Necrotizing fasciitis of the abdominal wall caused by Serratia marcescens

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Abstract

In this article, we present the first case of necrotizing fasciitis affecting the abdominal wall caused by Serratia marcescens and share results of a focused review of S. marcescens induced necrotizing fasciitis. Our patient underwent aorto-femoral bypass grafting for advanced peripheral vascular disease and presented 3 weeks postoperatively with pain, erythema and discharge from the incision site in the left lower abdominal wall and underwent multiple debridement of the affected area. Pathology of debrided tissue indicated extensive necrosis involving the adipose tissue, fascia and skeletal muscle. Wound cultures were positive for Serratia marcescens. She was successfully treated with antibiotics and multiple surgical debridements. Since necrotizing fasciitis is a medical and surgical emergency, it is critical to examine infectivity trends, clinical characteristics in its causative spectrum. Using PubMed we found 17 published cases of necrotizing fasciitis caused by Serratia marcescens, and then analyzed patterns among those cases. Serratia marcescens is prominent in the community and hospital settings, and information on infection presentations, risk factors, characteristics, treatment, course, and complications as provided through this study can help identify cases earlier and mitigate poor outcomes. Patients with positive blood cultures and those patients where surgical intervention was not provided or delayed had a higher mortality. Surgical intervention is a definite way to establish the diagnosis of necrotizing infection and differentiate it from other entities.

Introduction

Necrotizing fasciitis, also known as flesh-eating disease, is a rare infection of the deeper layers of skin and subcutaneous tissues, and easily spreads across the fascial plane within those tissues. As bacterial toxins and the immune response cause vasocostriction of the vasculature, the fascial spaces become avascular resulting in necrosis, which also prevents penetration of antibiotics into the tissues.1 Common causes are Group A streptococcus (GAS) (Streptococcus pyogenes), Staphylococcus aureus, Vibrio vulnificus, Clostridium perfringens, and Bacteroides fragilis. Mortality ranges from 4.2 to 38% with improving prognosis as time to treatment decreases.2 Serratia marcescens, a motile bacillus, gram-negative, facultative anaerobe, is an opportunistic pathogen of increasing importance. It is part of normal colon flora, and is also found in soil, sewage, and water. This Enterobacteriaceae organism also typically colonizes the respiratory and urinary tracts and causes infections in those organ systems.3 S. marcescens is often an opportunistic infection and may cause osteomyelitis, septic arthritis, endocarditis, and, rarely cellulitis or necrotizing fasciitis.4 Soft tissue infections due to gram-negative organisms are relatively uncommon, and typical predisposing factors include: a history of trauma, alcoholism, peripheral vascular disease, systemic lupus erythematosus, immunosuppression, diabetes mellitus, urinary tract infection (UTI), bacteremia, pneumonia, infective arthritis, burns, and renal failure.5 Other predisposing factors include: antibiotic use (most often first generation cephalosporins), steroid use, surgical instrumentation, urinary catheters, respiratory equipment, intravenous lines, injections, lacerations, abscesses, or ulcers.6

Common presentations of necrotizing fasciitis within 48 hours of infection include skin erythema and swelling at the affected site (97.6%), pyrexia (61.9%), hypotension (33.3%), altered consciousness (28.6%), bulbar lesions (26.2%), and crepitus (9.5%). Hypotension, altered consciousness, ventilator support, ALT > two-fold of normal, serum creatinine >177 µmol/L, thrombocytopenia (<100×109/L), and worsening symptoms within 48 hours of admission have been associated with higher fatality rates.7 Lack of response to narrow-spectrum antibiotics, bullae formation, or a rapidly worsening clinical course, should heighten the suspicion for uncommon organisms like S. marcescens.

Reports of S. marcescens necrotizing fasciitis cases have increased in the literature. In this study, we identify characteristics, trends, and risk factors of those infections to better prepare the medical community and prevent poor outcomes.

Materials and Methods

PubMed was used to search for cases of necrotizing fasciitis caused by Serratia marcescens published in the English language literature between 1966 and 2013. Keywords that were used included: Serratia marcescens, necrotizing, and fasciitis. We identified eleven additional cases of necrotizing fasciitis due to S. marcescens in the literature since the latest review in 2001, which are included in Table 1.4,7,14-22 Cases were categorized as being healthcare-associated infections or community-acquired infections based on the Centers for Disease Control and Prevention (CDC) definition of healthcare-associated infections (HAIs) as infections that patients acquire during the course of receiving healthcare treatment for other conditions.23

Case Report

We recently identified a case involving a 51-year-old African-American woman who pre-
sented 3 weeks postoperatively after undergoing a bifemoral bypass and left distal femoral aneurysm repair for symptomatic peripheral vascular disease. She had a long standing history of smoking and hypertension. On presentation, the patient reported having fever, chills, nausea, vomiting, and diarrhea for 6 days, redness and odorous discharge from her surgical incision site for one day. Additional history revealed that she was cleaning her wound with well water. On examination, there was erythema extending from the periumbilical area to her left groin, along with severe tenderness to palpation in that area. No crepitus was felt on palpation. Her temperature on admission was 36.9°C, blood pressure was 162/109 mmHg, respirations were 24 breaths per minute, and she was in no acute distress. Her white blood cell count was 9.8×10^9/L, C reactive protein (CRP) was 16.57 nmol/L, serum creatine kinase level was 57 U/L, serum electrolytes, renal, liver functions and immunoglobulin levels were normal and blood and urine cultures were negative. The patient was started on intravenous vancomycin and meropenem and underwent multiple debridements of the abdominal wound and left groin region. Intraoperative findings confirmed the presence of deep necrotic fascia and non-adherent subcutaneous tissue. Histopathology of debrided tissue indicated acute and chronic inflammation with infiltration of granulocytes and necrosis involving the fibroadipose tissue and skeletal muscle. Intraoperative wound cultures were positive for Serratia marcescens. Based on sensitivities, antibiotics were changed to intravenous levofloxacin MIC <2 mcg/mL. She also received negative-pressure wound therapy using vacuum assisted closure. The patient improved clinically and was successfully discharged on oral levofloxacin to complete a total of 3 weeks of treatment.

## Results

Since 1966, there have been 17 documented cases of necrotizing fasciitis caused by S. marcescens (Table 1). Eight (47%) of those cases occurred within the past five years. Among all of the identified cases, 10 (59%) impacted a leg, and 1 (5.9%) affected the forearm, the cervical spine, the axilla and chest wall, a venous access site, the oropharynx, the chest wall, or the lower abdomen. Thirteen (77%) of the cases had blood cultures that were positive for S. marcescens. Ten cases (59%) were community-acquired infections, 9 (53%) involved surgical sites, 6 (36%) involved traumatic wounds, and 4 (24%) involved vascular access devices.

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Age</th>
<th>Sex</th>
<th>Risk</th>
<th>Precipitating factor</th>
<th>Site of infection</th>
<th>Type</th>
<th>S. marcescens cultures</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rimailho et al.13</td>
<td>1987</td>
<td>74</td>
<td>M</td>
<td>Immuno-compromized</td>
<td>Dilofencan consumption</td>
<td>Leg</td>
<td>CA</td>
<td>Blister and blood</td>
<td>None</td>
<td>Died</td>
</tr>
<tr>
<td>Bornstein et al.11</td>
<td>1992</td>
<td>77</td>
<td>F</td>
<td>Renal failure</td>
<td>Pain during dialysis</td>
<td>Axilla and chest wall</td>
<td>HA</td>
<td>Wound, bullae, blood</td>
<td>Antibiotics and SD</td>
<td>Recovered</td>
</tr>
<tr>
<td>Zipper et al.12</td>
<td>1996</td>
<td>55</td>
<td>F</td>
<td>Diabetes</td>
<td>Left below-knee amputation</td>
<td>Leg</td>
<td>CA</td>
<td>Wound</td>
<td>Antibiotics</td>
<td>Recovered</td>
</tr>
<tr>
<td>Huang et al.15</td>
<td>1999</td>
<td>73</td>
<td>M</td>
<td>Nephrotic syndrome</td>
<td>Steroid therapy</td>
<td>Lower leg</td>
<td>HA</td>
<td>Necrotic tissue, blood</td>
<td>Antibiotics and SD</td>
<td>Recovered</td>
</tr>
<tr>
<td>Huang et al.15</td>
<td>1999</td>
<td>40</td>
<td>M</td>
<td>Uremia, peritoneal dialysis, SLE</td>
<td>Pneumonia with cultures for S. marcescens, steroid and nabumetone</td>
<td>Left calf and thigh</td>
<td>CA</td>
<td>Necrotic tissue, blood</td>
<td>Antibiotics and SD</td>
<td>Recovered</td>
</tr>
<tr>
<td>Liang et al.15</td>
<td>2001</td>
<td>66</td>
<td>F</td>
<td>Healthy</td>
<td>None</td>
<td>Leg</td>
<td>CA</td>
<td>Blood</td>
<td>Antibiotics</td>
<td>Died</td>
</tr>
<tr>
<td>Newton et al.15</td>
<td>2002</td>
<td>2</td>
<td>F</td>
<td>Healthy</td>
<td>Pharyngitis</td>
<td>Cervical spine</td>
<td>CA</td>
<td>Wound, blood</td>
<td>Antibiotics and SD</td>
<td>Died</td>
</tr>
<tr>
<td>Bachmeyer et al.14</td>
<td>2004</td>
<td>49</td>
<td>M</td>
<td>Small cell lung cancer, DM</td>
<td>Chemotherapy and cellitis</td>
<td>Right leg</td>
<td>HA</td>
<td>Tissue, bullae, blood</td>
<td>Antibiotics</td>
<td>Recovered</td>
</tr>
<tr>
<td>Curtis et al.1</td>
<td>2005</td>
<td>51</td>
<td>M</td>
<td>ESRD, T2DM, CHF</td>
<td>Scraped knee on rock in river</td>
<td>Left leg</td>
<td>CA</td>
<td>Wound</td>
<td>Antibiotics and SD</td>
<td>Died</td>
</tr>
<tr>
<td>Statham et al.17</td>
<td>2009</td>
<td>6</td>
<td>M</td>
<td>Immunocompetent</td>
<td>Suspected pharyngitis</td>
<td>Oro-pharynx</td>
<td>CA</td>
<td>Wound, blood</td>
<td>Antibiotics and SD</td>
<td>Recovered</td>
</tr>
<tr>
<td>Motitsi et al.18</td>
<td>2011</td>
<td>37</td>
<td>M</td>
<td>Healthy</td>
<td>Human bite</td>
<td>Forearm</td>
<td>CA</td>
<td>Wound</td>
<td>SD</td>
<td>Died</td>
</tr>
<tr>
<td>Naito-Galvan et al.7</td>
<td>2012</td>
<td>57</td>
<td>F</td>
<td>CML, immunocompromized</td>
<td>Minor trauma</td>
<td>Right thigh</td>
<td>HA</td>
<td>Blister, blood</td>
<td>Antibiotics</td>
<td>Died</td>
</tr>
<tr>
<td>Prelog et al.19</td>
<td>2012</td>
<td>15</td>
<td>F</td>
<td>Acute lymphocytic leukemia</td>
<td>Venous access port implantation</td>
<td>Left axilla, venous HA access port site</td>
<td>Wound</td>
<td>Antibiotics and SD</td>
<td>Recovered</td>
<td></td>
</tr>
<tr>
<td>Wen et al.19</td>
<td>2012</td>
<td>40</td>
<td>F</td>
<td>Nephrotic syndrome, cyclosporine use</td>
<td>Chemotherapy 10 days prior</td>
<td>Left leg</td>
<td>CA</td>
<td>Wound, blood</td>
<td>Antibiotics</td>
<td>Died</td>
</tr>
<tr>
<td>Rehman et al.11</td>
<td>2012</td>
<td>54</td>
<td>F</td>
<td>SLE, end-stage renal disease</td>
<td>Central venous catheter, AV fistula Iigation, steroid therapy</td>
<td>Chest wall</td>
<td>HA</td>
<td>Wound, blood</td>
<td>Antibiotics and SD</td>
<td>Died</td>
</tr>
<tr>
<td>Present case</td>
<td>2013</td>
<td>97</td>
<td>F</td>
<td>Heat failure, CKD</td>
<td>Heart failure exacerbation</td>
<td>Right leg</td>
<td>HA</td>
<td>Wound (post-mortem)</td>
<td>Antibiotics</td>
<td>Died</td>
</tr>
</tbody>
</table>

3D, Surgical debridement; CA, community-acquired infection; HA, healthcare-associated infection; SLE, systemic lupus erythematosus; ESRD, end-stage renal disease; T2DM, Type II diabetes mellitus; PVD, peripheral vascular disease.
and 7 cases (41%) were healthcare-associated infections. An overwhelming number of cases (81.3%) had pre-existing open wounds. Seven cases (41%) were immunocompromized, 5 cases (29%) had kidney disease, and 4 cases (24%) had diabetes. A majority (59%) of the cases were among females. Three of the cases (18%) were children.

Nine out of seventeen cases (53%) died as a result of necrotizing fasciitis and its complications. Six of the cases (67%) that died had community-acquired infections. Individuals with positive versus negative blood cultures were more likely to die (88% vs. 75%). Patients who did not receive surgical debridement had inferior outcomes; they had a mortality of 71% compared to 40% among those who received surgical intervention as opposed to debridement. Additionally, all patients (18%, n=3) who received only antibiotics and the one patient who received only surgical debridement died. All previously healthy patients (18%, n=3) also died. A majority of those that recovered had a surgical procedure in the hospital prior to symptom onset such as below the knee amputation or venous access port implantation (75%, n=8).

Discussion and Conclusions

Necrotizing fasciitis is a deep infection of the subcutaneous tissue that results in progressive destruction of fascia and fat. The disease is classified as type I (polymicrobial infection), type II (monomicrobial) and type III gas gangrene, or clostridial myonecrosis. Type I infection involves anaerobic species in combination with one or more facultative anaerobic streptococci (other than group A) and members of the Enterobacteriaceae family. Type II infection is commonly caused by group A streptococci or other beta-hemolytic streptococci that are isolated alone or in combination with one or more facultative anaerobic streptococci. Type III infection involves a broad spectrum of bacteria, including anaerobic species such as Bacteroides fragilis and Peptostreptococcus species.

The disease is marked by rapid progression, fever, hypotension, decreased pulse pressure, and respiratory distress. Necrotizing fasciitis is often confused with cellulitis, and the diagnosis can be challenging due to its heterogeneous presentation. Patients with necrotizing fasciitis typically present with pain, swelling, erythema, and rapid progression of symptoms.

Necrotizing fasciitis is a medical emergency and requires prompt diagnosis and aggressive treatment. Early recognition and timely surgical intervention are crucial for survival. Patients with necrotizing fasciitis are at risk for complications such as sepsis, multiorgan failure, and death.

References


