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Fatal Serratia Marcescens Necrotizing Fasciitis in an Immunocompetent Man

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Abstract

Necrotizing fasciitis is a difficult-to-treat, life-threatening infection that affects the superficial fascia and the subcutaneous tissue, causing gradual tissue necrosis, often due to a combination of aerobic and anaerobic bacteria. We describe the case of a 57-year-old immunocompetent man who presented with pain in his left leg, local oedema, and dyspnoea. Clinical examination and subsequent histopathological findings were consistent with the diagnosis of necrotizing fasciitis. Tissue biopsies and blood cultures demonstrated the presence of *Serratia marcescens*. Adequate antibiotic treatment and rapid surgical debridement were accomplished. Despite intensive and aggressive treatments, the patient quickly developed refractory multiple organ failure leading to death. To the best of our knowledge, this is the first fatal case of *S. marcescens* related necrotizing fasciitis in an immunocompetent man. The patient's fulminant evolution contributes to the uniqueness of this case.

Keywords: Serratia marcescens; Necrotizing fasciitis; Soft tissue infection; Community-acquired infection; Immunocompetent patient; Septic shock

Introduction

Serratia marcescens

Serratia marcescens, a Gram-negative facultative anaerobic bacterium, is an opportunistic pathogen often found in healthcare settings, belonging to the Enterobacteriaceae family. *S. marscecens* can cause a wide spectrum of infections involving urinary tract, bloodstream, skin, soft tissue, bones, respiratory tract, eyes, and central nervous system. *S. marcescens* is a very unusual cause of soft tissue infections, appearing only in the presence of certain risk factors, such as immunosuppression, diabetes mellitus, antibiotic and/or steroid exposure, alcoholism, trauma, and peripheral

vascular disease [1].

The mechanism through which *S. marcescens* induces soft tissue infections remains unclear; however, it could potentially be linked to the release of virulence factors by the microorganism. *Serratia sp.* releases a variety of compounds, including hemolysin, nuclease, lipases, lecithinase, siderophores, metalloproteases, and notably proteases. These proteases possess characteristics that can enhance vascular permeability, trigger epidermal tissue necrosis, incite dermal inflammation and swelling, and contribute to the infiltration of polymorphonuclear leukocytes into muscle tissue. [2] In addition, vasoconstriction caused by bacterial toxins and immune responses results in fascial spaces not reached by blood vessels, leading to necrosis, and hindering antibiotic penetration into tissues.

Serratia infections are challenging to treat due to their frequent resistance to multiple antibiotics. Resistance mechanisms involve enzymes like cephalosporinase and plasmid-encoded beta-lactamases. These bacteria often possess inherent or acquired resistance, including inducible chromosomal AmpC β-lactamases, for ampicillin and first-generation cephalosporins. Effective treatment options include aminoglycosides, third- and fourth-generation cephalosporins, and carbapenems.

Serratia marcescens is also an infrequent yet highly dangerous trigger for a particular type of soft tissue infection such as necrotizing fasciitis, a medical condition also known as "flesh-eating disease". [3] Cases of necrotizing fasciitis related to *S. marcescens* have recently become more prevalent in the published literature [1, 4].

Necrotizing fasciitis

Necrotizing fasciitis, first described in the early 20th century [5, 6], is a life-threatening infection primarily affecting superficial fascia and subcutaneous tissue, causing gradual tissue destruction. It commonly occurs in the lower limbs but can affect other body parts. [7] Typically, it involves a mix of aerobic and anaerobic bacteria, making it a polymicrobial infection.

Early detection is challenging, as it can resemble cellulitis or abscesses. Diagnosis often relies on late signs like crepitus, skin necrosis, and bullae. [8] However, their absence doesn't rule out the disease [9].

Necrotizing fasciitis has a significant mortality rate, especially when caused by uncommon pathogens like *S. marcescens*. The first reported case of *S. marcescens*-associated lower limb necrotizing fasciitis dates back to 1987 [10].

At the beginning, muscle and dermal tissue are unaffected, but necrosis occurs in the fascia and subcutaneous tissue due to skin vessels microthrombosis, leading to devascularization and the formation of necrotic tissue. [11]

Another common finding is blisters, resulting from necrolysis, as consequence of the same ischemia-triggered mechanism.

Histological progression involves gradual declines in neutrophilic response and polymorphonuclear infiltration, alongside heightened bacterial proliferation. This corresponds to increased tissue ischemia, reduced perfusion, and intensified bacterial growth. As histopathological staging advances, mortality rates also rise [12].

Classifications

Two distinct forms exist for necrotizing fasciitis. Type I involves mixed aerobic and anaerobic bacteria, commonly emerging post-surgery in aged and diabetic patients. Type II signifies monomicrobial infections caused by *Group A Streptococcus* or *Staphylococcus aureus*, affecting younger, healthy individuals. Some Authors [1, 13] argue for a third type related to *Vibrio vulnificus* and gas gangrene, yet scientific consensus varies. Multimicrobial infections are more frequent than monomicrobial, with the latter impacting younger individuals with minor trauma history.

Clinical staging of necrotizing fasciitis proposed by Wang et al. [7] describes symptoms and signs that evolve with the disease as the infection progresses (Table 1). Initial evaluation reveals erythema, tenderness, warmth, and swelling in most patients (stage 1): this early-stage necrotizing fasciitis may be confused with other soft tissue infections. Blistering occurs in only 41% of early-stage patients: as the disease progresses, blisters increase (stage 2), often followed by late-stage indicators such as skin crepitus, anesthesia, and necrosis (stage 3).

Clinical stage	**Stage 1 (early)**	**Stage 2 (intermediate)**	**Stage 3 (late)**
Clinical features	Erythema	Blister/Bullae	Crepitus
	Tenderness		Skin
	Calor		Anesthesia
	Swelling		Necrosis

Table 1: Clinical stages of necrotizing fasciitis. Skin alterations are heterogeneous: the region displaying the most progressed changes should define the clinical stage [7].

The Laboratory Risk Indicator For Necrotizing Fasciitis (LRINEC) score [9] is a helpful tool to differentiate necrotizing fasciitis from milder skin infections. It employs routine lab tests to predict necrotizing fasciitis (Table 2): the maximum score is 13; 6 or higher raises suspicion, while 8 or higher strongly predicts the disease. An elevated LRINEC score (≥ 8 out of 13 points) is reported to have a sensitivity of only 32.4% (95% Confidence interval (CI) 22.0%-45.1%) and specificity of 93.9% (95% CI 80.9%-98.2%)

[14], so despite the utility of this score, it's crucial to emphasize that necrotizing fasciitis is primarily diagnosed through surgical evaluation.

Variable (units)	Score
C-reactive protein (mg/L)	
< 150	0
≥ 150	4
Total white cell count (x10,000/µL)	
< 15	0
15-25	1
> 25	2
Hemoglobin (g/dL)	
> 13.5	0
11-13.5	1
< 11	2
Sodium (mEq/L)	
≥ 135	0
< 135	2
Creatinine (µmol/L)	
≤ 141	0
> 141	2
Glucose (mmol/L)	
≤ 10	0
> 10	1

Table 2: The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score [9].

Case Presentation

We present the case of a 57-year-old man of Filipino origin on vacation in Switzerland who arrived from Manila two weeks earlier. He came to our Emergency Department for worsening pain in his left leg, associated with local oedema and dyspnoea for about 24 hours. His past medical history included arterial hypertension, hiatus hernia, dyslipidemia, and recurrent tonsillitis, without hospitalization during the last year. The patient stated that he had not used alcohol or intravenous drugs in the past or at present, that he had not suffered any recent trauma, and that he had not recently undergone any dental work.

Physical exam at presentation revealed a tympanic temperature of 36°C, blood pressure of 88/50 mmHg, heart rate 100 bpm, and SpO_2 100% in room air. The patient showed no wounds on his leg. Laboratory values included creatinine 291 µmol/L, C-reactive protein 312 mg/L, platelets $95x10^9$/L, white blood cells $0.5x10^9$/L, neutrophils $0.23x10^9$/L, pH 7.33, lactates 3.9 mmol/L, hemoglobin 150 g/L, sodium 130 mmol/L, glycemia 7.8 mmol/L, creatine kinase 6073 IU/L. The calculated LRNEC score was 8 out of 13 (high risk for necrotizing soft tissue infection). Leptospirosis, HIV, and *Treponema pallidum* infection were ruled out.

Given the clinic, a Revised Geneva Score of 12 points (high risk for pulmonary embolism) and a Wells Score of 6 points (moderate risk), a CT angiography was performed which excluded pulmonary embolism; an ultrasound of the left lower limb excluded vascular occlusions. Therefore, in the suspicion of necrotizing fasciitis, broad-spectrum antibiotic therapy with Vancomycin, Clindamycin, and Imipenem/Cilastatin was immediately set up after obtaining blood cultures.

Due to the high suspicion of necrotizing fasciitis, the patient was urgently taken to the operating room where he underwent a fasciotomy of the left leg. Necrosis of the soleus muscle, lateral and medial gastrocnemius muscle was observed during the operation, without pus leakage, but with compartment syndrome of the leg. Subsequently, histopathology confirmed the diagnosis of necrotizing fasciitis. For hypotension with hyperlactatemia and anuria, in the setting of septic shock, crystalloids and ever-increasing doses of Norepinephrine were administered.

Following surgery, the patient was admitted to the Intensive Care Unit.

Due to superimposed hypovolemic shock, resulting from capillary leak and hemorrhage after the fasciotomies, the patient required massive transfusions. A multi-organ failure was therefore outlined, with persistent lactic acidosis and associated severe consumption coagulopathy, requiring supplementation with coagulation factors and plasma.

The PiCCO advanced cardiac output monitoring results were consistent with a mixed shock form (septic and hypovolemic), requiring massive fluid administration and high doses of catecholamines.

Continuous hemodiafiltration was started, due to renal failure and impending combined metabolic acidosis.

Given the progressive worsening of the multiorgan failure, the patient underwent above-the-knee amputation on the first day of hospitalization and, given the subsequent clinical worsening, hip disarticulation surgery on the same day.

Eight hours after hospital admission, the cultures turned positive for *Serratia marcescens* without any other pathogens in biopsies performed in the operating room and in blood cultures (Table 3). The antibiotic therapy was changed to Vancomycin, Meropenem, and Ceftazidime/Avibactam.

Antibiotic	Susceptibility
Ampicillin	Resistant
Amxocillin/Clavulanic acid	Resistant
Piperacillin/Tazobactam	Sensitive
Cefuroxime	Resistant
Cefepime	Sensitive
Ceftazidime/Avibactam	Sensitive (Minimum Inhibitory Concentration 0.25 mg/L)
Meropenem	Sensitive
Ertapenem	Sensitive
Imipenem	Sensitive
Ciprofloxacin	Sensitive
Tobramycin	Sensitive
Gentamicin	Sensitive
Sulfamethoxazole/Trimethoprim	Sensitive

Table 3: Antibiotic susceptibilities of Serratia marcescens isolated from biopsies and blood cultures.

Our patient received hydrocortisone in the setting of septic shock, to treat critical illness-related corticosteroid insufficiency, and intravenous immunoglobulin, prior to microbiological results. Despite massive volume replacement, high doses of amines, multiple transfusions, rapid surgery and appropriate antibiotic therapy, the patient died of overwhelming septic shock 27 hours after hospital admission.

Discussion

We performed a literature search in PubMed spanning from 1966 up to 2023. Our search terms included "*Serratia marcescens*" and "necrotizing fasciitis". Furthermore, reference lists from identified articles were reviewed for potential additional cases. We identified 28 cases of necrotizing fasciitis due to *S. marcescens* (Table 4), with a mortality of 46.4% (13 patients out of 28) and a mean age of 49.1 years (standard deviation ±23.8 years). 18 patients (64.3%) contracted the infection in the community, while 10 (35.7%) in a hospital setting: 21 patients (75%) presented comorbidities or were immunocompromised, while only 7 (25%) were healthy or immunocompetent. No healthy or immunocompetent patient receiving extensive care like our patient died: thus, to the best of our knowledge, our patient represents the first fatal case of community-acquired necrotizing fasciitis due to *Serratia* in an immunocompetent patient.

Author	**Year**	**Age (years)**	**Sex**	**Risk factors**	**Precipitating factor**	**Infection site**	**Type**	**S. marcescens cultures**	**Treatment**	**Outcome**
Rimailho et al.[10]	1987	74	M	Immunocompromised	Diclofenac use	Leg	CA	Blister, blood	None	Died
Bornstein et al.[17]	1992	37	F	Renal failure on hemodialysis	Pain during dialysis	Axilla, chest wall	HA	Wound, bullae, blood	Antibiotics, SD	Survived
Zipper et al.[18]	1996	55	F	DM	Left below-knee amputation	Leg	CA	Wound	Antibiotics	Survived
Huang et al.[19]	1999	73	M	Nephrotic syndrome	Steroid therapy	Leg	HA	Tissue, blood	Antibiotics, SD	Survived

Huang et al.[19]	1999	40	M	Uremia, peritoneal dialysis, SLE	Pneumonia with positive cultures for *S. marcescens*, steroid and nabumetone	Calf, thigh	CA	Tissue, blood	Antibiotics, SD	Survived
Liangpunsakul et al.[20]	2001	66	F	Healthy	None	Leg	CA	Blood	Antibiotics	Died
Newton et al.[21]	2002	2	F	Healthy	Pharyngitis	Cervical spine	CA	Wound, blood	Antibiotics, SD	Died
Bachmeyer et al.[22]	2004	49	M	Small-cell lung cancer, DM	Chemotherapy, cellulitis	Leg	HA	Tissue, bullae, blood	Antibiotics	Survived
Curtis et al.[23]	2005	51	M	ESRD, T2DM, CHF	Scraped knee on rock in river	Leg	CA	Wound, blood	Antibiotics, SD	Died
Statham et al.[24]	2009	6	M	Immunocompetent	Suspected pharyngitis	Oropharynx	CA	Wound, blood	Antibiotics, SD	Survived
Motsitsi et al.[25]	2011	37	M	Healthy	Human bite	Forearm	CA	Wound	SD	Died
Vano-Galvan et al.[26]	2012	57	F	CML, immunocompromised	Minor trauma	Thigh	HA	Blister, blood	Antibiotics	Died
Prelog et al.[27]	2012	15	F	Acute lymphocytic leukemia	Venous access-port implantation	Axilla, venous access-port site	HA	Wound	Antibiotics, SD	Survived
Wen[28]	2012	40	F	Nephrotic syndrome, cyclosporine use	Chemotherapy	Leg	CA	Wound, blood	Antibiotics	Died

Rehman et al.[29]	2012	54	F	SLE, ESRD	Central venous catheter, arteriovenous fistula ligation, steroid therapy	Chest wall	HA	Wound, blood	Antibiotics, SD	Died
Cope et al.[30]	2013	97	F	Heart failure, CKD	Heart failure exacerbation	Leg	HA	Wound (post-mortem)	Antibiotics	Died
Lakhani et al.[1]	2015	51	F	DM, PVD	Bifemoral bypass, left distal femoral aneurysm repair	Abdomen	HA	Wound, blood	Antibiotics, SD	Survived
Majumdar et al.[31]	2016	54	F	DM, ESRD	Leg wound due to calciphylaxis, skin biopsy	Leg	HA	Wound	Antibiotics, SD	Died
Hagiya et al.[32]	2016	64	M	Cirrhosis, hepatocellular carcinoma	Leg wound due to burn	Leg	CA	Tissue, blood	Antibiotics	Died
Lin et al.[33]	2016	56	M	DM, betel nut chewing	Tooth extraction	Oropharynx	CA	Blood	Antibiotics, SD	Died
Tyler et al.[34]	2016	12	F	Healthy	Leg trauma	Leg	CA	Wound	Antibiotics, SD	Survived
Heigh et al.[35]	2016	60	M	DM, CAD, cirrhosis	Laryngectomy	Leg, foot	HA	Tissue, blood	Antibiotics, SD, AKA	Survived
Marin et al.[3]	2017	55	F	DM, CAD	None	Leg, foot	CA	Wound, blood	Antibiotics, SD	LAMA
Marin et al.[3]	2017	50	M	DM	Foot traumas	Foot	CA	Tissue	Antibiotics, SD	LAMA
Roberts et al.[13]	2018	73	F	Healthy	None	Leg	CA	Wound, blood	Antibiotics, SD, AKA	Survived
Chidambaram et al.[36]	2021	3	M	Healthy	Snake bite	Calf	CA	Tissue	Antibiotics, SD	Survived

Aftab et al.[37]	2023	56	M	ESRD, lupus nephritis, renal cell carcinoma	Steroid therapy	Leg	CA	Wound, tissue, blood	Antibiotics, SD	Survived
Sakkab et al.[38]	2023	90	M	T2DM, CKD, PVD, chronic hepatitis C	Chronic ankle wound	Ankle	CA	Wound, blood	Antibiotics	Died
Present case	2023	57	M	Immunocompetent	None	Leg	CA	Tissue, blood	Antibiotics, SD, AKA	Died

Table 4: Necrotizing fasciitis due to *Serratia marcescens* cases, from 1966 to 2023. AKA, above-the-knee amputation; CA, community-acquired infection; CAD, coronary artery disease; CHF, chronic heart failure; CKD, chronic kidney disease; CML, chronic myelomonocytic leukemia; DM, diabetes mellitus; ESRD, end-stage renal disease; HA, health-care-associated infection; LAMA, left against medical advice; SD, surgical debridement; PVD, peripheral vascular disease; SLE, systemic lupus erythematosus; T2DM, type II diabetes mellitus. (Adapted from Roberts et al. [13]).

Timely intervention and disease outcome significantly hinge on early recognition through high suspicion and appropriate management strategies, including prompt broad-spectrum empiric antibiotic treatment, surgical exploration involving debridement, and necessary hemodynamic support. [15] When diagnosis is uncertain, an early soft tissue biopsy on frozen sections can offer a definitive answer [8].

Recommended antibiotic protocols before identifying specific organism(s) involve a carbapenem or a beta-lactam with a beta-lactamase inhibitor, coupled with clindamycin for its antitoxin effects. Additionally, coverage against methicillin-resistant *S. aureus* is advised [16].

Most patients with necrotizing fasciitis exhibit underlying comorbidities like diabetes, immunosuppression, and alcoholic liver disease, which predispose them to soft tissue infections. [7] Initially, patients may lack systemic symptoms, particularly if they have such comorbidities. These conditions can lead to a subdued immune response, masking even severe infections. However, to the best of our knowledge, our patient was not immunocompromised, had no history of steroid use, was not diabetic, had no relevant comorbidities, had a negative CT scan for tumors, had no history of neutropenia nor had he ever had a history of alterations in blood components. So, as far as we could verify and by taking an accurate past and present medical history, our patient was immunocompetent. Healthy patients, like our, often delay seeking treatment and receive less intensive care.

In addition to the particularity of the case of monomicrobial fasciitis due to *Serratia*, the initial biopsies taken during the first surgery showed muscle necrosis, without any involvement of the fascia. This condition appears to conflict with the definition of necrotizing fasciitis, which involves necrosis of fascia and subcutaneous tissue, leaving muscle and dermal tissue unaffected. However, necrosis of the muscle fascia was also demonstrated in subsequent biopsies taken a few hours after the previous ones. The distinct pathological findings observed in the first biopsies might stem from early biopsy sampling when the necrosis was not yet extensive, or from samplings that did not involve the areas most affected by the necrosis.

Another factor that may have led to poor patient outcome may be the presence of bacteraemia. In fact, the presence of bacteraemia has been previously recognized as a predisposing factor for poor outcomes. [1] It is undoubtedly understandable that the greater the invasion, including haematogenous involvement, the greater the systemic compromise, with the greater probability of an unfortunate outcome.

Conclusion

We presented a very rare fatal case of necrotizing fasciitis due to a monomicrobial infection by *Serratia marcescens* in an immunocompetent patient. Even if our patient came to the hospital quickly and was timely and aggressively treated, the evolution was fulminant, although the presenting clinic represented a stage 1 (early) infection.

The rapid and severe progression of the patient's condition contributes to the distinctive nature of this case, which should lead clinicians to consider necrotizing fasciitis even in those patients

without risk factors and with an early clinical presentation.

Declarations

Contributors: NN: involved in clinical care of the patient, literature search, preparation of tables, and drafting the manuscript. MC, PM, CB, AP: involved in clinical care of the patient and supervising the study. Contributed substantially to editing, revising, and finalizing the manuscript before submission. All authors have reviewed and agreed on the final manuscript.

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