the poison®

2026 | Vol. 3 | No. 2: 9-18

REVIEW

Necrotic skin lesions at the crossroads of toxicology and infectious disease: Distinguishing loxoscelism from necrotizing skin and soft tissue infections

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Submitted: 02/04/2026 - Accepted: 04/06/2026 - Published: 30/06/2026 - DOI: https://doi.org/10.62129/MKYH8845

ABSTRACT. Loxoscelism produces a spectrum of cutaneous and systemic manifestations driven by sphingomyelinase D, the principal dermonecrotic toxin. The resulting necrotic lesion is often clinically indistinguishable at initial presentation from several severe infectious conditions, most notably necrotizing skin and soft tissue infections (NSTIs), community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infections with necrotizing features, and ecthyma gangrenosum. Diagnostic errors carry consequences in both directions: necrotizing fasciitis misdiagnosed as a spider bite may delay life-saving surgical intervention, whereas loxoscelism managed as a bacterial infection exposes patients to unnecessary antimicrobial therapy and may delay appropriate supportive care. An additional layer of complexity arises from secondary bacterial superinfection of *Loxosceles* wounds, a genuine complication that requires targeted therapy but appears to be substantially less common than current prescribing practices suggest. This narrative review examines the pathophysiological mechanisms underlying the shared clinical phenotype of these conditions, describes the clinical and laboratory features that facilitate meaningful bedside differentiation, and proposes a practical framework for clinicians evaluating an acute necrotic wound of uncertain etiology. Particular emphasis is placed on the recognition of NSTIs as surgical emergencies that should never be excluded solely on the basis of ancillary investigations.

Keywords: *Loxosceles; Spider bites; Necrotizing fasciitis; Methicillin-resistant Staphylococcus aureus; Soft tissue infections.*

Necrotic skin lesions represent one of the most diagnostically challenging presentations encountered in emergency and acute care medicine.[1] Clinicians are often confronted with an evolving wound that may reflect envenomation, a rapidly progressive bacterial infection, or, less commonly, a noninfectious inflammatory process, each requiring distinct and often divergent management strategies.[1,2] This diagnostic challenge is particularly pronounced in regions where *Loxosceles* spiders are endemic, including South America, Central America, and parts of North America, where clinical suspicion of loxoscelism is both warranted and, paradoxically, susceptible to overdiagnosis.[3]

Loxoscelism is the clinical syndrome caused by envenomation by spiders of the genus *Loxosceles*, among which *L. laeta*, *L. intermedia*, and *L. reclusa* are considered the species of greatest medical relevance.[3-5] The venom is a complex mixture of biologically active toxins, with sphingomyelinase D (SMaseD) recognized as the principal mediator of the dermonecrotic and hemolytic manifestations that characterize the syndrome.[2,5]

The cutaneous form, representing the vast majority of cases, typically manifests as a painful, progressively enlarging plaque with a characteristic livedoid appearance that evolves over several days into a necrotic eschar.[2,4,5] In contrast, the less common viscerocutaneous variant is characterized by systemic involvement, including hemolysis, hemoglobinuria, and acute kidney injury, and is associated with substantial morbidity and potentially life-threatening complications.[2,5]

Critically, the same phenotype—necrosis, erythema, warmth, and progressive tissue destruction—is also the hallmark of necrotizing soft tissue infections (NSTIs), a heterogeneous group of bacterial infections characterized by rapid spread along fascial planes and high mortality rates that

escalates with each hour of delayed surgical intervention.[1] The overlap is not merely academic: multiple case series have documented patients with necrotizing fasciitis initially misdiagnosed as spider bites, with surgical exploration delayed by hours or days.[1,6-8] Conversely, misattribution in the opposite direction (empirically treating loxoscelism as a bacterial infection) is perhaps more common and carries equally important implications for antibiotic stewardship and appropriate care.

This review adopts an infectious disease perspective to address a problem traditionally framed in toxicological terms. Our aim is to provide clinicians with a practical, pathophysiology-based framework for the differential diagnosis of acute necrotic wounds, with particular emphasis on the features that distinguish loxoscelism from NSTIs, the conditions most commonly confused with it, and the specific scenario of secondary bacterial superinfection in *Loxosceles* wounds.

DISCUSSION

Pathogenesis of the Loxosceles wound: Why it mimics infection

Understanding the mechanisms by which *Loxosceles* venom produces tissue injury is essential to explaining both its remarkable ability to mimic infectious processes and the genuine diagnostic difficulty of distinguishing these entities at the bedside.[2,4,5]

The primary dermonecrotic component of *Loxosceles* venom is SMaseD, a phospholipase D enzyme that hydrolyzes sphingomyelin and lysophosphatidylcholine, generating bioactive lipid mediators that initiate a cascade of local inflammation. SMaseD activates the complement cascade, with evidence supporting involvement of both classical and alternative pathways, promoting deposition of the membrane attack complex on erythrocytes and endothelial cells.[7,8] The resulting endothelial injury promotes platelet aggregation, microvascular thrombosis, and ultimately ischemic necrosis of the dermis and subcutaneous tissue.[2,3] Concurrently, complement activation and cytokine release promote marked neutrophil recruitment to the wound site, creating a highly inflammatory microenvironment characterized by erythema, edema, warmth, and tenderness—findings that are clinically indistinguishable from bacterial cellulitis.[9,10]

The livedoid pattern that frequently precedes frank necrosis reflects patchy microvascular occlusion, producing alternating areas of pallor and erythema.[3,5] Although not pathognomonic, this livedo reticularis-like appearance represents an important early diagnostic clue when present.[2,3] Over the subsequent 48–72 hours, the central area evolves into a dry eschar, often surrounded by a violaceous ring and an outer zone of erythema, creating the classic "red, white, and blue" sign of loxoscelism.[5,11,12]

The intense local inflammatory response creates a wound environment that is not only clinically similar to infection but also genuinely susceptible to secondary bacterial colonization and superinfection.[1,6,9,10] Necrotic tissue provides a favorable substrate for microbial growth, while disruption of the skin barrier creates a portal of entry for pathogens. This dual reality—that loxoscelism both mimics infection and may itself become complicated by infection—is central to the diagnostic and therapeutic challenges addressed in this review.[13,14]

The clinical spectrum of loxoscelism

Cutaneous loxoscelism. Cutaneous loxoscelism accounts for approximately 85–90% of cases. Patients typically present with a painful, pruritic lesion that develops within 2–8 hours after envenomation, although many bites go unnoticed at the time of occurrence. Lesions most commonly involve covered areas of the body, particularly sites where clothing comes into close contact with the skin during sleep, including the axillae, thighs, and trunk, reflecting the peridomestic behavior of *Loxosceles* spiders.[2,3,5,11]

The early lesion typically presents as an erythematous macule that may subsequently develop a central vesicle or bulla. Over the ensuing 12–72 hours, the central area progresses to necrosis, forming a dry, stellate eschar surrounded by induration and erythema. The eschar usually becomes well demarcated over 1–2 weeks before separating to reveal a deep ulcer that heals slowly over weeks to months and may ultimately leave permanent scarring. Fever is typically absent or low grade in uncomplicated cutaneous disease; its presence should prompt consideration of either viscerocutaneous loxoscelism or secondary bacterial superinfection.[2,3,5,7,8,11,13]

Viscerocutaneous loxoscelism. Viscerocutaneous loxoscelism represents the severe systemic variant of the syndrome and is associated with substantially higher morbidity.[15] It occurs in a minority of patients, with estimates ranging from 1% to 13% of envenomation cases in Latin American series, with children appearing to be at disproportionately higher risk.[4,7] Systemic manifestations typically develop within 24–72 hours after envenomation and include fever, chills, nausea, vomiting, and arthralgia. The hallmark of systemic involvement is intravascular hemolysis, which manifests as declining hemoglobin levels, elevated lactate dehydrogenase, indirect hyperbilirubinemia, and hemoglobinuria.[2,5,8,11,15]

Acute kidney injury may develop secondary to hemoglo-

bin-mediated tubular toxicity and represents the principal determinant of mortality in severe cases. Disseminated intravascular coagulation has been reported in severe viscerocutaneous loxoscelism and constitutes a life-threatening complication.[11,14,15] The combination of systemic hemolysis, fever, and an evolving necrotic wound creates a clinical picture that substantially overlaps with septic shock of any origin, and distinguishing these entities from severe NSTI with bacteremia requires active diagnostic evaluation rather than passive observation.[1,9,10,12,16]

Necrotizing soft tissue infections: The differential that cannot wait

Overview. NSTIs comprise a spectrum of rapidly progressive bacterial infections involving the skin, subcutaneous tissue, superficial fascia, deep fascia, or muscle. Their defining characteristic is the capacity for explosive spread along tissue planes, accompanied by systemic toxicity and mortality rates that range from 20 to 40% in large series despite optimal management. No diagnostic test, imaging modality, or laboratory score can replace the judgment of an experienced clinician and surgeon; decisions regarding operative exploration should never be delayed while awaiting confirmatory investigations.[1,6,9,10,12] An overview of the differential diagnoses is provided in Table 1.

Necrotizing fasciitis. Necrotizing fasciitis (NF) is traditionally classified into type I (polymicrobial) and type II (monomicrobial, predominantly caused by *Streptococcus pyogenes*). Type I NF predominates among older patients with underlying comorbidities, including diabetes mellitus, peripheral vascular disease, and immunosuppression, and typically involves synergistic infections caused by mixed aerobic and anaerobic organisms. In contrast, type II NF may affect previously healthy individuals and often follows a more fulminant clinical course. Type III NF, most commonly associated with *Vibrio vulnificus* or *Clostridium* species, is less frequent but carries particularly high mortality.[9,10,17]

The earliest cutaneous manifestation of NF is erythema overlying the affected fascial compartment, often clinically indistinguishable from cellulitis. This rapidly progresses to edema, blistering, skin discoloration, and ultimately frank necrosis. The pathognomonic finding is pain out of proportion to the visible skin changes. In later stages, paradoxical cutaneous anesthesia may develop as progressive nerve destruction occurs. Crepitus, although present in approximately 50% of type I NF cases due to gas-forming organisms, is frequently absent in type II NF disease and should not be considered prerequisite for diagnosis.[1,6,10]

The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score, which incorporates C-reactive protein, leukocyte count, hemoglobin, sodium, creatinine, and glucose levels, has been proposed as a tool for risk stratification. However, its sensitivity for NF is imperfect, with multiple studies reporting clinically confirmed cases in patients with low LRINEC scores. The LRINEC score should therefore be considered only an adjunctive risk assessment tool; a low score does not exclude NF and should never be used to justify delaying surgical exploration in a patient with significant clinical suspicion.

Magnetic resonance imaging (MRI), when available, has the highest sensitivity for detecting fascial plane involvement and may be useful in equivocal cases without hemodynamic instability. Computed tomography (CT) can demonstrate gas tracking along fascial planes, particularly in type I NF. However, both modalities carry the risk of delaying operative management in unstable patients or those with a high clinical suspicion of NF, and the surgical aphorism bears repeating: if NF is clinically suspected, the operating room serves as both the diagnostic and therapeutic intervention.[9,10,17-21]

Community-associated methicillin-resistant Staphylococcus aureus with necrotizing features. Community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) has emerged as a major cause of skin and soft tissue infections, particularly in regions where the USA300 lineage is prevalent. In contrast to healthcare-associated MRSA strains, CA-MRSA isolates frequently carry the Panton-Valentine leukocidin (PVL) gene, which encodes a bicomponent toxin capable of inducing leukocyte lysis and is strongly associated with necrotizing skin infections and, in its most severe presentation, necrotizing pneumonia.[22-24]

PVL-positive CA-MRSA infections typically present as furuncles or carbuncles that may evolve into necrotic ulcers, often with a central zone of tissue destruction that mimics the eschar seen in loxoscelism.[22,23,25,26] Perilesional erythema may be extensive, and systemic manifestations, including fever and leukocytosis, are common. Bacteremia occurs in approximately 20–30% of cases involving skin and soft tissue infection and may produce a septic clinical picture that overlaps with viscerocutaneous loxoscelism.[1,11,15,24,25]

The epidemiologic context represents a critical differentiating factor. CA-MRSA skin infections tend to cluster in settings characterized by close physical contact, including households, athletic facilities, prisons, and military barracks, as well as among individuals with known exposure to colonized contacts. In contrast, loxoscelism typically occurs in the setting of potential exposure to *Loxosceles* habitats, such as stored clothing and footwear, woodpiles, or cluttered

TABLE 1. Clinical differential diagnosis of necrotizing skin and soft tissue infections.

Feature	Cutaneous loxoscelism	Viscerocutaneous loxoscelism	NF	CA-MRSA necrosis	EG
Onset after event	Hours–days	Hours–days	Hours (rapid)	Hours–days	Days (immunocomp.)
Typical lesion	Livedoid plaque → necrosis → eschar	Cutaneous findings + systemic manifestations	Erythema → bullae → necrosis with extension along fascial planes	Folliculitis → furuncle → necrotic ulcer	Hemorrhagic pustule → black eschar
Progression speed	Days (relatively slow)	Days–weeks	Hours (alarming)	Days	Variable
Pain	Often disproportionate initially, may decrease as necrosis develops	Variable	Severe, followed by paradoxical reduction due to cutaneous anesthesia	Moderate to severe	Often mild (immunocomp.)
Systemic signs	Absent or mild	Fever, hemolysis, AKI, hemoglobinuria	High fever, tachycardia, hypotension, shock	Variable; bacteremia possible	Fever and sepsis in the setting of neutropenia
Crepitus/gas	Absent	Absent	Present in ~50% of type I cases	Absent	Absent
Host	Immunocompetent	Any; children at higher risk	Diabetes mellitus, immunocomp., recent surgery	Immunocompetent (community)	Hematologic malignancy, neutropenia
Epidemiologic clue	Endemic region; potential exposure through clothing or stored items	Same	Recent wounds, surgery, or skin disruption	Skin-to-skin contact; crowded settings	Chemotherapy, prolonged neutropenia
Laboratory findings	Usually normal	Hemolytic anemia, elevated LDH, AKI	Elevated CRP, CK, and LDH; leukocytosis; LRINEC score ≥6	Leukocytosis; bacteremia in ~30%	Bacteremia; positive blood cultures for *Pseudomonas* spp.
Microbiology	No primary pathogen	No primary pathogen unless superinfected	Polymicrobial (type I) or GAS (type II)	CA-MRSA (PVL-positive)	*P. aeruginosa*; less commonly *Fusarium* spp.
Urgent surgery	No	No	Yes; do not delay for imaging	Rarely (incision and drainage only)	No

NF: necrotizing fasciitis; CA-MRSA: community-associated methicillin-resistant *S. aureus*; EG: ecthyma gangrenosum; AKI: acute kidney injury; LDH: lactate dehydrogenase; CRP: C-reactive protein; CK: creatine kinase; LRINEC: Laboratory Risk Indicator for Necrotizing Fasciitis; GAS: Group A *Streptococcus*; PVL: Panton-Valentine leukocidin.

peridomestic environments in endemic regions.[25,27,28] The diagnostic challenge is particularly pronounced in regions endemic for loxoscelism where CA-MRSA is also prevalent, and where the absence of a witnessed spider bite is the rule rather than the exception.

Ecthyma gangrenosum. Ecthyma gangrenosum (EG) is a distinctive cutaneous lesion most commonly associated with *Pseudomonas aeruginosa* bacteremia, although it may also be caused by other Gram-negative organisms and, more rarely, by fungi such as *Fusarium* spp. or *Aspergillus* spp. in the setting of prolonged neutropenia.[29,30] The lesion initially presents as an erythematous macule that rapidly progresses through vesicular and bullous stages before evolving into a necrotic ulcer with a black eschar surrounded by an erythematous halo.[1]

The key distinguishing factor in EG is the host context: the vast majority of cases occur in patients with hematologic malignancies, solid organ transplantation, advanced HIV-associated immunosuppression, or prolonged neutropenia of any cause.[6,29,31] The presence of documented neutropenia or recent chemotherapy exposure in a patient presenting with a necrotic skin lesion should always prompt consideration of EG and urgent blood culture collection, given the strong association with bacteremia and its high attributable mortality.

Importantly, the skin lesion in EG represents a metastatic manifestation of bacteremia rather than a primary wound infection; surgical intervention is not indicated, and systemic antipseudomonal therapy remains the cornerstone of treatment.[17,29-31]

Clinical red flags for necrotizing skin and soft tissue infections

Given the life-threatening nature of NSTIs and the consequences of diagnostic delay, it is useful to frame the differential diagnosis around the identification of features that should immediately raise suspicion for a necrotizing process and prompt urgent surgical consultation. These red flags are bedside clinical findings rather than laboratory or imaging results; clinical assessment takes precedence,[1,9,10,29,31] as summarized in Table 2.

A useful bedside approach is the "pain–appearance discordance": when the patient's pain is disproportionate to the visible skin findings, NF must be actively excluded.[17,18,23] This discordance reflects the anatomical progression of infection deep to the skin, where the fascia and nerves are destroyed while the overlying dermis may initially appear deceptively intact.[9,10,12] Conversely, in cutaneous loxoscelism, the extent of visible tissue damage is often proportionate to—or exceeds—the patient's subjective pain, particularly as the necrotic eschar matures and central anesthesia develops.[2,5-7]

Secondary bacterial superinfection of Loxosceles wounds

Overview. Secondary bacterial superinfection of *Loxosceles* wounds is a clinically relevant complication that warrants consideration both in the differential diagnosis and in the ongoing debate regarding antibiotic prescribing practices. *Loxosceles*-induced necrosis creates a local wound environment that is conducive to bacterial colonization.[25,27,28] The disruption of the skin barrier provides direct access for cutaneous flora, while the complement-consuming and leukocyte-attracting properties of the lesion may paradoxically impair local antimicrobial defense mechanisms.[26]

The reported incidence of clinically significant superinfection in loxoscelism is difficult to quantify, given the paucity of prospective studies and variability in diagnostic criteria across case series.[4] What is clear, however, is that bacterial superinfection is neither universal nor the most common clinical course of cutaneous loxoscelism, and that routine empirical antibiotic prescribing for all presentations of loxoscelism is not supported by robust evidence.[5,7,8,11,14]

TABLE 2. Bedside red flags for necrotizing skin and soft tissue infections.

Clinical sign	Significance	Action
Pain disproportionate to appearance	Hallmark of NF; nerve involvement precedes visible skin change	Emergency debridement; do NOT await imaging
Rapid progression (hours)	Bacterial spread along fascial planes outpaces cutaneous signs	Immediate surgical exploration
Skin anesthesia overlying lesion	Ischemic necrosis of cutaneous nerves; late sign of NF	Emergency debridement
Crepitus or gas on palpation/imaging	Gas-forming organisms (*Clostridium* spp., mixed flora)	Emergency debridement
Hemodynamic instability	Septic shock secondary to NSTI	Resuscitation + emergency debridement
Failure to improve on IV antibiotics at 24–48 h	NSTI or severe SSTI not adequately source-controlled	Surgical reassessment; do not wait
Bullae or violaceous skin discoloration	Dermal ischemia from deep fascial necrosis	High suspicion for NF; urgent review
Wooden-hard texture of subcutaneous tissue	Necrotic fascia; distinguishes NF from cellulitis on palpation	Operative exploration

NF: necrotizing fasciitis; NSTI: necrotizing soft tissue infection; IV: intravenous; SSTI: skin and soft tissue infection.

Distinguishing superinfection from uncomplicated loxoscelism. Several clinical and laboratory features may assist in the identification of secondary bacterial superinfection. The key principle is dynamic assessment: loxoscelism without superinfection typically follows a relatively predictable course, with stabilization of the wound margins within 48–72 hours as the effects of the venom subside. Features that deviate from this trajectory—particularly expanding perilesional erythema, purulent exudate, fever persisting or worsening beyond 48 hours, or progressive leukocytosis—should raise suspicion for superinfection.[20,22,25,29] A summary of clinical and laboratory features distinguishing both entities is provided in Table 3.

Microbiology. The most likely causative organisms in superinfected loxoscelism wounds are those comprising normal cutaneous flora, such as *S. aureus* (including MRSA, particularly in endemic regions) and *S. pyogenes*, and, less commonly, Gram-negative organisms in moist anatomical sites. Wound cultures obtained prior to initiating antibiotics are strongly recommended, both to confirm the diagnosis of superinfection and to guide targeted therapy.[1,6,9,10]

Antibiotic selection. The reflexive prescription of antibiotics for all presentations of loxoscelism is a deeply embedded but poorly supported clinical practice. The theoretical concern that necrotic wounds will invariably become infected has not been borne out in systematic analyses of outcomes with conservative wound management.[4,14] Prophylactic antibiotics have not been shown to reduce the incidence of superinfection, wound complications, or the need for surgical debridement in uncomplicated cutaneous loxoscelism.[14] Antibiotics are indicated when superinfection is clinically evident, as outlined above. In this context, empirical therapy should target *S. aureus*; in regions or populations with a high prevalence of CA-MRSA, coverage should be extended to MRSA pending culture results.[24,27,28]

Diagnostic testing of acute necrotic wounds: Limitations, laboratory evaluation and imaging

Limitations. A fundamental limitation in the evaluation of patients with suspected loxoscelism is the absence of a validated, readily available confirmatory diagnostic test in routine clinical practice. In contrast to NSTIs, which may be supported (though not definitively excluded) by imaging, and bacterial infections, which can be confirmed by culture, loxoscelism remains a clinical and epidemiological diagnosis, with no point-of-care serological or toxicological test currently in widespread use.[32] Fig. 1 presents a stepwise

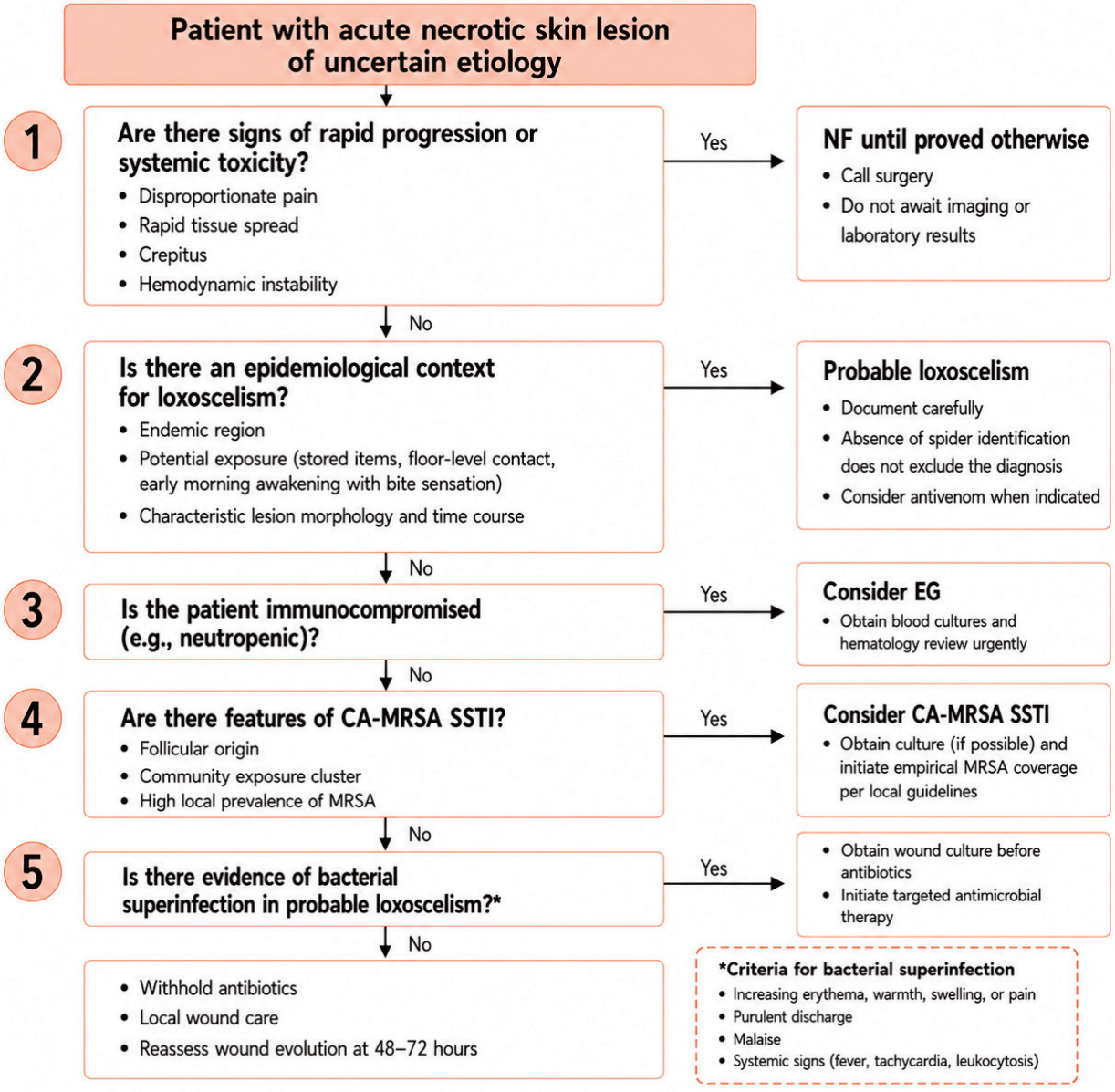
TABLE 3. Clinical features of uncomplicated versus superinfected loxoscelism.

Parameter	Uncomplicated loxoscelism	Superinfected loxoscelism
Fever	Absent or low-grade	Moderate to high; persistent
Wound appearance	Necrosis with clean margins, dry eschar	Erythema extending beyond necrotic margin; purulent exudate; edema
Local warmth/induration	Moderate, decreasing over days	Progressive, increasing
Odor	Absent	Malodorous discharge
Lymphadenopathy	Minimal	Regional lymphadenitis
CRP/leukocytosis	Normal or mildly elevated early	Markedly elevated; leukocytosis with left shift
Likely pathogens	Not applicable	*S. aureus* (including MRSA); *S. pyogenes*; skin flora
Antibiotic indication	No	Yes, guided by local resistance patterns
Wound management	Conservative; dry dressing; avoid debridement in acute phase	Debridement of necrotic tissue; wound cultures before starting antibiotics

CRP: C-reactive protein; MRSA: methicillin-resistant *S. aureus*.

diagnostic algorithm for the assessment of acute necrotic wounds in endemic settings or when the etiology remains uncertain.

Laboratory evaluation. In patients presenting with an acute necrotic wound of uncertain etiology, the initial laboratory evaluation should include a complete blood count with differential (leukocytosis in NSTIs; hemolytic anemia in viscerocutaneous loxoscelism); CRP and procalcitonin (markedly elevated in severe bacterial infection, and typically normal or only mildly elevated in loxoscelism); LDH, haptoglobin, and indirect bilirubin (markers of hemolysis); serum creatinine and electrolytes (to assess AKI); urinalysis, including dipstick testing for hemoglobinuria; and blood cultures in all patients with fever or hemodynamic instability.[2,11,15]



NF: necrotizing fasciitis; EG: ecthyma gangrenosum; CA-MRSA: community-associated methicillin-resistant *S. aureus*; SSTI: skin and soft tissue infection.

Figure 1. Proposed diagnostic framework for acute necrotic wounds presenting in endemic areas or without apparent etiology (AI-assisted figure).

Experimental serological assays detecting *Loxosceles* venom antigens or anti-*Loxosceles* antibodies have been developed in research settings and have shown reasonable sensitivity in serum and wound fluid samples within the first 48–72 hours after envenomation. However, these tests are not commercially available in most countries, and their use is currently restricted to specialized toxicology centers and research settings. The development of a bedside immunochromatographic assay for Loxosceles venom antigens represents a key unmet need and a priority research gap with direct clinical implications.[32-34]

The LRINEC score should be calculated when NF is part of the differential diagnosis. However, a score below the diagnostic threshold does not exclude disease and should not delay surgical evaluation in patients with high clinical suspicion.[19-21]

***Imaging*.** Plain radiographs have limited sensitivity for NSTIs but may demonstrate soft tissue gas in type I NF. CT with IV contrast is the imaging modality of choice when clinical conditions permit delay of surgical management. Typical findings include gas within fascial planes, fascial thickening, fluid tracking along fascial compartments, and lack of contrast enhancement, all of which are supportive but not diagnostic.

MRI, when available, provides superior soft tissue resolution and is the most sensitive imaging modality for early NF, demonstrating fascial thickening and signal abnormalities involving both superficial and deep fascial layers. It should be emphasized that imaging must never delay surgical exploration when clinical suspicion for NSTI is high.[18]

In uncomplicated loxoscelism without concern for NSTI, imaging is not routinely indicated. Doppler ultrasonography may be used to assess local perfusion in large or deep lesions, although it rarely alters clinical management.[1,3,5]

CONCLUSIONS

Loxoscelism and NSTIs share a similar clinical phenotype yet require fundamentally different management approaches: conservative, supportive care for the former and, depending on the specific entity, urgent surgical intervention or aggressive medical therapy for the latter. The most consequential diagnostic error is the misdiagnosis of NF as a spider bite, as no other condition in this differential carries a comparable mortality risk associated with delayed treatment. When NF is clinically suspected, no ancillary investigation should delay intervention; surgical exploration remains both the most sensitive diagnostic modality and the definitive treatment. Finally, antimicrobial stewardship in loxoscelism begins with diagnostic accuracy: antibiotic therapy for presumed superinfection should be supported by compatible clinical findings and, whenever possible, microbiological evidence.

Funding

No specific funding was received for this review from any public, commercial, or not-for-profit funding agency.

Conflicts of interest

PF declares having received financial compensation for consulting and speaking engagements from bioMérieux Argentina.

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