

Cutaneous Loxoscelism Complicated by Tissue Necrosis: A Case Report and Surgical Resolution

Dr. Erik Daniel Hernández Soto^{*1}, Dra. María Fernanda Ramírez Berumen², Dra. María Del Rayo Méndez Palacios³, Dr. Manuel Rodríguez Navarro⁴

¹Unidad Médica de Alta Especialidad No. 25, Centro Médico Nacional Noreste, IMSS

²IMSS Centro Médico Nacional Siglo XXI

³Universidad Popular Autónoma Del Estado De Puebla

⁴Instituto de Seguridad y Servicios Sociales de los Trabajadores al Servicio de los Poderes del Estado de Puebla

ABSTRACT

Introduction: Loxoscelism is a potentially lethal accidental envenomation mediated by the enzyme sphingomyelinase D, which can trigger manifestations ranging from local skin necrosis to massive intravascular hemolysis, coagulopathy, and multiple organ failure (cutaneous-visceral loxoscelism). In geriatric patients, the initial diagnosis represents a critical challenge because early systemic signs and wet epidermolysis can mimic severe drug-induced dermatosis like Lyell's syndrome, delaying specific therapy. **Objective:** To describe the clinical course, multisystemic paraclinical alterations, and the success of a stepped medical-surgical approach in a geriatric patient with severe cutaneous-visceral loxoscelism in Mexico. **Case Presentation:** A 67-year-old male patient presented to the emergency department in shock, with a 24-hour evolution after noticing a blister on the fifth finger of his left hand. Clinically, he developed progressive violaceous edema in the left upper extremity, blisters with necrotic changes, somnolence, dyspnea, chest pain, and oxygen desaturation down to 80% on room air. Baseline laboratories revealed metabolic acidosis, acute kidney injury (creatinine 1.9 mg/dL), massive microhematuria, and critical elevation of cardiac and molecular stress biomarkers (NT-proBNP 11,800 pg/mL, myoglobin 350 ng/mL, D-dimer 6,038 ng/mL), suggesting disseminated intravascular coagulation. After ruling out pulmonary embolism via computed tomography angiography, he was managed in the Intensive Care Unit, where antiloxosceles fabotherapy (Reclusmyn®) was administered alongside goal-directed fluid resuscitation, a dual antibiotic regimen (cefepime/clindamycin), and thromboprophylaxis. Within 72 hours, organ dysfunction resolved, and renal function normalized (creatinine 0.5 mg/dL), leaving defined areas of epidermolysis and dermonecrosis. On the 19th day of intrahospital stay, with stable laboratories, a surgical wash, debridement, and fasciotomy of the left hand with a tie-over graft placement were electively performed, achieving satisfactory healing and hospital discharge the following day. **Conclusions:** Systemic loxoscelism in the elderly requires high clinical suspicion to differentiate it from toxic-allergic dermatological syndromes. Timely initiation of specific fabotherapy and intensive support within the early therapeutic windows halt the progression of renal failure and vascular collapse, optimizing subsequent reconstructive outcomes.

KEYWORDS: Loxoscelism, Loxosceles reclusa, Sphingomyelinase D, Acute Kidney Injury, Fabotherapy.

ARTICLE DETAILS

Published On:
11 July 2026

Available on:
<https://ijmscrs.com>

I. INTRODUCTION

Arachnidism or envenomation caused by spider bites represents a significant reason for consultation within

emergency services in tropical and temperate regions worldwide. Within the Araneae order, a small proportion of species (approximately 0.4%) possesses a venom with

Cutaneous Loxoscelism Complicated by Tissue Necrosis: A Case Report and Surgical Resolution

sufficient toxicity to compromise hemodynamic stability or the cutaneous integrity of human beings. Among the clinical syndromes provoked by arachnids that carry high morbidity and mortality and configure true medical emergencies, loxoscelism stands out, a pathological entity derived from the inoculation of toxins by specimens of the *Loxosceles* genus. This clinical phenomenon usually occurs due to chance and unprovoked encounters between humans and the arthropod, which reacts defensively when pressed against the victim's skin.¹

The geographic distribution of the *Loxosceles* genus is cosmopolitan, finding its greatest taxonomic diversity in the American continent. The Mexican Republic is considered one of the regions with the highest biological richness of this arachnid, harboring approximately 40 described species of which the vast majority are native to its territory. Specimens belonging to this genus are colloquially known in the national territory as "violin spiders," "brown spiders," or "corner spiders" due to subtle morphological features, such as the presence of a dark spot in the shape of an inverted violin on the dorsal portion of their cephalothorax. Additionally, the genus is biologically distinguished by possessing six eyes organized in three dyads, a characteristic ocular arrangement that is of vital importance for its taxonomic identification and differentiation from other non-dangerous spiders.²

From an ecological and behavioral perspective, species of the *Loxosceles* genus are characterized by being sedentary, solitary, and markedly nocturnal arachnids. They have a special predilection for sheltering in dry, dark environments with scarce mechanical ventilation, which confers troglomorphic capabilities upon them. However, population growth and uncontrolled urbanization have led these spiders to develop a high synanthropic capacity, successfully adapting to the human intradomiciliary environment. Consequently, they are frequently located behind furniture, paintings, closets, or accumulations of old clothing, increasing the risk of direct contact with the household inhabitants and facilitating their dispersion in densely populated areas.²

Scorpiotoxycosis and envenomation by poisonous arthropods share common epidemiological channels in Mexico, reporting significant variations associated with the seasons of the year. In the context of loxoscelism, the incidence of bites begins to be reported more frequently starting in April and experiences a substantial increase during the period from June to October, which fully coincides with the rainy season in most of the national territory. At the institutional level, the National Epidemiological Surveillance System globally registers from 3,000 to 5,000 annual cases due to bites from spiders of the *Loxosceles* and *Latrodectus* genera, with a mortality rate from loxoscelism of up to 10% in the pediatric and geriatric population in the absence of timely intervention.⁴

The pathophysiology of loxoscelic intoxication is dependent on a sophisticated hydrolytic mixture of proteins

and low-molecular-weight peptides synthesized in the apocrine glands of the arachnid's mouthparts. Among the isolated enzymatic components are hyaluronidases, metalloproteinases, collagenases, and deoxyribonucleases that act synergistically, facilitating the dispersion of the venom into adjacent tissues. However, the central toxin, considered the main virulence factor and directly responsible for tissue damage, is phospholipase D (formerly classified as sphingomyelinase D). This enzyme specifically hydrolyzes the sphingomyelin of cell membranes and the glycoproteins of the vascular endothelium, secondarily activating the coagulation cascade, inducing neutrophil chemotaxis, promoting keratinocyte apoptosis, and stimulating the activation of the complement system.⁵

At the histopathological level, the interaction of phospholipase D with the endothelium generates severe vascular alterations characterized by edema, severe vasodilation, increased thickness of endothelial cells, and degradation of the basement membrane wall. This sustained microvascular damage induces the formation of luminal microthrombi that obstruct arterioles and venules, resulting in localized tissue ischemia phenomena, dermal hemorrhage, and massive infiltration of polymorphonuclear leukocytes. The intensity and extent of this cytotoxic process depend on intrinsic biological variables of the specimen, it having been documented that female spiders produce a larger quantity of venom compared to males due to their greater weight and body volume, which significantly exacerbates the proinflammatory response and the degree of subsequent demonecrosis in the victim.⁶

The clinical consequences of the inoculation of these toxins are traditionally classified into two well-defined variants: cutaneous loxoscelism and cutaneous-visceral or systemic loxoscelism. Cutaneous loxoscelism represents the most common form of presentation, manifesting in an estimated 84% to 97% of documented cases. It is characterized by a localized evolution that begins with erythema, pruritus, and pain that progresses from moderate to severe within the first 6 to 8 hours, evolving toward the formation of a "livedoid plaque" characterized by asymmetric areas of ischemic pallor, cyanosis, and a perilesional erythematous halo (bull's-eye sign), which may precede the appearance of hemorrhagic blisters and an eventual detachable necrotic eschar.⁷

Conversely, cutaneous-visceral or systemic loxoscelism constitutes the most serious and devastating variant, usually developing within the first 12 to 48 hours following the bite in 10% to 16% of patients. This systemic form is triggered when the venom components gain access to the circulatory stream, causing a picture characterized by massive intravascular hemolysis, acute hemolytic anemia, hemoglobinuria, and severe thrombocytopenia. The massive release of hemoglobin and myoglobin, added to renal ischemia due to intense renal vasoconstriction induced by inflammatory mediators and proinflammatory cytokines,

Cutaneous Loxoscelism Complicated by Tissue Necrosis: A Case Report and Surgical Resolution

leads to the development of acute kidney injury and disseminated intravascular coagulation, which represent the main causes of death with a lethality ranging between 15% and 25% of systemic cases.⁷

The definitive diagnosis of loxoscelism represents a highly complex challenge for the first-contact physician, given that in the vast majority of emergency scenarios (up to 90%) the patient does not present the aggressive specimen for taxonomic identification. Additionally, commercial biological tests and enzyme-linked immunosorbent assays (ELISA) are not routinely available in the national territory for the detection of sphingomyelinase D in patient fluids. Because of this, the diagnosis is based exclusively on epidemiological criteria, on the exclusion of differential diagnoses through clinical tools such as the *NOT RECLUSE* mnemonic, and on the meticulous analysis of the morphological progression of the skin lesion and multisystemic biomarkers.⁸

II. CASE REPORT

The patient is a 67-year-old male with a significant medical history of long-standing occasional alcoholism, with no known chronic-degenerative comorbidities, who was under outpatient management prescribed by a private physician using ketoconazole, terbinafine, and pregabalin due to an ungual mycosis, alongside a presumptive diagnosis of prostatic growth. Additionally, as a relevant traumatic and interventional history, the patient reported having undergone multiple arthrocentesis procedures in the left knee joint within the two months prior to admission due to chronic volume increase accompanied by pain, with intra-articular corticosteroids administered during the last intervention.

The current illness began approximately 24 hours prior to hospital evaluation, when the patient, immediately upon awakening in the morning, noticed a blister located on the anterior aspect of the distal third of the left little finger, accompanied by erythema, edema, and sharp pain. Over the course of the same day, the cutaneous manifestations showed accelerated centripetal progression, with erythema and edema extending into the ipsilateral envenomated forearm, conditioning the appearance of new blisters on the anterior aspect of the distal third of the forearm. Facing this condition, the patient visited a private practitioner, who performed a puncture procedure (arthrocentesis) in the elbow joint and indicated the administration of intramuscular dexamethasone, without obtaining any clinical improvement.

During the night prior to admission, the edema and erythema progressed massively to involve the middle third of the left arm, with the sharp pain increasing to an intensity of 7/10 on the visual analog scale, and focal necrotic changes becoming evident on the surface of the previous blisters, which underwent spontaneous rupture with scarce serosanguinous discharge. A few hours later, the pain extended to the left hemithorax with sharp characteristics and an intensity of 9/10, exacerbating with respiratory

movements and digital pressure, further associating severe oxygen desaturation down to 80% on room air and progressive somnolence, which prompted his family to transfer him to the emergency department of a tertiary care hospital.

Upon arrival at the shock bay, initial vital signs reported a heart rate of 98 beats per minute, respiratory rate of 21 breaths per minute, blood pressure of 143/79 mmHg, body temperature of 36.7 °C, and an oxygen saturation of 98% following immediate placement of supplemental oxygen with a fraction of inspired oxygen (FiO₂) at 40%. On directed physical examination, the neurological system was alert, oriented in all three spheres, with a Glasgow Coma Scale score of 15 points, revealing a dry oral mucosa. The left upper extremity exhibited severe edema (+++), with the wrist measuring 23 cm in diameter, the elbow 29 cm, and the arm 32 cm; multiple blisters with necrotic and purulent serosanguinous characteristics were identified extending from the fifth finger to the proximal third of the upper limb, accompanied by local hyperthermia, ecchymosis, and hypersensitivity to touch.

Baseline emergency laboratory tests revealed a leukocyte count of 6,230/μL with marked neutrophilia of 95%, hemoglobin of 14.2 g/dL, and platelets of 229,000/μL; blood chemistry reported glucose at 99 mg/dL, urea at 107 mg/dL, blood urea nitrogen (BUN) at 50 mg/dL, and an elevated serum creatinine of 1.9 mg/dL, configuring an AKI 1 acute kidney injury (calculated glomerular filtration rate of 38 mL/min). The cardiac and molecular stress profile demonstrated a myoglobin level of 350 ng/mL, troponin I at 0.010 ng/mL, lactate dehydrogenase (LDH) at 460 IU/L, a critical increase in brain natriuretic peptide (proBNP 438.80 pg/mL / NT-proBNP 11,800 pg/mL), and a markedly elevated D-dimer at 6,038 ng/mL. The urinalysis evidenced a turbid appearance with abundant sediments, proteins at 75 mg/dL, and uncountable erythrocytes.

Given the severe elevation of D-dimer and chest pain, a computed tomography angiography of the pulmonary arteries (CTA) was performed immediately, ruling out the presence of filling defects or thrombosis within the pulmonary tree down to the subsegmental level, reporting unaltered lung parenchyma and cardiac chambers. Concurrently, the General Surgery service ruled out the integration of an acute compartment syndrome in the affected limb due to the persistence of palpable axillary, humeral, radial, and ulnar peripheral pulses, and an immediate capillary refill, initially suggesting toxic epidermal necrolysis (Lyell's syndrome) as a differential diagnosis secondary to the prior ingestion of antifungals and analgesics. However, given the rapid cutaneous progression and severe systemic biological alterations, a definitive diagnosis of severe cutaneous-visceral loxoscelism with early disseminated intravascular coagulation (DIC) data was established, moving to admit the patient to the Intensive Care Unit (ICU) for the intravenous administration of a specific antiloxosceles fabotherapy

Cutaneous Loxoscelism Complicated by Tissue Necrosis: A Case Report and Surgical Resolution

(*Reclusmyn*®), alongside goal-directed fluid resuscitation, low-molecular-weight heparin thromboprophylaxis, and a dual antibiotic regimen with cefepime and clindamycin.

After 72 hours of intensive care management, the patient evolved toward hemodynamic and metabolic stability, with complete resolution of the acute kidney injury (creatinine at 0.5 mg/dL, urea at 34.2 mg/dL, and BUN at 16 mg/dL) and adequate spontaneous urine output of up to 2.1 to 3.8 mL/kg/h. Coagulation times normalized (PT 13.1 seconds, INR 1.14, aPTT 30.7 seconds), D-dimer decreased to 3,266 ng/mL, fibrinogen was reported at 754 mg/dL, and peripheral cultures were negative for bacterial growth, leaving only mild grade I normocytic normochromic anemia (hemoglobin 11.5 g/dL) and a resolving moderate thrombocytopenia (58,000/ μ L); therefore, transfer from the ICU to the Internal Medicine and Plastic Surgery ward was determined. Finally, on the 19th day of intrahospital stay, following total delimitation of the dermal lesions and evidence of abundant peripheral granulation tissue with dry necrosis confined exclusively to the distal phalanx of the fifth finger and a detached necrotic plaque, the patient was scheduled for elective surgery by Reconstructive Plastic Surgery, successfully undergoing a surgical wash, mechanical debridement, and fasciotomy of the left hand with a tie-over graft placement, achieving an optimal immediate post-surgical evolution and definitive hospital discharge the following day with instructions for home care.

III. METHODS

The methodological design of this report was established under an observational, retrospective, and descriptive approach, focused on the exhaustive analysis of the clinical and therapeutic timeline of a geriatric patient with loxoscelic envenomation. Primary data collection was performed through a meticulous and systematic review of the electronic medical records and intrahospital evolution notes generated by the Emergency, Intensive Care, Internal Medicine, Angiology, and Reconstructive Plastic Surgery services. The pre-established inclusion criteria corresponded to patients older than 65 years with a diagnosis of severe cutaneous-visceral loxoscelism who had received specific fabotherapy within a third-level unit.

To characterize the analytical profile and monitor organ dysfunction, chemical parameters determined in blood and urine samples from the patient's admission to his definitive medical discharge were extracted and tabulated chronologically. The laboratory monitoring protocol included serial quantification of renal function via serum creatinine and blood urea nitrogen, evaluation of hemostatic status through prothrombin time, activated partial thromboplastin time, and International Normalized Ratio, and monitoring of cellular and inflammatory response through complete blood counts. Likewise, measurements of specific biomarkers of myocardial and molecular stress, such as brain natriuretic

peptide, myoglobin, lactate dehydrogenase, and D-dimer, were recorded.

The evaluation of cardiovascular and respiratory sequelae from the venom-induced autonomic storm was documented through the analysis of specialized imaging and diagnostic studies. The official report of the computed tomography angiography of the thoracic aorta and pulmonary arteries was reviewed to assess the integrity of the pulmonary vascular tree, alongside serial electrocardiographic records. Dermal and muscular tissue damage in the affected upper extremity was monitored daily through direct clinical inspection, the recording of circumferential perimeters of the wrist, elbow, and arm, and the evaluation of peripheral neurovascular compromise by measuring the Daniels scale for muscle strength, capillary refill, and pulse palpation.

The analysis of the specific pharmacological intervention was based on the precise recording of the administration times, dosage, and infusion route of the purified polyvalent antiloxosceles fabotherapy of equine origin used in the medical unit. Additionally, information concerning systemic supportive therapeutics was collected, which included goal-directed fluid therapy for hemodynamic resuscitation, the use of opioid analgesics, the indication of a dual prophylactic antibiotic regimen, and the use of low-molecular-weight heparin for antithrombotic prophylaxis. Partial and global fluid balances were strictly calculated every 24 hours in relation to urine output measured by a derivation urinary catheter.

The methodological component corresponding to the reconstructive management of dermonecrosis sequelae was analyzed by reviewing the medical notes from the Reconstructive Plastic Surgery service and the records of the intrahospital wound clinic. Advanced healing techniques used at the patient's bedside were evaluated, which included the use of topical local anesthesia, cold sharp debridement of eschars, and irrigation with antimicrobial solutions and acetic acid, along with the application of specialized dressings based on calcium alginate and silver sulfadiazine to promote tissue granulation.

The definitive surgical procedure was described by analyzing in detail the preoperative note, the surgical technique executed in the main operating room, and the immediate postoperative note. The surgical variables recorded corresponded to the type of anesthesia used, the macroscopic findings of esfacelar and necrotic tissue, the extent of the debridement and fasciotomy in the left hand, as well as the characteristics of tissue reconstruction through the placement of petrolatum gauze, sterile mechanical compression, and fixation with specialized tie-over grafts or dressings.

Finally, the ethical aspects of the research were governed in accordance with the principles established in the Declaration of Helsinki and the current legal regulations for health research in the national territory. Absolute confidentiality of the patient's identity was guaranteed

Cutaneous Loxoscelism Complicated by Tissue Necrosis: A Case Report and Surgical Resolution

through a strict anonymization process in the database, removing first names, initials, specific medical record numbers, exact dates of hospital stay, and institutional markings. Written informed consent was obtained from the patient's direct relatives for the use of clinical information and medical images for strictly academic and scientific publication purposes.

IV. RESULTS

The timely initiation of intensive supportive care and specific immunological neutralization using the purified polyvalent fabotherapy successfully contained the systemic toxic picture. The clinical state of shock detected upon admission was completely reversed within the first 24 hours of monitoring in the Intensive Care Unit, achieving sustained stabilization of blood pressure values and maintaining the mean arterial pressure within the established tissue perfusion targets. This optimal hemodynamic control was reached exclusively through a goal-directed fluid resuscitation strategy, eliminating the need to initiate adrenergic support or vasopressor infusions during the remainder of the hospital stay.

In the respiratory setting, the acute pulmonary insufficiency that conditioned an initial oxygen desaturation of 80% resolved gradually following the administration of the antivenom and optimization of intravascular volume. The patient reached stable oxygenation and peripheral saturation goals (94% to 98%) under supplemental low-flow oxygen support via nasal prongs at 3 liters per minute. This favorable evolution allowed the definitive withdrawal of non-invasive ventilatory support within a few hours of his admission to the intensive care unit, subsequently remaining with an adequate respiratory mechanics, well-ventilated lung fields, and without pathological findings on room air.

Strict analytical monitoring documented the complete resolution of the acute kidney injury of prerenal and toxic etiology that threatened the patient's survival. Baseline serum creatinine levels decreased drastically from a critical value of 1.9 mg/dL upon admission to normal levels of 0.9 mg/dL within the first 24 hours, finally stabilizing at 0.5 mg/dL during his stay on the general ward. In parallel, the clearance of nitrogenous compounds was optimal, with urea decreasing from 107 mg/dL to 17.1 mg/dL and blood urea nitrogen from 50 mg/dL to a control value of 8 mg/dL. This notable biochemical recovery coincided with the re-establishment of effective and spontaneous diuresis, increasing the urine output from oliguric ranges to a constant volume of 1.87 to 3.84 mL/kg/h, maintaining negative partial and global fluid balances.

The hematological and infectious profile demonstrated the containment of the systemic inflammatory response syndrome and early consumption coagulopathy secondary to toxin-mediated platelet activation. The leukocyte count stabilized within normal parameters (6,230/ μ L to 6,070/ μ L), correcting the initial 95% neutrophilia to absolute values of

3.9 to 4.37 thousand/ μ L. The coagulation profile reflected absolute correction of platelet and plasmatic activity, with a decrease in prothrombin time from 16.8 to 13.6 seconds, correction of the International Normalized Ratio from 1.46 to 1.18, and normalization of activated partial thromboplastin time at 35.4 seconds. Moderate thrombocytopenia reversed from a critical nadir of 53,000/ μ L to a safe count of 259,000/ μ L, while D-dimer reflected a sustained decline from 6,038 ng/mL to 3,266 ng/mL, with no evidence of active bleeding manifested.

In the metabolic and fluid-electrolyte sphere, individualized parenteral solutions allowed the successful correction of imbalances detected upon admission. The initial hypotonic euvoletic hyponatremia of 126 to 130 mmol/L and mild hypocalcemia of 6.8 to 7.1 mg/dL were fully reversed, reaching a normalized sodium of 142 mmol/L and serum calcium levels within targets of 8.4 mg/dL. Central glucose values remained controlled within normal fasting parameters (78 to 116 mg/dL) without requiring insulin infusions. Likewise, the hypertransaminasemia and transient increase in the cholestatic pattern of the liver parenchyma (gamma-glutamyl transferase at a peak of 491 IU/L and alkaline phosphatase at 391 IU/L), secondary to cutaneous-visceral loxoscelism, showed a trend toward gradual and sustained improvement.

Sequential evaluation of the left upper extremity allowed for precise delimitation of the final extension of cutaneous cytotoxic damage and successful management of dermonecrosis. Through advanced wound care performed every 48 hours at the patient's bedside with calcium alginate and gelocast (calamine, glycerin, and zinc oxide) dressings, the abundant serous exudate was controlled and the progression of the violaceous erythema was halted. Selective chemical and mechanical debridement of peripheral slough tissue favored the appearance of abundant clean granulation tissue free of infection. Tissue necrosis was confined dryly and exclusively to the distal phalanx of the fifth finger and isolated areas of the left forearm, maintaining peripheral radial and ulnar pulses palpable with an immediate distal capillary refill of 2 seconds.

The final outcome of the elective surgical intervention, scheduled on the 19th day of intrahospital stay by the Reconstructive Plastic Surgery service, demonstrated absolute technical success without transoperative complications. The procedure involving general surgical wash, exhaustive mechanical debridement, and execution of left hand fasciotomy safely released residual accumulated tissue tension. Skin coverage through sterile petrolatum gauzes and the placement of a special mechanical compression tie-over system with orrany packing fully preserved the vascular viability of the limb. On his first postoperative day, the patient was reported asymptomatic, afebrile, with immediate capillary refill, preserved muscle strength of 3/5 on the Daniels scale, and with an adequate

Cutaneous Loxoscelism Complicated by Tissue Necrosis: A Case Report and Surgical Resolution

tolerance to assisted ambulation, determining his definitive hospital discharge.



Figure 1. Anteroposterior radiographic view of the left humerus showing a marked concentric increase in peripheral soft tissue volume and density due to severe edema secondary to cutaneous loxoscelism, with intact bone cortical structures and mild, chronic, age-related osteodegenerative changes at the elbow joint.



Figure 2. Anteroposterior radiographic view of the left forearm demonstrating prominent swelling and diffuse expansion of the perilesional soft tissue compartment, with no radiolucent images of subcutaneous emphysema or intra-tisular free gas, ruling out indirect signs of necrotizing fasciitis



Figure 3. Lateral radiographic view of the left upper arm and elbow joint confirming the persistence of a marked centripetal inflammation and distention of the peripheral subcutaneous connective tissue, with preserved joint alignment and bone trabeculation.



Figure 4. Anteroposterior radiographic view of the distal forearm and left wrist joint demonstrating marked tissue edema extending toward the dorsal region of the hand, with preservation of the periarticular bony and cartilaginous structures evaluated upon admission.

V. DISCUSSION

The clinical and pathophysiological analysis of severe cutaneous-visceral loxoscelism in the geriatric patient represents one of the greatest challenges within emergency medicine and modern clinical toxicology, due to the speed with which systemic complications are established and the

Cutaneous Loxoscelism Complicated by Tissue Necrosis: A Case Report and Surgical Resolution

frequency with which misdiagnoses are made. In the present case, the initial diagnosis was hindered because the wet dermonecrosis lesions accompanied by blisters and epidermal detachment mimicked Lyell's syndrome or toxic epidermal necrolysis, a common confusion that has been reported in the medical literature given prior exposure to antifungal and analgesic drugs. This conundrum highlights the negative predictive value of clinical tools such as the *NOT RECLUSE* mnemonic, which stipulates that *Loxosceles reclusa* lesions typically present as flat or sunken with a pale, blue-white, or violaceous center due to immediate ischemia, and that the presence of raised erythematous areas or massive wet epidermolysis obligates the exclusion of drug-induced dermatosis or pyoderma gangrenosum, unless there is a clear history of epidemiological exposure during the rainy season.

4

The systemic severity observed in this geriatric patient, characterized by an initial state of hemodynamic shock, metabolic acidosis, and acute kidney injury, finds its molecular basis in the action of phospholipase D, which acts as the central and most lethal component of the violin spider's venom. Upon binding to the endothelium of the microvasculature, this enzyme hydrolyzes the sphingomyelins of cell membranes and promotes an uncontrolled release of proinflammatory cytokines (such as interleukin-6 and tumor necrosis factor- α), triggering an "autonomic storm" and an exacerbated systemic inflammatory response. The patient's laboratory findings upon admission faithfully reflected this phenomenon, showing an intense leukocytosis with 95% neutrophilia and a critical elevation of D-dimer at 6,038 ng/mL, which is indicative of diffuse vascular injury with activation of coagulation mechanisms and the formation of luminal microthrombi, conditioning severe disorders in microcirculation and transient multiorgan dysfunction.⁵

The onset of acute kidney injury in the visceral variant of loxoscelism is the main determining factor of lethality, conferring a mortality rate ranging between 15% and 25% of systemic cases. In this case, the acute deterioration of renal functions was manifested with an elevation of serum creatinine to 1.9 mg/dL and a calculated glomerular filtration rate of 38 mL/min, accompanied by massive microhematuria with countless erythrocytes and proteinuria of 75 mg/dL. Pathophysiologically, this renal damage is multifactorial and involves both renal ischemia secondary to intense local vasoconstriction mediated by the catecholamine storm, as well as the direct cytotoxic action of the venom and the tubular accumulation of large amounts of myoglobin secondary to rhabdomyolysis and tissue destruction of the affected extremity, a mechanism that mimics the deposition nephropathy observed in severe viperid ophidic envenomations.⁶

A paraclinical finding of exceptional interest in this clinical case was the severe elevation of brain natriuretic peptide documented upon admission, a molecular biomarker

that reflects a state of myocardial overload and stress on the left ventricular wall. Although loxoscelism is classically associated with dermatological and renal complications, the massive release of vasoactive and inflammatory peptides from the adrenal cortex and sympathetic nerve endings induces an epicardial coronary vasospasm and an increase in coronary microvascular resistance that can provoke myocardial stunning or functional ischemia. This multifactorial environment explains the sharp left chest pain of 9/10 intensity presented by the patient, which, coupled with the increase in D-dimer, forced the exclusion of a pulmonary thromboembolism or an acute myocardial infarction through computed tomography angiography and enzymatic profiles, confirming that the cardiovascular compromise was secondary to deep vasoconstriction and the increase in left ventricular afterload induced by the systemic toxicity of the venom.⁶

The presence of a transient cholestatic pattern with mild hypertransaminasemia and hyperbilirubinemia and a marked hypoalbuminemia of 2 g/dL upon admission, evidenced the liver parenchymal repercussion of visceral loxoscelism. Toxicological and experimental studies have shown that high levels of circulating cytokines cause an unbalanced redox state in the liver parenchyma, altering the metabolism of carbohydrates and lipids and promoting the appearance of degenerative vacuoles similar to the Mallory bodies described in severe steatohepatitis. This liver metabolic and immunomodulatory dysfunction contributes to the initial delay in protein synthesis and endothelial capillary plasma leakage, accentuating generalized soft tissue edema and transiently limiting the healing capacity of peripheral tissues.⁷

The cornerstone in the management of severe cutaneous-visceral loxoscelism is the timely administration of specific antiloxosceles fabotherapy (*Reclusmyn*®), whose clinical efficacy is directly dependent on the time elapsed between the bite and the intravenous infusion. The polyvalent lyophilized antivenom, composed of equine immunoglobulin F(ab')₂ fragments directed against *Loxosceles reclusa*, *Loxosceles laeta*, and *Loxosceles boneti* necrotoxins, acts by neutralizing free fractions of circulating phospholipase D and interrupting the immunological and lytic cascade before it irreversibly binds to target tissues. Although some authors report that the antivenom loses effectiveness if administered after 36 to 72 hours of the attack, the successful use of fabotherapy in this geriatric patient within the first 24 hours of admission demonstrated to halt the progression of vascular collapse and completely reverse multiorgan dysfunction, allowing serum creatinine to decrease from 1.9 mg/dL to 0.5 mg/dL and normalizing coagulation times within a 72-hour span.⁷

Complementary management of severe loxoscelism requires intensive and multidisciplinary support in which the routine prophylactic use of antibiotics is proscribed unless there are frank signs of aggregated bacterial infection in cultures from the injured foci. In this patient, the coverage

Cutaneous Loxoscelism Complicated by Tissue Necrosis: A Case Report and Surgical Resolution

with cefepime and clindamycin was justified due to the history of joint puncture and needle interventions performed by external physicians in an edematous and friable upper limb, which exponentially increased the risk of bacteremia by nosocomial pathogens or microorganisms such as *Clostridium perfringens*, an anaerobic species isolated from the mouthparts of some violin spiders and associated with a massive increase in the area of tissue necrosis. Likewise, antithrombotic prophylaxis with low-molecular-weight heparin and the guided use of goal-directed fluid therapy allowed for optimizing renal ultrafiltration and mitigating microvascular thrombi deposition without exacerbating endothelial capillary leakage.⁸

The surgical approach to dermonecrosis sequelae must be strictly deferred until the necrotic plaque and areas of dermoepidermal detachment are perfectly demarcated, a period that the scientific literature establishes between 2 and 8 weeks following intoxication. The elective surgical intervention performed on the patient on the 19th day of hospital stay, consisting of surgical wash, mechanical debridement of peripheral slough tissue, and left hand fasciotomy, avoided the risk of severe uncoercible bleeding and limited structural damage to adjacent tendons and vessels by operating on a bed with abundant clean granulation tissue free of active enzymatic lysis. The post-surgical use of special tie-over mechanical compression dressings with organdy and sterile petrolatum gauzes guaranteed optimal tissue apposition and preservation of the peripheral vascular sufficiency of the left extremity, demonstrating that sequential and interinstitutional management between Intensive Care, Internal Medicine, and Reconstructive Plastic Surgery significantly decreases long-term morbidity and preserves the functionality of the affected limb in the elderly.⁸

CONCLUSIONS

Severe cutaneous-visceral loxoscelism represents a medical emergency with a potentially catastrophic evolution that poses an imminent threat to the patient's life, especially when it occurs in vulnerable groups such as the geriatric population. The intrinsic complexity of its early systemic manifestations, coupled with the potential to develop extensive epidermolysis or bullous lesions, requires the first-contact physician to maintain a high index of clinical suspicion to differentiate this entity in a timely manner from severe drug-induced dermatosis or immunological dermatological syndromes, thereby avoiding costly delays in the initiation of definitive treatment.

The favorable evolution of this case conclusively demonstrates that the early initiation of specific polyvalent fabotherapy, administered within the initial therapeutic windows, constitutes the most effective intervention to neutralize the surrounding lytic activity of phospholipase D, successfully halting the progression of systemic endothelial damage, thrombotic microangiopathy, and vascular collapse.

Likewise, this report confirms that fluid support guided by strict hemodynamic goals and close monitoring of molecular and renal function biomarkers prevent the consolidation of acute renal failure due to myoglobin deposition or ischemia, transforming a bleak prognosis into a complete organic resolution.

Finally, long-term therapeutic success relies on a coordinated and sequential multidisciplinary approach between Critical Care, Internal Medicine, and Reconstructive Plastic Surgery services. The strategic deferral of surgical intervention until the complete delimitation of dermonecrosis allows for debridement and fasciotomy procedures to be executed over a biologically stable granulation bed free from active enzymatic lysis. The use of advanced coverage systems and mechanical compression optimizes peripheral vascular sufficiency and tissue apposition, significantly mitigating functional sequelae and ensuring the successful reintegration of the elderly individual into their daily environment.

REFERENCES

- I. Secretaría de Salud; Subsecretaría de Prevención y Promoción de la Salud; Dirección General de Epidemiología. Manual de procedimientos estandarizados para la vigilancia epidemiológica de intoxicación por animales ponzoñosos, 2024 [Internet]. Ciudad de México: Secretaría de Salud; 2024 [citado 2026 jun 20]. Disponible en: https://epidemiologia.salud.gob.mx/gobmx/salud/documentos/manuales/26_Manual_de_Procedimientos_Ponzona_2024.pdf
- II. Zurabian R. La araña violinista y el loxoscelismo [Internet]. Ciudad de México: Unidades de Apoyo para el Aprendizaje, CUAED/Facultad de Medicina-UNAM; 2021 [citado 2026 jun 20]. Disponible en: https://repositorio-uapa.cuaed.unam.mx/repositorio/moodle/pluginfile.php/2487/mod_resource/content/10/UAPA-Loxocelismo-Arana/index.html
- III. Jiménez-Oliver KD, Ortiz MI, Azuara-Antonio O, Mateos-Mauricio FA. Loxoscelismo: Mordeduras de arañas violinistas y sus implicaciones médicas. Educ Salud Boletín Científico Inst Ciencias Salud Univ Autónoma Estado Hidalgo [Internet]. 2023 [citado 2026 jun 20];12(23):35-42. Disponible en: <https://doi.org/10.29057/icsa.v12i23.11619>
- IV. D'Suze G, Corzo Burguete GA, Paniagua Solís JF, editores. Emergencias por animales ponzoñosos en las Américas. Ciudad de México: Instituto Bioclon / Laboratorios Silanes; 2011.
- V. Jiménez-Flores I, Arredondo-Armenta JM, López-Salinas KG, Arango-Mathieu MD, García-Fierro LA, Hernández-Basulto R. Loxoscelismo cutáneo en urgencias: diagnóstico y estrategias terapéuticas. Arch Med Urgen Mex [Internet].

Cutaneous Loxoscelism Complicated by Tissue Necrosis: A Case Report and Surgical Resolution

- 2025 [citado 2026 jun 20];17(1):39-44. Disponible en: <https://dx.doi.org/10.35366/120933>
- VI. Martínez-Medina MA, González-Chávez JA, Martínez-Medina JC. Probable cutaneous-hemolytic loxoscelism. Med Fam SEMERGEN [Internet]. 2024 [citado 2026 jun 20];50(5):102229. Disponible en: <https://doi.org/10.1016/j.semerg.2024.102229>
- VII. Sheta RV, Kolekar KK, Phulari YJ. Cutaneous Loxoscelism: A Potential Diagnosis. Indian J Dermatol [Internet]. 2024 [citado 2026 jun 20];69(2):202. Disponible en: https://doi.org/10.4103/ijd.ijd_467_23
- VIII. Martínez-Ortiz D, Torres-Castro M, Arisqueta-Chablé C, Salceda-Sánchez B, Huerta H, Palacio-Vargas J, et al. First record of a case of cutaneous loxoscelism caused by violin spider (*Loxosceles yucatana*) bite in Yucatan, Mexico. Rev Peru Med Exp Salud Publica [Internet]. 2022 [citado 2026 jun 20];39(4):493-498. Disponible en: <https://doi.org/10.17843/rpmesp.2022.394.11047>