture. (**B**) Right lower extremity two days prior to discharge. Marked improvements in resolution of inflammation. Bullae previously noted are absent.

Table 1
Laboratory values on admission.

Values	On presentation	Reference values
White blood cells	7.8	(4.5–11.0 thou/mm^3)
Hematocrit	38.4	(35.0–49.0 %)
Platelet	97	(150–450 thou/mm^3)
Sodium	136	(136–145 mmol/L)
Potassium	4.6	(3.5–5.1 mmol/L)
Chloride	101	(98–107 mmol/L)
Bicarbonate	27	(21–32 meq/L)
BUN	42	(7–18 mg/dL)
Creatinine	4.53	(0.60–1.30 mg/dL)
Glucose	111	(74–106 mg/dL)
Calcium	8.8	(8.5–10.1 mg/dL)
Phosphorus	5.1	(2.5–4.9 mg/dL)
Magnesium	2.3	(1.8–2.4 mg/dL)
Albumin	3.0	(3.5–5.0 g/dL)
Lactic acid	3.0	(0.4–2.0 mmol/L)
Creatinine kinase	14	(39–308 units/L)

BUN; blood urea nitrogen.

Notable on the RLE were bullae (Fig. 1A). His initial laboratory findings on presentation are shown in Table 1. Blood and wound cultures were sent, and he was started on empiric treatment with cefazolin and vancomycin pending culture results. His blood and wound cultures came back positive for *P. putida* and his antibiotic regimen were appropriately adjusted after sensitivity was established (Table 2). A summary of the patient's hospital course can be found in Table 3. His repeat blood cultures were negative and his RLE swelling and erythema had significantly improved at discharge (Fig. 1B).

Discussion

Given its ability to break down organic compounds, it is highly likely that *P. putida* was contracted from the contaminated bucket used by the patient. Chemical materials used for roof coating contains organic compounds conducive for *P. putida* to thrive. This important piece of history was, thus, important in identifying a source for his exposure. Also, *P. putida* is known to be mostly soil-dwelling. Strong suspicion for it as a cause of cellulitis should be considered if a patient reports exposure of open wounds to soil or contaminated water.

P. putida, like other species in the genus, has a predilection forimmunocompromised patients [9,10]; however, our suspicion for a Pseudomonal infection was low because our patient was a non-diabetic and he was not immediately forthcoming with information about cleaning his wound. Also, the presence of bullae, typically seen in streptococcal soft tissue infection, further swayed us away from a possible Pseudomonal infection. In addition, because there was pus draining from one of the openings on his shin, suspicion for a Staphylococcal infection was high. These aspects together guided our choice for empiric antibiotic coverage before definitive culture results were obtained.

The sensitivities of the isolated *P. putida* to antibiotics are shown in Table 1. As shown, this strain was sensitive to almost all antimicrobials tested; however, there are many reported cases of *P. putida* infection caused by highly resistant strains, some known to have a 30-day mortality as high as 39 % [11,12]. Interestingly, such resistant strains have been mostly been reported in Asian countries where most *P. putida* cases have been observed [5,9]. Thus, as cases of *P. putida* infection are increasingly being observed in the USA, it might be expected that bacterial resistance will develop. For this reason, careful consideration of empiric antibiotic coverage is warranted in cases of suspicious *P. putida* infection. Therefore we propose that, in addition to the known risk factors for Pseudomonal infection, the presence of an immunosuppressed state or chronic medical condition such as ESRD, and exposure of open skin to contaminated water or soil, should further increase the suspicion for *P. putida* soft tissue infection. Also, of note, the presence or absence of bullae should not be used as a defining feature, as bullae formation is non-specific and could be present in any soft tissue infection.

In conclusion, we have presented a case of bullous cellulitis caused by *P. putida*. Most previously documented cases are from Asian countries, with a few documented cases showing marked antibiotic resistance and, in a limited number of cases, causing fatality. Once suspicion for *P. putida* infection is high, prompt initiation of appropriate empiric antibiotic coverage is needed to ensure optimal outcome. Therefore, physicians must be keen to act when identifiable risk factors for such Pseudomonal infections are present.

Table 2
Sensitivity of *Pseudomonas putida* to antimicrobials.

Antibiotic	Reaction	MIC
Gentamicin	S	< = 1
Tobramycin	S	< = 1
Amikacin	S	< = 2
Ciprofloxacin	S	< = 0.25
Ceftazidime	S	4
Cefepime	R	> = 64
Piperacillin/Tazobactam	I	64
Meropenem	S	2

S: Sensitive; I: Indeterminate; R: Resistant; MIC: Minimal inhibitory concentration.

Table 3
Timeline of events.

Date	Events
Hospital day 1	Patient presents with RLE swelling, erythema, and pain. Blood and wound cultures sent; empiric treatment with cefazolin and vancomycin initiated
Hospital days 2–3	Positive blood and wound cultures for *Pseudomonas putida*; cefazolin and vancomycin discontinued; Infectious disease consulted; patient started on cefepime/ciprofloxacin; interval worsening of inflammation in the RLE; bands noted on CBC; interval worsening of bullae; CT scan of extremities unremarkable for free air; wound care consulted; compression stockings and warm compresses applied.
Hospital day 4	Antibiotic switched to meropenem based on sensitivity results; cefepime/ciprofloxacin discontinued due to interval worsening of inflammation; repeat blood culture sent; resolution of bands previously noted in CBC
Hospital day 7	Repeat blood culture negative; swelling and inflammation improved
Hospital day 10	Completed meropenem treatment; inflammation markedly improved; patient noted to have melena; hemoglobin of 7.7; initiated pantoprazole and GI consulted; planned EGD on 01/14
Hospital day 14	EGD shows grade A esophagitis and a nonbleeding gastric ulcer; recommended to continue pantoprazole and follow up after discharge
Hospital day 15	Patient received 1-unit PRBC with dialysis because his hemoglobin was <7.0; He was later discharged from hospital, following stabilization of his hemoglobin, in stable conditions.

RLE: Right lower extremity; CBC: Complete blood count; CT: computed tomography; EGD: Esophageal gastroduodenoscopy; PRBC: Packed red blood cell.

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CRediT authorship contribution statement

Joshua K. Salabei: Conceptualization, Writing - original draft, Writing - review & editing. **Troy J. Fishman:** Writing - review & editing. **Aya Marachi:** Writing - review & editing. **Veronica M. Lopez:** Writing - review & editing. **Yvette Bazikian:** Writing - review & editing, Supervision. **Matthew Calestino:** Writing - review & editing, Supervision.

Declaration of Competing Interest

The authors declare that there are no conflicts of interest regarding the publication of this article

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