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Case Report

A Case of Cutaneous Loxoscelism Associated to Fever: Emergency Care Considerations

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Received: February 16, 2023; **Published:** February 18, 2023

Abstract

Cutaneous lesions with necrotic eschar may be manifestation of a wide range of pathologies - from chronic fungal infection, tertiary syphilis, leprosy, leishmaniasis or Rickettsial vasculitis to *Loxosceles* spp. bite. The diagnosis of cutaneous loxoscelism in the absence of an insect bite exposure history is clinical, excluding other causes in base of the age, patient's general conditions and eventual corresponding geographic and endemic area. The association between loxoscelism and cutaneous lesions is known, the fever is considered a symptom of systemic loxoscelism.

Keywords: *Loxosceles spp. Bite; Necrotic Eschar; Erythematous Painful Lesions; Cutaneous Loxoscelism; Systemic Loxoscelism*

Introduction

Loxoscelism is a rare illness resulting from the bite of the spider of *Loxosceles* species, distributed worldwide. In Italy the *Loxosceles rufescens* is native [1]. The species is reported in the Mediterranean area, especially in southern Europe, North Africa, Turkey, Iran [2].

Initial manifestation of mild form, alternatively denominated cutaneous loxoscelism, range from minimal skin lesion, skin erythematous rash, ecchymosis, cellulitis, abscess, which may progress to necrotic eschar or necrotic arachnidism. The systemic loxoscelism is associated to fever and arthralgia, the most severe form may lead to hemolysis, acute kidney injury, coagulopathy, and death [3].

According to the data of Italian Toxicology and Poison Center about 1% of patients with cutaneous loxoscelism evolve in systemic form [1].

The other species of *Loxosceles* documented to produce necrotic bites are *L. arizonica, L. deserta, L. laeta* and *L. reclusa*, or the specie of *Latrodectus. Loxosceles* venom contains hyaluronidase, alkaline phosphatase, 5-ribonucleotide phosphohydrolase, and especially sphingomyelinase D. Sphingomyelinase D causes release of choline and N-acylsphingosine phosphate from red blood cell membrane, which stimulates platelet aggregation and dermonecrosis [4], responsible of the cutaneous lesion with necrotic eschar.

Case Report

A 16-year-old male presented to the emergency department with a 2-day history of cutaneous rash on his left leg and fever without other systemic symptoms. The medical history was not significative, no exposure to animals or insect was confirmed.

On physical examination patient was apyretic, presenting suspected initial abscess lesion of diameter of about 5 cm, characterized by redness, pain, and soft swelling of the left leg in tibial region, without demarcation line. Physical examination was negative for lymphadenopathy, organomegaly, or other cutaneous manifestations. Nasopharyngeal swab for SARS-CoV-2 was negative. The patient was discharged home with oral antibiotic treatment with amoxycillin/clavulanic acid, to be followed by his general practitioner.

Six days later he represented to the emergency department with the erythematous slightly elevated lesion with necrotic eschar with sharp margins, that measured approximately 2 cm x 2 cm. Escharectomy was performed with the leakage of abundant purulent and necrotic material. Residual cavity deepened into the subcutaneous for about 1 cm without apparent involvement of the bone plane. The laboratory studies were within the reference range: white blood cell count of 8.84 K/µL (Reference Range 4.00 - 11.00 K/µL), hemoglobin of 14.4 g/dL (RR 13.0 - 18.0 g/dL), platelets of 237 K/µL (RR 130 - 400 K/µL), PCR of 0,66 mg/dL (RR 0.0 - 0.8 mg/dL), total bilirubin of 0.4 mg/dL (RR 0.1 - 1.3 mg/dL), INR 1.02 (RR 0.9 - 1.27 mg/dL), creatinine 0.72 mg/dL (RR 0.5 - 1.3 mg/dL). The leukocyte differential and absolute neutrophil count were within the reference range. Left leg x-rays showed no focal bone lesions. Urine dipstick was negative.

The photos of the lesion were submitted to toxicologist and arachnologist for evaluation and infectious disease specialist consulted. Excluding wide range of pathologies presenting erythematous painful lesions, abscesses, ulcers, or necrotic eschars we focused on cutaneous loxoscelism.

Considered poor clinical benefit from administration of amoxycillin/clavulanic acid, we administered as infectious diseases specialist indicated. The aim was to prescribe an extremely effective drug able to avoid or reduce the hospitalization costs and the patient's convalescence. This drug is a lipoglycopeptide recently approved as antibiotic and antimycotic, suitable for gram-positive skin and soft tissue infection in adult patients, including *Streptococcus pyogenes* and methicillin-resistant *Staphylococcus aureus*. The patient's conditions allowed a full-dose treatment consisting of 1000 mg diluted in 500 ml glucose solution at 5% concentration intravenous on the first day of treatment. In 7 days a second dose was provided with a posology reduced to 500 mg EV. Dalbavancin efficacy and long half-life (147/258 hours) [5] allowed a safe and effective treatment of the patient without the need of hospitalization and patient outcome.

The 13th day after the first evaluation in ED the young patient was revisited. The laboratory exams remained within the reference range, especially renal function, inflammatory markers, blood count, bilirubin. Hemolysis and hematuria have never appeared. Wound swab resulted negative. Patient remained apyretic since the first evaluation.

The 42th day the young patient presented a little, 2 mm per 2 mm, slightly excavated non complicated eschar.

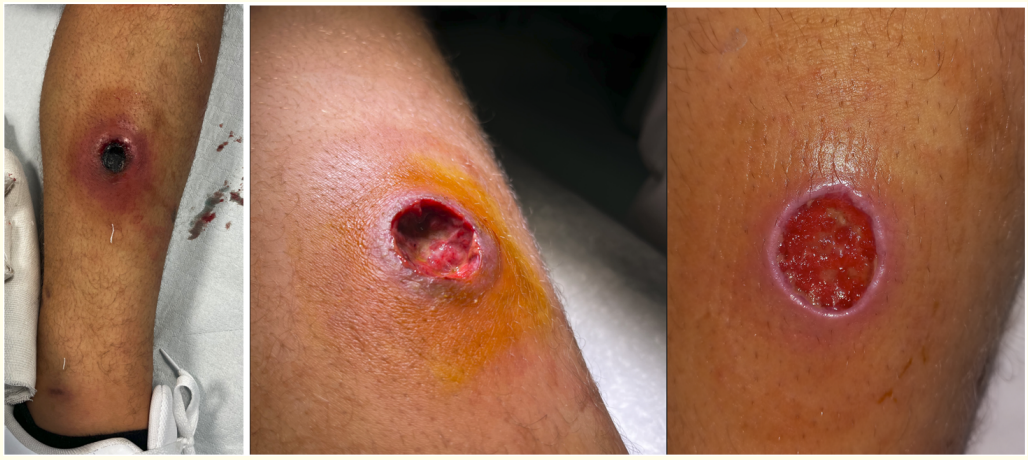

Figure 1: *Susp. Loxosceles spp. bite before and after escharectomy with wound debridement. On the third picture the same lesion the 13th day after the first evaluation in ED: clean bottom with granulation tissue, little areas of fibrin slough and objective improvement. Granulation tissue matrix will fill the wound that heal by second intention.*

Discussion

Phenome-wide association study confirmed associations of systemic loxoscelism with other phenotypes, for example rash, hemolytic anemia, haemoglobinuria, acute renal failure, disseminated intravascular coagulation, septicemia, sepsis and other. Hemolysis reaches a peak at 2 days after admission [6].

The underlying pathogenesis of systemic loxoscelism was not completely understood, but sphingomyelinase D is known to cause both direct toxin-mediated hemolysis and complement-mediated erythrocyte destruction [7].

In some cases of *Loxosceles reclusa* envenomation with acute hemolytic anemia in adolescent population the positive direct Coomb's test was described [8].

Lesions with necrotic eschar may be seen in chromoblastomycosis, a chronic subcutaneous fungal infection, endemic in tropical and subtropical climates or in immunocompromised patients [9].

Other rare causes of growth with necrotic eschar include for example tertiary syphilis, leprosy, leishmaniasis or Rickettsial vasculitis [10]. None of these was considerable in our 16-years old patient.

In endemic areas, suspicion for systemic loxoscelism should be high in individuals, especially children and younger adults, presenting with a cutaneous ulcer and hemolysis and/or coagulopathy, even in the absence of an insect bite exposure history [6].

Conclusion

The association between loxoscelism and superficial cellulitis or abscess is known, the fever is considered a pre-monitor symptom of systemic loxoscelism. The diagnosis of cutaneous loxoscelism in the absence of an insect bite exposure history is clinical, excluding other

causes in base of the age, patient's general conditions and corresponding geographic and endemic area. Therapy is symptomatic, including escharectomy of the delimited lesion, antibiotic prophylaxis of bacterial infection and correct wound medication.

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Volume 7 Issue 3 March 2023