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Case Report

Acute Kidney Injury Requiring Prolonged Kidney Replacement Therapy After *Loxosceles* Envenomation in an Adolescent: A Case Report with 14-Month Follow-Up

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Abstract

Loxoscelism is the clinical syndrome that follows envenomation by spiders of the genus *Loxosceles*. The venom is dominated by phospholipases D, which produce dermonecrosis, intravascular hemolysis, thrombocytopenia, and renal failure in experimental models. Acute kidney injury (AKI) is the least common of the systemic manifestations and the most lethal, and children account for a disproportionate number of deaths. Here, we present a case report of a previously healthy 15-year-old boy bitten on the left lower limb during a trip to Monterrey, in northeastern Mexico. He presented four days later with severe limb pain, progressive necrotic skin lesions, and shock, and was admitted to the pediatric intensive care unit with multiorgan failure. The nephrology service documented KDIGO stage 3 AKI, with a serum creatinine of 6.5 mg/dL, an estimated glomerular filtration rate of 9.9 mL/min/1.73 m², and a urine sediment showing uncountable erythrocytes and granular casts. Creatine kinase peaked at 89,637 U/L, with disseminated intravascular coagulation, hemolysis, and thrombocytopenia; blood cultures grew *Streptococcus pyogenes*. He received four sessions of continuous kidney replacement therapy followed by intermittent hemodialysis over four weeks, alongside fasciotomy, serial debridement with negative-pressure wound therapy, and a split-thickness skin graft, and was discharged after 59 days. The two filtration markers did not agree: before discharge the estimated glomerular filtration rate was 118.4 mL/min/1.73 m² by creatinine and 43.1 mL/min/1.73 m² by cystatin C. The gap narrowed over the following year, and at fourteen months the estimated glomerular filtration rate was 98 mL/min/1.73 m² without proteinuria. Because dialysis-treated AKI in the setting of multiorgan failure carries a long-term risk of chronic kidney disease and hypertension, and because creatinine alone may overestimate recovery after rhabdomyolysis, recovery of glomerular filtration should not be taken as the end point of nephrology follow-up.

Keywords: *Loxoscelism; acute kidney injury; cystatin C; kidney replacement therapy; case report.*

Introduction

Loxoscelism is a clinical syndrome caused by the bite of spiders of the genus *Loxosceles*, a group of small, reclusive arachnids distributed across the Americas and parts of Africa, southern Europe, and Oceania. Envenomation presents in two forms. The cutaneous form is common and rarely threatens life, while the systemic or cutaneous-visceral form is far less frequent and accounts for essentially all mortality [1]. Mexico harbors a large number of described species, and bites concentrate in the north and center of the country during the warm months.

Most envenomations remain confined to the skin. In the largest Latin American series, 232 of 267 notified cases (86.9%) were purely cutaneous, whereas 35 patients (13.1%) developed the cutaneous-visceral form with intravascular hemolysis. Acute renal failure appeared in 17 patients (6.4%), and all four deaths were in children younger than 14 years [2]. This age distribution is the reason loxoscelism concerns the pediatric nephrologist at all.

The venom is dominated by a family of phospholipases D (PLDs), enzymes originally characterized as sphingomyelinase D. Under experimental conditions they reproduce almost the entire clinical picture: local inflammation and dermonecrosis, thrombocytopenia, intravascular hemolysis, and renal failure [1]. The renal evidence is unusually direct. Mice given recombinant wild-type PLD develop glomerular edema, collapse of Bowman's space, and proteinaceous deposits within the tubular lumen, together with hematuria and rising blood urea, whereas an isoform mutated in the catalytic domain produces none of this, which ties nephrotoxicity to catalytic activity rather than to a nonspecific inflammatory effect [3]. Work with *L. gaucho* venom in rats adds a hemodynamic dimension: glomerular filtration rate and renal blood flow fell sharply within an hour of venom infusion and renal vascular resistance rose, with immunohistochemical deposition of myoglobin and hemoglobin, yet freshly isolated proximal tubules exposed to venom in vitro were not injured [4]. Human proximal tubular cells, in contrast, do undergo apoptosis after exposure to venom or recombinant sphingomyelinase D, in parallel with secretion of matrix metalloproteinases 2 and 9 [5]. These mechanisms are best read as additive rather than mutually exclusive.

Clinically, AKI staged by the KDIGO criteria was documented in 13.3% of an unselected Brazilian cohort of 45 patients with loxoscelism [6]. Dialysis-requiring AKI has been reported from Brazil, India, and the United States [7–9]. Mexican descriptions remain scarce, and in our review of the published literature we found no report of an adolescent who required continuous kidney support and subsequently recovered renal function. Our aim is to describe the renal and surgical course of such a patient, and to set out what dialysis-treated AKI of this severity implies for medium- and long-term nephrology follow-up.

Case Presentation

A previously healthy 15-year-old boy with no relevant personal or family history was bitten on the left lower limb on 24 May 2025 during a trip to Monterrey in northeastern Mexico. He did not see the spider clearly and did not bring it for further identification. Pain in the left knee and leg began shortly after the bite.

Over the following days the pain intensified, and necrotic skin lesions appeared over the affected limb. He deteriorated systemically and was first assessed at a general hospital in the State of Mexico, where multiorgan failure was recognized and a shock state was managed as septic shock. He was subsequently referred to the Hospital Infantil de México Federico Gómez and admitted to the pediatric intensive care unit on 28 May 2025.

On arrival he was hemodynamically unstable on three vasopressors, with a blood pressure of 90/45 mmHg and a capillary refill time of seven seconds, and was intubated for respiratory distress. Extensive necrotic lesions were observed in the left lower limb, with vesicles extending distally to the foot, and over the following days, they extended proximally toward the pelvis and both upper limbs, predominantly on the left (Figures 1 and 2). Distal pulses were palpable but diminished, and Doppler study showed preserved flow in both limbs.

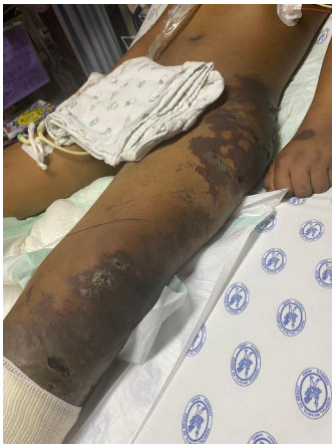

Figure 1. Left lower limb during the first days of admission, showing confluent dusky discoloration, bullae, and progressive necrosis; surgical marking outlines the advancing border.

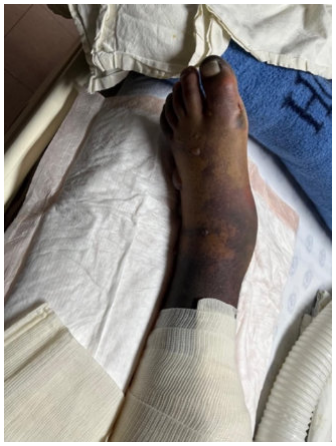

Figure 2. Distal extension of the cutaneous involvement to the left foot, with dusky discoloration and early bulla formation.

The pediatric nephrology service documented AKI, KDIGO stage 3, based on oliguria below 0.3 mL/kg/h sustained over the first 24 hours together with a serum creatinine that had risen from 4.76 mg/dL at the referring hospital to 6.5 mg/dL on arrival, giving an estimated glomerular filtration rate of 9.9 mL/min/1.73 m² [10]. The urine sediment showed uncountable erythrocytes and granular casts. Laboratory studies showed a peak creatine kinase of 89,637 U/L, consistent with massive rhabdomyolysis, and disseminated intravascular coagulation with active bleeding: prothrombin time 14.4 s, INR 1.24, activated partial thromboplastin time rising to 115.8 s, D-dimer 16,468 ng/mL, and fibrinogen falling to 175 mg/dL. Lactate dehydrogenase rose to 2,781.8 U/L and aminotransferases to AST 948 U/L and ALT 131 U/L, while platelets fell from 118 to 30 ×10³/μL and hemoglobin from 14.2 to 6.8 g/dL, requiring repeated transfusion. Blood cultures grew *Streptococcus pyogenes*, and streptococcal toxic shock was diagnosed alongside the envenomation. Urine output and serum creatinine, rather than the biochemical markers of hemolysis alone, drove the decision to begin kidney support. The laboratory course is summarized in Table 1.

Given the progressive loss of kidney function, together with fluid accumulation and the solute load generated by ongoing muscle breakdown, continuous kidney replacement therapy was initiated. He received four sessions and, from 12 June 2025, was changed to intermittent hemodialysis on a Tuesday–Thursday–Saturday schedule; the final session was on 24 June 2025, four weeks after admission. In parallel, the surgical teams performed a fasciotomy of the affected limb on 30 May, two debridements with surgical washout and negative-pressure wound therapy on 12 and 26 June, and later a split-thickness skin graft to the left lower limb, harvested from the right leg, on 3 July (Figure 3).

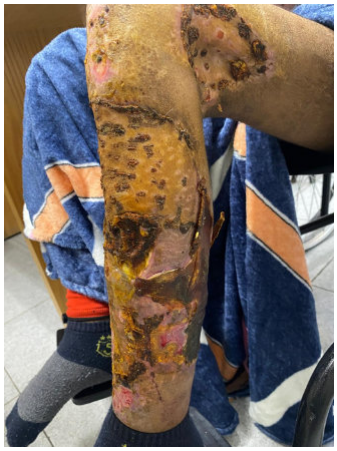

Figure 3. Left lower limb after fasciotomy and serial debridement, showing the extent of the necrotic and granulating surface before definitive skin grafting.

Recovery was not linear. On 5 July serum creatinine rose again to 4.52 mg/dL in the context of acute pyelonephritis due to multidrug-resistant *Klebsiella pneumoniae*, and fell thereafter without a further requirement for dialysis. Blood pressure remained at the 90th percentile for age and sex, between 117/44 and 145/80 mmHg; hydralazine was started and later changed to oral verapamil, with which readings returned to the 50th percentile. He was discharged on 26 July 2025, after 59 days in hospital, on verapamil, cholecalciferol, calcium polystyrene sulfonate, a phosphate binder, and low molecular weight heparin due to thrombotic risk.

Before discharge, kidney function was assessed with both filtration markers. Cystatin C was 2.01 mg/L, and using the CKiD U25 equations the estimated glomerular filtration rate was 118.4 mL/min/1.73 m² when calculated from creatinine but 43.1 mL/min/1.73 m² when calculated from cystatin C [11]. Enoxaparin was dosed on the cystatin C estimate rather than the creatinine one. Body weight fell from 56 kg on admission to 46 kg, and anthropometry documented malnutrition, with arm circumference and skinfold z-scores of −2.41 and −13.67; the nephrology team recorded a high creatinine–cystatin C gap and a considerable loss of renal functional reserve.

He has since been followed in the outpatient pediatric nephrology clinic. Serum creatinine was 0.59 mg/dL at two months and 0.50 mg/dL at six months, when cystatin C had improved to 1.37 mg/L and the urine protein-to-creatinine ratio was 0.12 mg/mg. At that visit the estimated glomerular filtration rate was 52 mL/min/1.73 m² by cystatin C and 90 mL/min/1.73 m² by the combined creatinine–cystatin C equation, a substantially narrower gap than before discharge. At eleven months serum creatinine was 0.62 mg/dL and the urinalysis was negative for protein and blood, with a sediment free of casts, erythrocytes, and leukocytes. Verapamil was reduced from 100 mg to 40 mg every eight hours as blood pressure normalized. At fourteen months serum creatinine was 0.66 mg/dL with an estimated glomerular filtration rate of 98 mL/min/1.73 m², and serum bicarbonate, albumin, and hemoglobin were normal (Table 1).

At the outpatient visit in July 2026, approximately fourteen months after envenomation, the grafted areas of the left lower limb had healed completely, with no residual ulceration or open wounds. The reconstructed skin was hyperpigmented and retained the reticular pattern of the meshed graft, with hypertrophic scar bands over the anterolateral aspect of the leg and around the knee, and hair regrowth over the intervening non-grafted skin (Figures 4 and 5). He was ambulatory and remained under periodic nephrology surveillance.

Figure 4. Left lower limb at outpatient follow-up in July 2026, approximately 14 months after envenomation. The grafted areas healed completely, with hyperpigmentation and residual hypertrophic scarring over the anterolateral leg and knee.

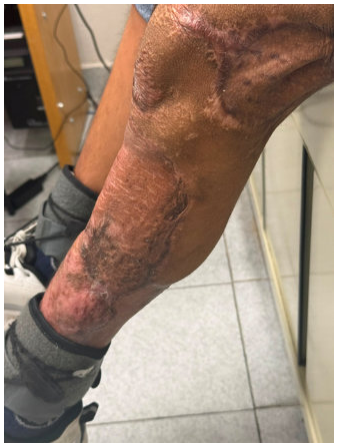

Figure 5. Detail of the grafted region at 14 months after envenomation, showing mature, well-integrated skin with a reticular pattern of the meshed graft, hypertrophic bands along the anterolateral leg, and hair regrowth over the intervening non-grafted skin.

Table 1. Laboratory course from admission to fourteen-month follow-up. Values are those recorded in the clinical record; dashes indicate the analyte was not measured at that time point.

Analyte (units)	Admission — 28 May 25	Peak / nadir — 29–31 May 25	2 months — 21 Jul 25	6 months — 8 Dec 25	14 months — 28 Jul 26
Creatinine (mg/dL)	6.5	6.46	0.59	0.50	0.66
Blood urea nitrogen (mg/dL)	69.1	73.7	37.0	14.9	19.8
Cystatin C (mg/L)	—	—	2.01*	1.37	—
eGFR, creatinine (mL/min/1.73 m²)	9.9	—	118.4*	—	98
eGFR, cystatin C (mL/min/1.73 m²)	—	—	43.1*	52	—
eGFR, creatinine–cystatin C (mL/min/1.73 m²)	—	—	—	90	—
Creatine kinase (U/L)	—	89,637	—	—	—
Lactate dehydrogenase (U/L)	358.8	2,781.8	196.8	—	178.0
Total bilirubin (mg/dL)	3.44	7.65	0.51	—	—
Platelets (×10³/µL)	118	30	725	352	324

Table 1. Continued...

Analyte (units)	Admission — 28 May 25	Peak / nadir — 29–31 May 25	2 months — 21 Jul 25	6 months — 8 Dec 25	14 months — 28 Jul 26
Hemoglobin (g/dL)	14.2	6.8	10.2	13.7	16.2
AST / ALT (U/L)	70 / —	948 / 131	41 / 62	—	—
aPTT (s)	44.5	115.8	—	—	—
D-dimer (ng/mL)	13,547	16,468	—	—	—
Fibrinogen (mg/dL)	—	175	—	—	—
Albumin (g/dL)	1.9	1.8	3.1	4.2	4.6
Bicarbonate (mmol/L)	—	—	20.7	25.8	27.0

*Measured 16 July 2025. AST, aspartate aminotransferase; ALT, alanine aminotransferase; aPTT, activated partial thromboplastin time.

Table 2. Timeline of the episode of care.

Date	Event
24 May 2025	Spider bite to the left lower limb during travel to Monterrey; pain in the knee and leg within hours.
24–27 May 2025	Progressive pain and necrotic skin lesions; assessed at a general hospital, where multiorgan failure was recognized.
28 May 2025	Admission to the pediatric intensive care unit. KDIGO stage 3 AKI (creatinine 6.5 mg/dL, eGFR 9.9 mL/min/1.73 m²); mechanical ventilation and three vasopressors; continuous kidney replacement therapy started.
30 May 2025	Peak creatine kinase 89,637 U/L. Fasciotomy of the left lower limb.
12 June 2025	Change to intermittent hemodialysis. First debridement with negative-pressure wound therapy.
24 June 2025	Final hemodialysis session, four weeks after admission.
26 June 2025	Second debridement with negative-pressure wound therapy.
3 July 2025	Split-thickness skin graft to the left leg, harvested from the right leg.
5 July 2025	Creatinine rose again to 4.52 mg/dL with *Klebsiella pneumoniae* pyelonephritis; no further dialysis required.
16–21 July 2025	Paired filtration markers: eGFR 118.4 mL/min/1.73 m² by creatinine, 43.1 by cystatin C.
26 July 2025	Discharge after 59 days, on verapamil and supportive therapy.
8 December 2025	Cystatin C 1.37 mg/L; urine protein-to-creatinine ratio 0.12 mg/mg.
28 April 2026	Urinalysis negative for protein and blood; sediment clear.
16 June 2026	Complete healing of the grafted areas; ambulatory.
28 July 2026	Creatinine 0.66 mg/dL, eGFR 98 mL/min/1.73 m²; normal bicarbonate, albumin, and hemoglobin.

Discussion

Acute kidney injury is the least frequent systemic complication of loxoscelism and is responsible for its mortality [1]. Most patients never leave the cutaneous stage; a minority progresses to the cutaneous-visceral form, characterized by intravascular hemolysis, coagulopathy, rhabdomyolysis, and renal involvement [2,6]. This case adds to the literature on three points: the duration of kidney support required, the recovery of renal function after it, and follow-up extending beyond the first year.

The diagnosis deserves scrutiny because this constellation of findings is not unique to envenomation. Necrotic skin lesions accompanied by hemolysis, coagulopathy, and acute kidney injury can also follow severe sepsis with disseminated intravascular coagulation and purpura fulminans, necrotizing soft tissue infection, other envenomations, or small-vessel vasculitis. Several features favored loxoscelism here: the lesion began at a single inoculation site after a witnessed arachnid bite, necrosis spread with the gravitational pattern described for this envenomation, and the patient had traveled to an endemic region in northern Mexico. The diagnosis remains clinical rather than laboratory-confirmed.

In this patient, AKI could not be attributed to a single mechanism. Hypoperfusion from the shock state would independently produce acute tubular injury. Rhabdomyolysis was extreme, with a creatine kinase of 89,637 U/L; França et al. showed in two Brazilian patients that muscle breakdown after *Loxosceles* bites, with creatine kinase levels of 6,841 and 1,718 U/L, contributes independently to renal failure, one of their patients requiring 50 days of dialysis [7]. Pigment nephropathy from circulating myoglobin and hemoglobin follows, and this is precisely the pattern Lucato et al. reproduced experimentally, with immunohistochemical deposition of both pigments in rat kidneys [4]. Superimposed on all of this is the catalytic nephrotoxicity of phospholipase D itself [3,5]. A second insult followed six weeks later, when pyelonephritis due to a multidrug-resistant organism drove creatinine back above 4 mg/dL.

The shock had at least two contributors. *Streptococcus pyogenes* was isolated from blood cultures and streptococcal toxic shock was diagnosed, so bacterial sepsis was real and required source control and antimicrobial therapy. Recombinant sphingomyelinase D, however, also activates human leukocytes and consumes complement in whole-blood models, producing a systemic inflammatory response that closely resembles endotoxic shock [12]. An extensive necrotic wound is simultaneously a portal of entry for bacteria and a reservoir of venom-modified tissue, and the two mechanisms are not mutually exclusive. Isolating an organism does not exclude a venom-mediated component, and in practice it did not alter the renal management.

Here, the outcome contrasts with the published fatalities. Rosen et al. described death from systemic loxoscelism less than one day after envenomation, before renal support could be delivered [13]. Lung and Mallory reported a 7-year-old with brown urine and glomerulonephritis in whom the diagnosis was reached only once the renal picture had declared itself [14]. The most plausible difference is timing and access rather than any property of the envenomation itself. Our patient reached a center with continuous kidney replacement capability while he was still salvageable, and support was continued for as long as the injury required, rather than being weaned on a fixed schedule.

Kidney replacement therapy is not the only treatment option available. Said et al. used plasma exchange in a 6-year-old child with massive hemolysis, principally to clear the plasma, so that routine laboratory monitoring became possible during continuous venovenous hemodiafiltration [15]. Anwar et al. reported the only published renal biopsy in a human with loxoscelism-associated renal failure, which remains the strongest histological evidence in humans [9]. Neither approach was required in this case, but both are worth considering when hemolysis is overwhelming or the renal course is atypical.

Surgical management was as important as renal management in this patient. Necrotic tissue is a continuing source of myoglobin, inflammatory mediators, and secondary infection, and acute compartment syndrome that can accompany severe local envenomation has been described in Mexico by Sánchez-Pérez et al., with a favorable outcome after fasciotomy [16]. Delaying decompression would have prolonged the muscle injury that was driving the pigment load on the kidney. The 13-month images show the cost of that necrotic burden even after successful reconstruction: extensive grafted skin with mature hypertrophic scarring, a reminder that renal recovery and functional recovery are not the same endpoint.

The point we most want to make concerns what happens after discharge, and our own patient illustrates it. An estimated glomerular filtration rate of 98 mL/min/1.73 m² is reassuring, but it is not proof of renal recovery. Before discharge his creatinine-based estimate was 118.4 mL/min/1.73 m², while the simultaneous cystatin C–based estimate was 43.1 mL/min/1.73 m², within the range of stage 3b chronic kidney disease [11]; after a creatine kinase of 89,637 U/L and a fall in body weight from 56 to 46 kg, creatinine underestimates renal impairment, and a single normal value should not be read as proof of recovery. Among 1,688 Canadian children who survived dialysis-treated AKI, 13.1% developed chronic kidney disease and 12.1% developed hypertension over a median follow-up of 9.6 years, with the greatest hazard in the first year after discharge [17]. A recent meta-analysis of 39 studies and 16,151 children placed the pooled cumulative incidence of chronic kidney disease after pediatric AKI at 17%, and stage 2–3 AKI carried nearly three times the odds of stage 1 (OR 2.84; 95% CI 1.49–4.15) [18]. Our patient sits squarely in that higher-risk group, and he required an antihypertensive before discharge. There is a second reason for extended surveillance specific to this envenomation: DiPaola et al. documented hemolysis appearing six days after the bite, after symptoms and laboratory values had already improved [19]. For both reasons, the patient remains in nephrology follow-up with serial creatinine, cystatin C, urine protein-to-creatinine ratio, and blood pressure measurements rather than being discharged after the initial recovery.

The strengths of this report are the completeness of the biochemical record, which allowed the renal course to be reconstructed from admission to fourteen months, and the availability of paired creatinine and cystatin C measurements at two separate time points. Antihypertensive therapy was well tolerated and was progressively reduced as blood pressure normalized, and the patient has attended scheduled nephrology follow-up throughout. This report also has limitations. The spider was never captured; therefore, the diagnosis rested on the clinical syndrome and epidemiological context, the same limitation reported in most published series and case reports of loxoscelism [2,6,8]. Species-specific antivenom was not administered. No renal biopsy was performed because the clinical trajectory did not justify the bleeding risk in a patient with active disseminated intravascular coagulation, and the concurrent streptococcal toxic shock and the later episode of pyelonephritis make it impossible to apportion the renal injury between venom, hypoperfusion, pigment load, and sepsis. Cystatin C was not measured beyond six months, so the later course of the creatinine–cystatin C divergence is unknown.

Conclusion

Loxoscelism belongs in the differential diagnosis of acute kidney injury in children and adolescents from endemic regions, particularly when necrotic skin lesions, hemolysis, and rhabdomyolysis appear together. Renal involvement varies in severity and may become severe enough to require kidney replacement therapy; this case illustrates that such an episode can still resolve when support is started early and the necrotic burden is addressed surgically. Recovery of glomerular filtration should not end the nephrology relationship: AKI of this severity predicts chronic kidney disease and hypertension years later, creatinine alone may overestimate recovery when muscle mass has been lost, and structured long-term surveillance is a reasonable standard of care for these patients.

Ethics Statement

Written informed consent was obtained from the patient's legal guardian, and written assent was obtained from the patient, for the publication of this case report and the accompanying clinical images. All directly identifying information has been removed. This study was conducted in accordance with the principles of the Declaration of Helsinki. According to institutional regulations, formal ethical committee approval was not required for a single anonymized case report.

Conflict of Interest

The authors declare no conflicts of interest related to the content of this article.

Author Contributions

– **Víctor Manuel Barajas Valencia:** Attending physician directly responsible for the clinical care of the patient; manuscript review and approval.

– **Gustavo Eduardo Aurioles Amozurrutia:** Literature search and appraisal of the evidence, bibliographic verification, review of the clinical record, writing of the final manuscript, preparation of figures and table, project coordination, and correspondence.

– **Abil Sofía Campos García:** Preparation of the preliminary version of the manuscript and literature review.

– **Irma Esther del Moral Espinosa:** Nephrology care of the patient during hospitalization and outpatient follow-up; manuscript review and approval.

– **Rebeca Gómez Chico Velasco:** Project leadership, supervision of the report, manuscript review, and refinement.

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