Case Report

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Pediatric Cutaneous Visceral Loxocelism with Renal and Pulmonary Involvement: A Case Report

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Abstract

Background and Clinical Significance: This case reports a severe pediatric presentation of cutaneous–visceral loxoscelism (CVL) following a suspected *Loxosceles* spider bite, highlighting rare systemic complications and the need for early intensive management; **Case Presentation**: A previously healthy 6-year-old girl developed acute hemolysis, respiratory failure, acute kidney injury requiring renal replacement therapy, coagulopathy, and diffuse alveolar hemorrhage, necessitating management in the intensive care unit; **Conclusions**: Pediatric CVL can rapidly progress to life-threatening multisystem involvement; early recognition and timely multidisciplinary supportive care are essential to achieve favorable outcomes.

Keywords: spider bites; cutaneous–visceral loxoscelism; acute kidney injury; diffuse alveolar hemorrhage; case report

1. Introduction and Clinical Significance

Loxoscelism is the envenomation resulting from the bite of a spider of the genus *Loxosceles*, commonly known as the violin or recluse spider [1]. These species are distributed across various regions worldwide; in North America, most cases are attributed to *Loxosceles reclusa*, while in South America, *L. laeta*, *L. intermedia*, and *L. gaucho* are predominant [2].

Clinically, loxoscelism most often presents in its cutaneous form (LC), characterized by a progressive dermonecrotic lesion that can range from an erythematous plaque to a deep ulcer [3]. However, in up to 20% of cases, systemic dissemination of sphingomyelinase D (SMD), a phospholipase responsible for tissue and microvascular injury, leads to the development of cutaneous-visceral or systemic loxoscelism (CVL), an uncommon but potentially life-threatening presentation [4,5].

CVL may present with fever, gastrointestinal symptoms, hemoglobinuria, massive intravascular hemolysis, and consequently jaundice, acute kidney injury (AKI), pulmonary involvement, and coagulation disorders, with a reported mortality exceeding 15%, typically occurring within 4 to 24 hours after the bite [4–6].

Pediatric age is a recognized risk factor for the development of severe forms, and delayed initiation of specific management has been identified as a key determinant of mortality [7]. In this context, early recognition of systemic manifestations and timely therapeutic intervention are crucial to improving clinical outcomes. The aim of this report is to describe the clinical course, systemic complications, and therapeutic management of a pediatric patient with CVL, providing relevant clinical insights that may enhance diagnostic suspicion and optimize management of this condition.

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2. Case Presentation

A 6-year-old female patient from Carhuaz, an Andean valley north of Lima, Peru, with no relevant medical history, suffered a venomous animal bite on the proximal third of the right arm. At the time of the incident, she experienced mild "stinging" pain without visible skin lesions; the area was cleaned with alcohol. Approximately four hours later, she developed an unquantified fever, and oral paracetamol 120 mg/5 mL (5 mL) was administered without resolution of the fever. Nine hours after the incident, disseminated purpuric lesions appeared on the thorax, prompting transfer to the local hospital; during transport, she exhibited generalized muscle weakness and jaundice.

At the local hospital, 5 mL of anti-*Loxosceles* serum was administered intravenously, oxygen therapy was initiated via nasal cannula at 3 L/min, and a Foley catheter was placed for strict urine output monitoring and early detection of hemoglobinuria. Despite these interventions, no clinical improvement was observed, and she was referred to a higher-level care center.

Upon arrival at the emergency department of the referral center, the patient was conscious and oriented, with oxygen support and a functional Foley catheter. Vital signs were as follows: blood pressure 94/59 mmHg, heart rate 116 bpm, respiratory rate 40 breaths per minute, temperature 36.9 °C, and oxygen saturation 96% on 3 L/min via nasal cannula.

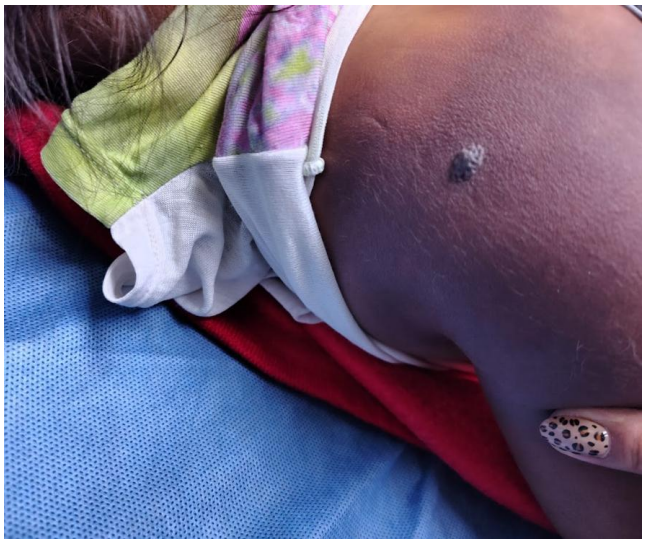

Figure 1. Approximately 24 hours after the incident, a necrotic lesion was observed on the proximal third of the lateral aspect of the right arm, showing extensive perilesional ecchymosis at the time of admission.

Physical examination revealed a necrotic lesion with indurated edema and extensive perilesional ecchymosis (Figure 1), pallor, conjunctival jaundice, respiratory distress with accessory muscle use, decreased bilateral breath sounds with left-sided predominance, and hematuria. Laboratory investigations were requested, including complete blood count, coagulation profile, haptoglobin, lactate dehydrogenase, urea, creatinine, liver function tests, serum electrolytes, and arterial blood gases (Table 1).

Table 1. Laboratory tests conducted at admission.

Laboratory Test	Result
Hematology	
Hb	8.0 g/dL
Hct	19.8%
WBC	27,970 /mm³
Segmented neutrophils	84%
Lymphocytes	14%

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Plt	200,000 /mm³
Direct/indirect Coombs test	Negative
Coagulation Profile	
PT	18.6 sec
INR	1.55
TT	18.3 sec
D-dimer	34,651 mg/L
Haptoglobin	24 mg/dL
LDH	185,600 U/L
Renal Function	
Creatinine	3.18 mg/dL
Urea	162 mg/dL
Liver Function Tests	
Albumin	1.9 g/dL
Globulin	7.5 g/dL
AST (TGO)	73 U/L
ALT (TGP)	32 U/L
Total bilirubin	8.42 mg/dL
Indirect bilirubin	7.94 mg/dL
Direct bilirubin	0.48 mg/dL
Electrolytes	
Na	137 mmol/L
K	6.2 mmol/L
Cl	107 mmol/L
Arterial Blood Gases	
pH	7.31
pCO_2	18.1 mmHg
HCO_3	9.2 mmol/L
Lactate	3.7 mmol/L

[1] Severe anemia (Hb < 10 g/dL), leukocytosis, markedly elevated D-dimer and LDH, acute kidney injury (elevated creatinine and urea), hypoalbuminemia, hyperbilirubinemia (predominantly indirect), hyperkalemia, metabolic acidosis (low HCO_3^- and pH), and elevated lactate were observed. Hb: Hemoglobin; Hct: Hematocrit; WBC: White Blood Cells; Plt: Platelets; PT: Prothrombin Time; INR: International Normalized Ratio; TT: Thrombin Time; LDH: Lactate Dehydrogenase; AST (TGO): Aspartate Aminotransferase; ALT (TGP): Alanine Aminotransferase; Na: Sodium; K: Potassium; Cl: Chloride; pCO_2: Partial Pressure of Carbon Dioxide; HCO_3: Bicarbonate.

Results indicated acute hemolytic anemia, AKI, coagulopathy, and respiratory compromise, prompting transfer to the intensive care unit (ICU). In the ICU, the patient received assisted mechanical ventilation via endotracheal intubation, vancomycin 15 mg/kg intravenous, intermittent hemodialysis, and fluid and electrolyte management. Forty-eight hours after the incident, the cutaneous lesion developed blisters, necrosis, and eschar formation, leading to surgical debridement performed by the pediatric surgery team (Figure 2).

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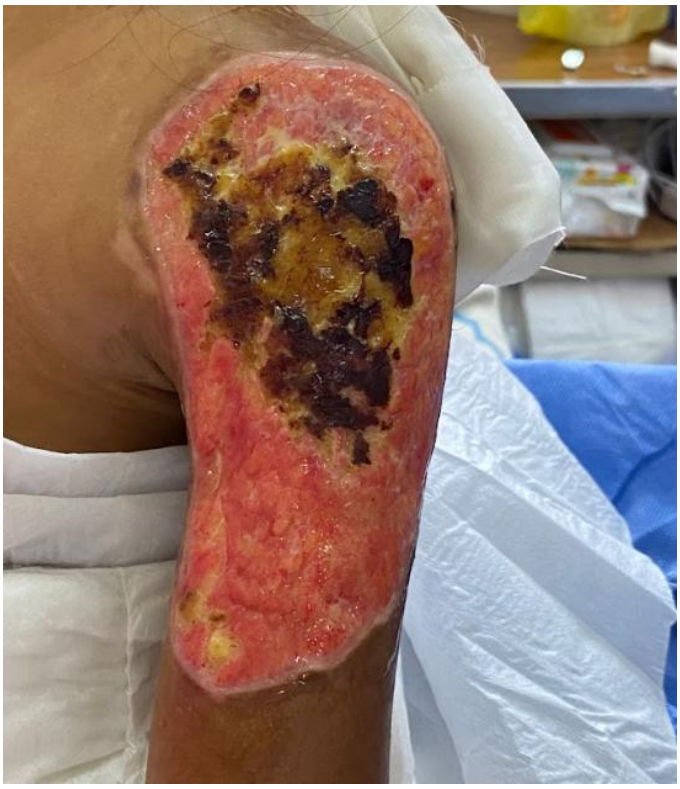
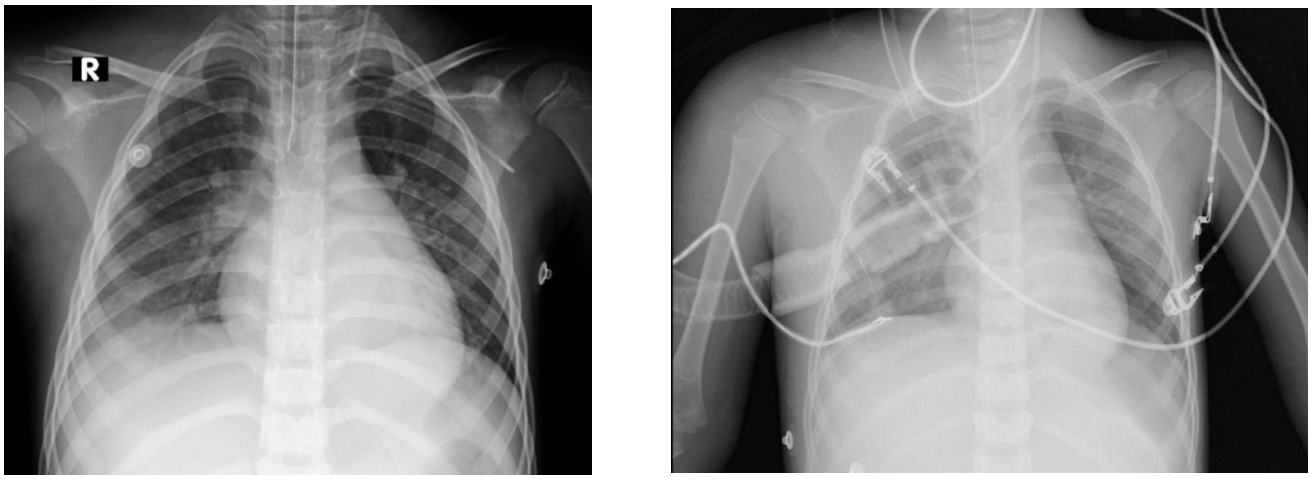


Figure 2. Dermonecrotic lesion after surgical debridement.

On the seventh day of hospitalization, the patient developed nosocomial pneumonia; antibiotic therapy was changed to piperacillin/tazobactam 80 mg/kg every 12 hours for 14 days. During a hemodialysis session, she experienced hemoptysis with a drop in hemoglobin to 6.5 g/dL and new bilateral alveolar infiltrates on chest radiography, consistent with diffuse alveolar hemorrhage (DAH) (Figure 3).



(**a**) (**b**)

Figure 3. (a) Chest X-ray obtained at the time of admission; (b) Chest X-ray showing diffuse bilateral alveolar infiltrates.

Renal function was monitored via serum urea and creatinine levels: on admission, values were 162 mg/dL and 3.18 mg/dL, respectively; on day 26, 117 mg/dL and 2.0 mg/dL; and on day 36, 31 mg/dL and 0.68 mg/dL. Urine output progressed from oliguria/anuria (<0.5 mL/kg/h) to normal ranges (>0.5–1 mL/kg/h), allowing discontinuation of hemodialysis and removal of the central venous catheter.

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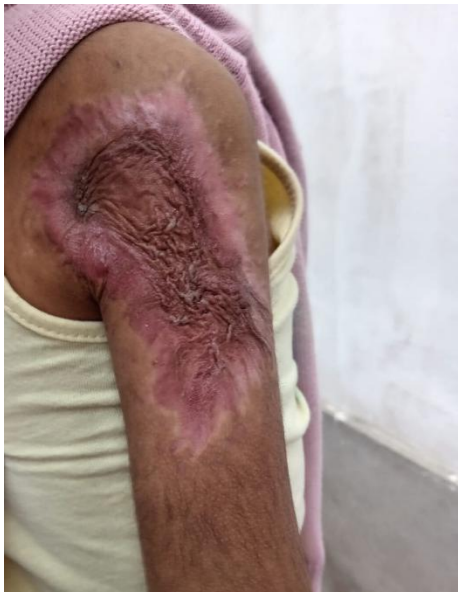

Figure 4. Postoperative wound at two months, located on the right shoulder and arm, showing a keloid scar with erythematous borders.

Following surgical debridement, the wound demonstrated viable granulation tissue. Porcine skin was applied to optimize the wound bed prior to autologous skin grafting, which was obtained from the scalp approximately one week after debridement. After two weeks of postoperative monitoring, the patient was discharged with scheduled outpatient follow-up. At one- and two-month follow-up visits, the wound exhibited a keloid scar with mildly erythematous borders and slight pruritus (Figure 4), with no evidence of pulmonary or renal sequelae or other long-term complications related to the clinical course.

3. Discussion

The violin or recluse spider (*Loxosceles* spp.) is distributed across various regions of the world. In North America, most cases are attributed to *L. reclusa*, whereas in South America, *L. laeta*, *L. intermedia*, and *L. gaucho* predominate [2]. This geographic distribution influences both the risk of envenomation and the most frequent clinical manifestations in each region [8,9]. It is important to note that the actual incidence of loxoscelism is uncertain due to the infrequent identification of the causative spider [8]. Nevertheless, CVL accounts for approximately 20% of envenomation cases caused by spiders of the genus *Loxosceles* and represents the most severe form of this condition [1,2,8].

The pathophysiology of CVL has been primarily linked to SMD present in the venom, a highly active type of enzyme responsible for both local and systemic damage [10]. These enzymes can induce microvascular and tissue injury, complement activation, and intravascular hemolysis, mechanisms that explain the characteristic triad of the visceral syndrome: hemolysis, AKI, and coagulopathy [5,6,8,10]. These findings were clearly evidenced in our patient, accounting for both the extent of cutaneous necrosis and the severity of systemic complications.

Regarding the progression of the cutaneous lesion, the literature describes that, following the initial bite, an erythematous macule or painful papule usually appears, which progresses within hours to the "red--white--blue" pattern, characterized by a pale center, an erythematous halo, and peripheral ecchymosis, and may evolve over several days into a livedoid plaque or necrotic ulcer [11,12]. In our case, the patient presented a necrotic lesion in the proximal third of the right arm, accompanied by extensive perilesional ecchymosis, findings that are consistent with the literature and reinforce the diagnosis (Figure 1). Furthermore, the proximal location of the bite and the patient's pediatric age (6 years) are recognized risk factors for progression to severe systemic manifestations [2,7,13,14]. In this context, available evidence indicates that systemic loxoscelism can present with

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severe intravascular hemolysis, coagulopathies, thrombocytopenia, renal dysfunction, and even fatal outcomes [1,2,4–7,13–15]. However, the DAH observed in our patient represents an uncommon finding, highlighting clinical variability.

Regarding therapeutic management, CVL requires the timely administration of antivenom, ideally within the first 12 to 24 hours [16]. Previous studies have shown that the antivenom is effective in cutaneous forms when administered early, preventing or reducing progression to systemic manifestations [17–19]. Nevertheless, in our patient, no discernible clinical improvement was observed despite administration 15 hours post-bite, suggesting that therapeutic efficacy depends not only on the timing of administration but also on the specific characteristics of the venom and the identification of the species involved. This limitation reflects the ongoing controversy regarding the relative efficacy of Brazilian polyvalent antivenoms versus Peruvian species-specific preparations, a matter relevant for public health policy and hospital stock management [20].

Given the rapid progression and severity of complications, ventilatory support, hemodialysis, fluid and electrolyte correction, antibiotic therapy, surgical debridement, and subsequent skin grafting was implemented. These interventions resulted in a favorable outcome, with resolution of renal failure, normalization of urinary output, withdrawal of dialytic support, and adequate reconstruction of the affected tissue.

4. Conclusions

As previously stated, LC can rapidly progress to severe and potentially life-threatening systemic involvement in pediatric patients. Early recognition of systemic manifestations and the timely initiation of individualized and comprehensive management were critical in achieving a favorable outcome. The absence of a clear clinical response to antivenom, despite its administration within the recommended time frame, reflects the variability in its efficacy and the persistent uncertainty reported in the literature regarding its impact on systemic forms. In this context, comprehensive supportive care remains the cornerstone of treatment. This report emphasizes the importance of maintaining a high index of clinical suspicion, optimizing early intervention, and strengthening the available evidence to improve the management and prognosis of severe loxoscelism in the pediatric population.

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Informed Consent Statement: Written informed consent has been obtained from the patient's legal guardians for publication of this case report and any accompanying images.

Data Availability Statement: All relevant data are included in this article. For any further inquiries, please contact the corresponding author.

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Abbreviations

The following abbreviations are used in this manuscript:

LC	Cutaneous loxoscelism
SMD	Sphingomyelinase D
CVL	Cutaneous-visceral loxoscelism
AKI	Acute Kidney Injury
Hb	Hemoglobin
Hct	Hematocrit
WBC	White Blood Cells
Plt	Platelets
PT	Prothrombin Time
INR	International Normalized Ratio
TT	Thrombin Time
LDH	Lactate Dehydrogenase
AST(TGO)	Aspartate Aminotransferase
ALT (TGP)	Alanine Aminotransferase
Na	Sodium
K	Potassium
Cl	Chloride
pCO_2	Partial pressure of carbon dioxide
HCO3	Bicarbonate
ICU	Intensive Care Unit
DAH	Diffuse Alveolar Hemorrhage

References

1. Sánchez-Olivas MA, Valencia-Zavala MP, Sánchez-Olivas JA, Sepulveda-Velázquez G, Vega-Robledo G. Loxoscelismo cutáneo necrótico. Informe de un caso. Rev Alerg México. 2011;58(3):171-176.
2. Droppelmann, K.; Majluf-Cáceres, P.; Sabatini-Ugarte, N.; Valle, E.; Herrera, H.; Acuña, D. Caracterización clínica y epidemiológica de 200 pacientes con loxoscelismo cutáneo y cutáneo visceral. *Rev. Medica De Chile* **2021**, *149*, 682–688, https://doi.org/10.4067/s0034-98872021000500682.
3. Lopes, P.H.; Squaiella-Baptistão, C.C.; Marques, M.O.T.; Tambourgi, D.V. Clinical aspects, diagnosis and management of Loxosceles spider envenomation: literature and case review. *Arch. Toxicol.* **2020**, *94*, 1461–1477, https://doi.org/10.1007/s00204-020-02719-0.
4. Albuquerque, P.L.M.M.; Tessarolo, L.D.; Menezes, F.H.; de Lima, T.B.; Paiva, J.H.H.G.L.; Júnior, G.B.d.S.; Martins, A.M.C.; Daher, E.d.F. Acute kidney injury due to systemic Loxoscelism: a cross-sectional study in Northeast Brazil. *Rev. da Soc. Bras. de Med. Trop.* **2018**, *51*, 695–699, https://doi.org/10.1590/0037-8682-0465-2017.
5. Severino, F.B.; Vivanco, P.; Mix, A. Loxoscelismo: revisión de la literatura a propósito de un caso. **2022**, *47*, 29–35, https://doi.org/10.11565/arsmed.v47i2.1894.
6. Rodríguez, C.A.; Martínez, N.M.H. Complicaciones de la picadura de araña Loxosceles fugescens. *Med. Gen. y de Fam.* **2024**, *13*, 175–178, https://doi.org/10.24038/mgyf.2024.043.
7. S., R.J.; I., H.H.; P., A.V. Loxoscelismo cutáneo-visceral por mordedura de araña Loxosceles laeta en niños: factores asociados. *Rev. De Investig. Veter- Del Peru* **2023**, *34*, e24949–e24949, https://doi.org/10.15381/rivep.v34i6.24949.
8. Isbister, G.K.; Fan, H.W. Spider bite. *Lancet* **2011**, *378*, 2039–2047, https://doi.org/10.1016/s0140-6736(10)62230-1.
9. Sampaio, V.S.; Gomes, A.A.; Silva, I.M.; Sachett, J.; Ferreira, L.C.L.; Oliveira, S.; Sabidò, M.; Chalkidis, H.; Guerra, M.G.V.B.; Salinas, J.L.; et al. Low Health System Performance, Indigenous Status and Antivenom Underdosage Correlate with Spider Envenoming Severity in the Remote Brazilian Amazon. *PLOS ONE* **2016**, *11*, e0156386–e0156386, https://doi.org/10.1371/journal.pone.0156386.

 doi:10.20944/preprints202512.2688.v1

10. Gremski, L.H.; da Justa, H.C.; Polli, N.L.C.; Schluga, P.H.d.C.; Theodoro, J.L.; Wille, A.C.M.; Senff-Ribeiro, A.; Veiga, S.S. Systemic Loxoscelism, Less Frequent but More Deadly: The Involvement of Phospholipases D in the Pathophysiology of Envenomation. *Toxins* **2022**, *15*, 17, https://doi.org/10.3390/toxins15010017.
11. Suárez-Ospino, N.I.; Díaz-Anaya, E.; Ochoa-Díaz, A.F. Dermatological Features as a Diagnostic Clue in Systemic Loxocelism Caused by Brown Recluse Spider Bite. *Am. J. Trop. Med. Hyg.* **2023**, *109*, 1207–1208, https://doi.org/10.4269/ajtmh.23-0246.
12. Mani, S.; Katzman, C.; Liu, V. Histopathology aiding diagnosis of viscerocutaneous loxoscelism in a nonendemic region. *JAAD Case Rep.* **2024**, *45*, 11–17, https://doi.org/10.1016/j.jdcr.2023.12.014.
13. Nguyen, N.; Pandey, M. Loxoscelism: Cutaneous and Hematologic Manifestations. *Adv. Hematol.* **2019**, *2019*, 1–6, https://doi.org/10.1155/2019/4091278.
14. Harry, S.; Brugioni, E.; Madhusudhana, S. Acute Hemolytic Anemia Caused by Loxoscelism Treated With Plasmapheresis: A Case Report. *J. Med Cases* **2022**, *13*, 219–224, https://doi.org/10.14740/jmc3828.
15. Mondal, S.; Nag, A.; Datta, J.; Das, A.; Agarwal, A.; Sinha, D.; Ete, T.; Chakraborty, A.; Ghosh, S. Acute kidney injury and dermonecrosis after Loxosceles reclusa envenomation. *Indian J. Nephrol.* **2014**, *24*, 246–8, https://doi.org/10.4103/0971-4065.133017.
16. Vargas, C.M.; Vásquez, V.F.; Ugarte, R.P. Actualización sobre manejo de araneismo en Perú. *Rev. Medica Hered.* **2017**, *28*, 200, https://doi.org/10.20453/rmh.v28i3.3189.
17. Pauli, I.; Puka, J.; Gubert, I.C.; Minozzo, J.C. The efficacy of antivenom in loxoscelism treatment. *Toxicon* **2006**, *48*, 123–137, https://doi.org/10.1016/j.toxicon.2006.05.005.
18. Malaque, C.M.S.; Novaes, C.T.G.; Piorelli, R.O.; Risk, J.Y.; Murad, J.C.; Lara, A.N.; Virgulino, C.C.; Miyaji, K.T.; Santoro, M.L. Impact of antivenom administration on the evolution of cutaneous lesions in loxoscelism: A prospective observational study. *PLOS Neglected Trop. Dis.* **2022**, *16*, e0010842, https://doi.org/10.1371/journal.pntd.0010842.
19. Azuara-Antonio, O.; Ortiz, M.I.; Jimenez-Oliver, K.D.; Hernandez-Cruz, L.E.; Rivero-Veras, G.; Hernandez-Ramirez, L. Clinical Evolution After Administering Antivenom in Patients With Loxoscelism. *J. Med Cases* **2023**, *14*, 378–386, https://doi.org/10.14740/jmc4163.
20. Salud (ETESA) CM de SD de P y PD de C de PU de E de T de. Efectividad del suero anti-loxosceles: síntesis de evidencia. Published online 2004:20-20.