**Serratia marcescens Rhabdomyolysis**

Miguel F. Carrascosa¹, José R. Salcines-Caviedes¹, María Carmen Fariñas²

¹Internal Medicine Department, Hospital of Laredo, Laredo, Spain; ²Infectious Diseases Unit, University Hospital Marqués de Valdecilla, University of Cantabria, Santander, Spain.

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**ABSTRACT**

*Serratia marcescens* has been recognized as an important cause of nosocomial and community-acquired infections. To our knowledge, we describe the first case of *S. marcescens* rhabdomyolysis, most probably related to acute cholecystitis and secondary bacteremia. The condition was successfully managed with levofloxacin. Keeping in mind the relevant morbidity and mortality associated with bacterial rhabdomyolysis, physicians should consider this possibility in patients with suspected or proven bacterial disease. We suggest *S. marcescens* should be regarded as a new causative agent of infectious rhabdomyolysis.

**Keywords:** Acute Cholecystitis; Acute Renal Failure; Myositis; Rhabdomyolysis; *Serratia marcescens*

1. **Introduction**

*Serratia marcescens* has been recognized as an important cause of nosocomial and community-acquired infections [1]. This pathogen is capable of producing a broad spectrum of human disease, including wound infections, pneumonia, meningitis,ocular and urinary tract infections, bacteremia, and endocarditis [1]. Besides, some musculoskeletal and joint syndromes have been attributed to it, bone and articular infections being the most frequent of them (Table 1) [2-8]. To our knowledge, we describe herein the first case of rhabdomyolysis due to *S. marcescens*.

2. **Case Report**

A 79-year-old man presented to our emergency department because of a 2-day history of malaise, asthenia, nausea, and fever. He denied having myalgias, abdominal or chest pain, or vomiting. His past medical history was relevant only for smoking (18 packs per year) and alcohol intake (20 gr ethanol per day). Specifically, there was no evidence of previous neuromuscular disorders, epilepsy, metabolic abnormalities, inflammatory or autoimmune muscle diseases, or recent traumatism or exposure to hypothermia. He was receiving no medications. His temperature was 39°C, blood pressure was 142/77 mm Hg, pulse rate was 114, and respiratory rate was 22. The arterial oxygen saturation while breathing on room air was 95%. Physical examination was notable for bilateral, scarce rhonchi and rales. Interestingly, there were no findings indicative of acute arterial occlusion or compartment syndrome. Laboratory studies disclosed the following significant data: BUN 24.7 mg/dL; creatinine, 0.71 mg/dL; WBCs, 18,800/μL (17,400 polymorphonuclear neutrophils); normal hemoglobin, hematocrit, and platelet count; CPK, 3,627 U/L (normal level, 33 - 185); troponin T, 184 ng/L (<14); lactate dehydrogenase, 800 U/L; aspartate aminotransferase, 198; alanine aminotransferase, 213 U/L; alkaline phosphatase, 138; gammaglutamyltransferase, 555 U/L; C-reactive protein, 6.57 mg/dL (<0.5); normal INR and partial thromboplastin time. The urine was positive for proteins but the presence of myoglobin was not assessed. Chest radiograph showed normal findings and an electrocardiogram revealed sinus tachycardia. Abdominal ultrasonography displayed mult...

**Table 1. Musculoskeletal and articular involvement in Serratia marcescens disease.**

<table>
<thead>
<tr>
<th>References</th>
<th>Type of Infection</th>
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<tbody>
<tr>
<td>[2,3]</td>
<td>Myositis (pyomyositis, necrotizing myositis)</td>
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<tr>
<td>[4]</td>
<td>Necrotizing fasciitis</td>
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<tr>
<td>[5]</td>
<td>Osteomyelitis</td>
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<td>[6]</td>
<td>Spondylodiscitis</td>
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<td>[7]</td>
<td>Septic arthritis in native joint</td>
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<td>[8]</td>
<td>Prosthetic joint infection</td>
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<tr>
<td>[Present report]</td>
<td>Rhabdomyolysis</td>
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ultiple cholelithiasis with slight gallbladder enlargement. Echocardiogram and cardiac magnetic resonance imaging were unremarkable. Serologic studies were negative for HIV, HAV, HBV, HCV, Epstein-Barr virus, varicella-zoster virus, herpes simplex virus, cytomegalovirus, Legionella pneumophila, Mycoplasma pneumoniae, and Coxiella burnetii. PCR analysis for influenza virus type A and B from two nasopharyngeal swab specimens was also negative.

Blood and urine samples were obtained for culture in the emergency room and therapy with intravenous levofloxacin (500 mg twice a day for two days, then 500 mg daily) was started. On the second hospital day, serum CK and troponin T level raised to 6,476 U/L and 60 ng/L, respectively. Blood cultures (two bottles on day 2) became positive for S. marcescens, which was sensitive to quinolones. The urine culture showed no growth. He became afebrile within 48 hours and the rest of the hospital course was uneventful. When the patient was discharged asymptomatic 8 days after admission, his serum CK concentration had decreased to 228 U/L. At a follow-up visit 1 month after discharge, he remained well and laboratory studies (including CK and troponin T values, liver function tests, and convalescent serologic assessment) revealed normal results. At a second follow-up evaluation 6 months after discharge, he went on symptoms free.

3. Discussion

Our patient developed S. marcescens bacteremia and rhabdomyolysis, most probably related to acute cholecystitis. Rhabdomyolysis has been associated with multiple etiologic agents, including viral and bacterial infections [9,10]. It should be distinguished from pyomyositis and myonecrosis in that there is no evidence of abscesses or localized site of muscle lesion [10]. Among bacteria, Legionella species, Streptococcus species, Francisella tularensis, and Salmonella species are the most commonly identified causes.

Although the underlying mechanism for rhabdomyolysis by S. marcescens is unknown, toxin generation and/or direct bacterial invasion of skeletal muscle could be implicated. Keeping in mind the relevant morbidity (acute renal failure in 57% of patients) and mortality (death in 38% of cases) associated with bacterial rhabdomyolysis [10], physicians should consider this possibility in patients with suspected or proven bacterial disease.

4. Conclusion

We suggest S. marcescens should be regarded as a new causative agent of infectious rhabdomyolysis.

REFERENCES