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Brief Reports

Community-Acquired Necrotizing Fasciitis Caused by *Serratia marcescens*: Case Report and Review

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Serratia marcescens is a gram-negative bacillus that has been recognized as a human pathogen since the 1960s [1]. This organism is known to cause a variety of infections including bacteremia, pneumonia, endocarditis, meningitis, and septic arthritis [2]. Community-acquired infections are uncommon and most often occur in immunocompromised or neutropenic persons [2, 3]. *Serratia marcescens* is also an unusual cause of soft tissue infections such as cellulitis and necrotizing fasciitis [3]. We report a case of community-acquired necrotizing fasciitis secondary to *Serratia marcescens* infection that occurred in an otherwise healthy woman.

A 66-year-old woman was admitted to the hospital because of left leg pain. She had apparently been well until 1 day earlier when she experienced rapid onset of pain, redness, and swelling in her left ankle. Six hours later the pain, redness, and swelling had spread from her left ankle to her left knee. No history of leg trauma or chronic leg ulcer was noted. The patient had previously been healthy. Upon examination, her temperature was 36 °C, pulse 112 beats/min, respirations 22 times/min, and blood pressure 100/80 mmHg. The patient was alert and oriented. Her left leg was markedly tender and swollen from the ankle to the knee, and the skin was erythematous with multiple areas of bluish discoloration. No crepitation was noted on palpation. The patient's lungs, heart, and abdomen were normal.

Laboratory tests revealed the following values: hemoglobin, 14.4 g/dl; leukocyte count, 4,900/mm^3 (54% granulocytes, 20% bands and 23% lymphocytes); platelet count, 207,000/mm^3; sodium, 139 mmol/l; potassium, 3.2 mmol/l; chloride, 106 mmol/l; carbon dioxide, 18 mmol/l (30 mmol/l, 2 weeks before admission); blood urea nitrogen, 28 mg/dl; serum creatinine, 1.4 mg/dl (0.7 mg/dl, 2 weeks before admission); and creatine kinase, 65 U/l. A left lower extremity radiograph revealed soft tissue swelling without evidence of gas. Blood was drawn for culture, and intravenous (i.v.) therapy was started with clindamycin (600 mg i.v. every 8 h) plus penicillin G (4×10^6 U i.v. every 4 h). One hour later, the patient's temperature was 34.5 °C, pulse 120 beats/min, respirations 26 times/min, and blood pressure 60/40 mmHg. At this time, the patient was lethargic and disoriented. Her left leg had developed bullae formation with a focal area of crepitation around the ankle. She was then intubated and mechanical ventilation was initiated. The clinical diagnosis of necrotizing fasciitis was made. A Gram stain of an aspirate of a bulla revealed numerous gram-negative bacilli with few leukocytes. Ceftriaxone (2 g i.v. every 4 h) was added to her therapy. Surgical debridement and left above-knee amputation was performed. A Gram stain of the deep tissue specimen also demonstrated gram-negative bacilli. Cultures of blood, fluid aspirated from a bulla, and surgical specimens yielded *Serratia marcescens* (Biochemical Identification Card; Gram Negative Identification Plus; bioMérieux Vitek, USA); anaerobic cultures of all samples were negative. Despite aggressive therapy with surgery and antibiotics, the patient remained hypotensive and developed multiorgan failure. She died after 2 weeks of aggressive treatment.

Serratia marcescens belongs to the family *Enterobacteriacea*e [1]. The majority of infections caused by this organism are nosocomial in origin. Bouza et al. [4] reported the results of a survey of 146 hospitalized patients who had *Serratia* bacteremia: only 8% of the cases were community acquired. *Serratia* has been reported as an uncommon cause of community-acquired soft tissue infection [3]. Necrotizing fasciitis caused by this organism is rare, especially in immunocompetent persons.

Using Medline, we searched the English-language literature published between January 1966 and May 1999. The terms utilized in the search were *Serratia marcescens*, soft tissue infection, cellulitis, and necrotizing fasciitis. Reference lists of the identified articles were also reviewed to find additional cases.

We found nine instances of *Serratia marcescens* community-acquired soft tissue infection described in the literature [3, 5–9]. Including our patient, the infections occurred in five males and five females whose median age was 57.5 years (range, 23–88 years) (Table 1). Risk factors included chronic leg edema, history of trauma, chronic leg ulcer, chronic renal failure, and diabetes mellitus. The common clinical presentation was cellulitis (80%). Of the eight patients who presented with cellulitis, one died secondary to septic shock and multiorgan failure, and the others survived after antibiotic therapy. Only two cases (1 of which is ours) of necrotizing fasciitis caused by *Serratia marcescens* have been reported. Our patient died despite aggressive treatment with surgery and antibio-

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Table 1 Clinical data of 10 patients with *Serratia marcescens* community-acquired soft tissue infection

Case no.	Year [reference]	Age (y)/sex	Underlying condition and/or risk factor	Site of infection	Clinical presentation	Antibiotic therapy	Outcome
1	1977 [5]	51/M	idiopathic cardiomyopathy, chronic leg edema	left lateral thigh	cellulitis, septic shock	nafcillin gentamicin	died
2	1983 [6]	60/M	peripheral neuropathy, steroid use, left leg trauma	left calf	cellulitis, septic shock	nafcillin gentamicin	recovered
3	1988 [7]	71/F	healthy, left hand injured with a wire	left hand	cellulitis	gentamicin	recovered
4	1988 [7]	23/M	healthy	dorsum of right digit	cellulitis	gentamicin	recovered
5	1988 [7]	52/M	diabetes, diabetic foot ulcer	right great toe	cellulitis	cefotaxime	amputation of great toe recovered
6	1991 [8]	88/F	chronic venous dermatitis and lower leg ulceration	left leg	cellulitis	cefazolin ciprofloxacin	recovered
7	1991 [8]	60/M	chronic venous dermatitis	left lateral malleolus	cellulitis	ciprofloxacin	recovered
8	1992 [3]	37/F	chronic renal failure	right axilla	cellulitis, septic shock	vancomycin ciprofloxacin	recovered
9	1996 [9]	55/F	diabetes mellitus	right leg	necrotizing fasciitis	ceftizoxime clindamycin	recovered
10	1999 [present report]	66/F	healthy	left leg	necrotizing fasciitis	clindamycin penicillin G ceftriaxone	died

tics. The other patient survived after treatment with antibiotics, surgical debridement, and fasciotomy.

In an animal experiment, injection of purified proteinase from *Serratia marcescens* into rat skin led to increased vascular permeability, necrosis of epidermal tissue, lifting off to the keratinized layer, dermal inflammation and edema, and infiltration of polymorphonuclear leukocytes into the subcutaneous fat and muscle [10]. These observations suggest that *Serratia marcescens* may secrete a proteinase that causes clinical necrotizing fasciitis, a possibility that deserves further investigation.

Third-generation cephalosporins, fluoroquinolones, or imipenem/cilastatin are recommended therapy for infections due to *Serratia marcescens*. However, antibiotic treatment of *Serratia* infection is complicated by the high frequency of multiple drug resistance. The mechanisms of resistance may be due to the production of the enzyme cephalosporinase by the gene in the bacterial chromosome or production of plasmid-encoded enzyme beta-lactamases. The fluoroquinolones may be particularly useful for treating infections caused by these beta-lactamase-resistant strains [1].

Although uncommon, *Serratia marcescens* can cause serious, potentially fatal soft tissue infections, such as necrotizing fasciitis, in an immunocompetent person. Treatment should include broad-spectrum antibiotics and aggressive surgical debridement of devitalized tissue. Antibiotic regimens should be tailored to the resistance patterns of organisms isolated from infected tissue. The mortality rate is high once multiple organ system failure develops.

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