REVIEW ARTICLE

Clinical Practice Recommendations: Snakebite-envenomation-associated Acute Kidney Injury

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Abstract

Background: Snakebite is a neglected tropical disease of major public health importance in India, contributing to significant morbidity and mortality. Acute kidney injury (AKI) is among the most serious complications of envenomation, particularly with viperid and some elapid species, and is associated with long-term chronic kidney disease (CKD) risk.

Objective: This article aims to provide evidence-based recommendations for the recognition, evaluation, and management of snakebite-envenomation-associated AKI (SAKI), integrating global best practices with national treatment protocols and resource realities.

Materials and methods: The authors reviewed available literature through PubMed and international recommendations, including the Government of India's Standard Treatment Guidelines (STG), World Health Organization (WHO) guidance, and Kidney Disease Improving Global Outcomes (KDIGO) AKI guidelines. As per the available evidence, the recommendations were formulated through expert panel deliberations.

Results: Key recommendations include early recognition of envenomation syndromes, prompt administration of polyvalent antisnake venom (ASV), protocolized monitoring of coagulation and renal parameters, and standardized use of kidney replacement therapy (KRT) according to KDIGO indications. Supportive management of venom-induced consumption coagulopathy (VICC), rhabdomyolysis, and thrombotic microangiopathy (TMA) is emphasized. Special considerations have been outlined for children, pregnant women, patients with preexisting kidney disease, and those bitten by non-"Big Four" snakes.

Conclusion: SAKI is preventable and treatable with timely intervention, but survivors remain at risk of CKD. With these recommendations, the authors have tried to standardize the diagnostic and therapeutic approach across general medicine, nephrology, and critical care medicine teams in India, aiming to reduce mortality and improve renal outcomes.

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Introduction

Snakebite envenomation is recognized by the WHO as a high-priority neglected tropical disease, with India bearing the highest global burden, accounting for an estimated 50,000–60,000 deaths annually, along with long-term disability among the survivors.[1–4] Among the systemic complications of envenomation, acute kidney injury (AKI) is particularly significant, both as a determinant of in-hospital mortality and as a contributor to chronic kidney disease (CKD).[1,5,6] The pathophysiology of snakebite-envenomation-associated AKI (SAKI) is multifactorial, involving venom-induced consumption coagulopathy (VICC), direct nephrotoxicity, pigment nephropathy from hemolysis or rhabdomyolysis, hemodynamic shock, thrombotic microangiopathy (TMA), and, in severe cases, acute cortical necrosis (ACN).[7–9] Indian data indicate that AKI develops in up to one-third of hospitalized snakebite patients, with 20–30% requiring kidney replacement therapy (KRT). Nevertheless, 20–40% of survivors demonstrate persistent renal impairment, highlighting the need for structured follow-up.[10–14] Despite the high burden, standardized nephrology-specific guidelines for the prevention and management of SAKI have been lacking in India. Existing protocols focus largely on antivenom administration and general supportive care, with limited emphasis on renal complications, dialysis practices, or long-term outcomes. To address this gap, the authors convened a multidisciplinary committee to develop evidence-based recommendations specific to SAKI. These recommendations are intended for use by general physicians, nephrologists, intensivists, emergency physicians, and dialysis teams across India. They integrate international best practices, including WHO and Kidney Disease Improving Global Outcomes (KDIGO) recommendations, with national realities such as the availability of polyvalent antivenom (ASV), variable access to kidney replacement modalities, and the high prevalence of rural presentations. The overarching aim is to improve early recognition, standardize treatment pathways, and optimize renal outcomes in patients with snakebite envenomation, while ensuring uniformity of practice across diverse healthcare settings.

Materials and Methods

The authors undertook a structured literature search and evidence synthesis. A comprehensive PubMed search was conducted up to July 2025 using the keywords "snakebite," "envenomation," "acute kidney injury," "renal failure," "venom-induced consumption coagulopathy," and "dialysis." Additional references were drawn from the WHO guidelines on snakebite management,[15] the Government of India Standard Treatment Guidelines (STG) for Snakebite,[16] and the KDIGO AKI guideline.[17] Only English-language publications were

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Table 1: Envenomation syndromes relevant to AKI

Syndrome and Indian snakes	*Key clinical markers*	*Kidney injury mechanisms*	*First-hour priorities (additions to standard ABCs)*	*Remarks*
Hemotoxic/Viperid (Russell's viper, Saw-scaled viper; some pit vipers)	Local swelling, bleeding, oozing, gingival bleed; deranged 20WBCT, low fibrinogen, increased D-dimer, prolonged PT/ aPTT	VICC with microthrombi/ bleeding; hypotension; hemolysis; ATN; AIN; TMA; ACN	Early polyvalent ASV (initial 10 vials in most; 5 vials may be considered for suspected saw-scaled viper), aggressive monitoring, early laboratory tests (CBC, PT/INR, fibrinogen, D-dimer), urine output target ≥0.5 mL/kg/h	Coagulopathy may persist/recur; re-dose guided by laboratory test results/bleeding
Neurotoxic/Elapid (Cobra, Krait)	Ptosis, ophthalmoplegia, bulbar weakness, descending paralysis ± respiratory failure	Rhabdomyolysis (cobra/ sea snake), hypoxia/ aspiration; ATN	Airway and ventilation; early ASV (10 vials stat; repeat if worsening after 1–2 hours); consider atropine-neostigmine test for postsynaptic blocks	Presynaptic (krait) paralysis responds poorly to anticholinesterase
Myotoxic (Sea snakes; some cobras)	Severe myalgia, dark urine, very high CK	Myoglobin-induced tubular injury; hyperkalemia	Early fluids (if no overload), urine alkalinization for significant myoglobinuria (expert practice); monitor potassium closely	Indian polyvalent ASV does not cover sea snake venoms
TMA phenotype (various vipers; Hypnale)	MAHA (increased LDH, schistocytes), thrombocytopenia, AKI often after initial VICC	Endothelial injury; may overlap with VICC	Supportive care; dialysis as needed; routine plasma exchange not supported; consider only if true TTP (ADAMTS13 <10%) suspected	Biopsy series show TMA/ACN in a subset

ABC, airway, breathing, and circulation; ACN, acute cortical necrosis; ADAMTS13, a disintegrin and metalloproteinase with thrombospondin type 1 motif, member 13; AIN, acute interstitial nephritis; aPTT, activated partial thromboplastin time; ASV, antisnake venom; ATN, acute tubular necrosis; CBC, complete blood count; CK, creatine kinase; MAHA, microangiopathic hemolytic anemia; PT, prothrombin time; TMA, thrombotic microangiopathy; TTP, thrombotic thrombocytopenic purpura; VICC, venom-induced consumption coagulopathy; 20WBCT, 20-minute whole blood clotting test

included. Priority was given to randomized controlled trials (RCTs), meta-analyses, and large cohort studies where available; however, given the scarcity of high-quality trials in this field, observational studies, registry data, and case series from India and other tropical regions were also reviewed. Evidence synthesis was performed with a focus on the epidemiology and burden of snakebite-associated AKI; mechanisms of renal injury; the role of antivenom and supportive therapy; dialysis and extracorporeal support; and long-term outcomes. The recommendations represent a synthesis of best available evidence and expert opinion, contextualized to the realities of nephrology and critical care practice in India.

Background

India contributes a large share of the global snakebite burden, with hemotoxic (viperid) and neurotoxic (elapid) bites predominating.[1–4] AKI occurs in up to ~1/3 of hospitalized victims and may require KRT.[10–14] SAKI mechanisms include VICC, hemodynamic shock, rhabdomyolysis/ myoglobinuria, hemoglobinuria, direct tubular toxicity, acute interstitial nephritis, TMA, and (less often) acute cortical necrosis.[7–9] Various envenomation syndromes relevant to AKI have been depicted in Table 1. Long-term outcomes are not benign—many of them have persistent renal abnormalities; a wide range (8–50%) can progress to CKD, supporting structured follow-up.[6,7,10,18]

Definition

Snakebite-envenomation-associated AKI refers to the development of AKI following confirmed or probable snake envenomation, after exclusion of alternative causes of renal dysfunction. AKI is defined and staged according to the KDIGO criteria,[17] based on changes in serum creatinine and/or urine output. The term also includes patients requiring KRT and those with partial or delayed renal recovery.

Initial Evaluation and Risk Stratification (First 0–6 Hours)

Prehospital/Triage

Reassure the patient and immobilize the bitten limb at heart level. No tourniquet, incision, suction, or ice should be applied over the affected limb. Remove the tight items and transport rapidly to an ASV-capable facility.[19–22]

Emergency Department/ICU Bundle

- Record the time, site, and species. Monitor vitals (blood pressure/pulse rate, rhythm, and volume/respiratory rate/peripheral oxygen saturation).
- Secure airway/ventilation (neurotoxic bites), large-bore peripheral/ central venous catheter, hemodynamic stabilization (balanced crystalloids; vasopressors if shock). Avoid dopamine infusion.
- Insert Foley's catheter for accurate monitoring of urine output; maintain hydration and target urine output ≥ 0.5 mL/kg/h unless anuric/overloaded. Alkalinize urine for pigmenturia with 100 mL of 8.4% sodium bicarbonate in 1 liter of 5% dextrose at 100 mL/h.
- Laboratory panel and monitoring (Table 2).
- Early polyvalent ASV according to the Indian STG (vide infra).[16]
- Avoid nephrotoxins [nonsteroidal anti-inflammatory drugs (NSAIDs), aminoglycosides, radiocontrast; adjust all drug doses to kidney function].
- A practical management bundle for SAKI in ICU/critical care has been summarized in Table 3.

ASV: Indications, Dosing, and Re-dosing

Antisnake venom is the only specific therapy capable of neutralizing circulating snake venom and remains the cornerstone of management in snakebite envenomation.[7,23] Early, adequate, and appropriately re-dosed ASV administration is critical in preventing systemic complications, including SAKI.[7,23–25]

Table 2: Diagnostic work-up and monitoring schedule in suspected SAKI

Domain	*Tests*	*Timing/frequency*	*How it informs decisions*
Coagulation (VICC)	20WBCT at bedside; PT/INR, aPTT, fibrinogen, D-dimer	Baseline; then q6h until normalization for 24 hours; earlier if bleeding	Guides ASV re-dosing in hemotoxic bites; D-dimer is sensitive for VICC
Hemolysis/TMA	CBC with platelets, reticulocyte count, smear for schistocytes; serum LDH, haptoglobin, and bilirubin (MAHA)	Baseline; repeat 12–24 hours if hemolysis is suspected	Distinguishes VICC ± TMA; falling platelets and schistocytes suggest TMA—no routine PEX unless TTP
Renal	Serum creatinine, BUN, electrolytes (potassium, bicarbonate, calcium, phosphate), serum CK, urinalysis (hematuria, myoglobin), urine microscopy, and albumin/protein–creatinine ratio	Baseline; repeat q12–24 hours; more often if unstable	KDIGO staging; detects hyperkalemia, rhabdomyolysis, and pigment nephropathy
Acid-base and lactate	ABG/VBG, serum lactate	Baseline; as per shock status	Guides fluids, vasopressors, and dialysis indications
Imaging	Renal ultrasound at bedside, chest X-ray	Early if oliguria/anuria or pulmonary edema	Rules out obstruction; assesses volume/ARDS
Others	Cross-match early, before ASV/venom effects confound results; pregnancy test where relevant	Early	Transfusion planning; obstetric coordination

ABC, airway, breathing, and circulation; ABG, arterial blood gas analysis; aPTT, activated partial thromboplastin time; ARDS, acute respiratory distress syndrome; ASV, antisnake venom; BUN, blood urea nitrogen; CBC, complete blood count; CK, creatine kinase; LDH, lactate dehydrogenase; MAHA, microangiopathic hemolytic anemia; PEX, plasma exchange; PT, prothrombin time; TMA, thrombotic microangiopathy; TTP, thrombotic thrombocytopenic purpura; VBG, venous blood gas analysis; VICC, venom-induced consumption coagulopathy; 20WBCT, 20-minute whole blood clotting test

Table 3: Practical management bundle for SAKI (ICU/ Critical Care)

Step	*What to do*	*Pearls and pitfalls*
Resuscitation	ABCs; early intubation if bulbar weakness; crystalloids; norepinephrine for shock	Avoid excessive fluids in oligo-anuria; monitor for pulmonary edema
Antivenom	Start promptly; re-dose as per clinical/ laboratory criteria	Do not underdose in children/pregnancy; watch for anaphylaxis
Laboratory tests and monitoring	q6h coagulation (VICC); strict input-output monitoring; creatinine/ electrolytes; CK	Early cross-match before ASV/FFP; bedside 20WBCT where labs are delayed
Coagulopathy	If bleeding/severe procedures: FFP/cryoprecipitate after ASV	Do not rely on blood products alone without ASV
Rhabdomyolysis	Fluids (unless overloaded), early hyperkalemia care; consider alkalinization	Pigment casts: avoid acidemia, avoid nephrotoxins
TMA phenotype	Supportive care; dialysis as needed; evaluate for MAHA	Routine PEX not supported; reserve for proven TTP (ADAMTS13 <10%)
Dialysis	IHD/SLED/CKRT per status; anticoagulation tailored to bleeding risk	Heparin-free or citrate in coagulopathy; run higher effluent in hyperkalemia
Drugs	Renally dose all meds; avoid NSAIDs, aminoglycosides, IV contrast	Antibiotics only if infected wounds/aspiration pneumonia
Documentation	Time of bite, first aid, snake species; ASV batch/dose/timing	Capture for pharmacovigilance, medico-legal, and quality loops

ABC, airway, breathing, and circulation; aPTT, activated partial thromboplastin time; ASV, antisnake venom; CK, creatine kinase; CKRT, continuous kidney replacement therapy; IHD, intermittent hemodialysis; LDH, lactate dehydrogenase; MAHA, microangiopathic hemolytic anemia; NSAID, nonsteroidal anti-inflammatory drug; PEX, plasma exchange; PT, prothrombin time; SLED, sustained low-efficiency dialysis; TMA, thrombotic microangiopathy; TTP, thrombotic thrombocytopenic purpura; VICC, venom-induced consumption coagulopathy; 20WBCT, 20-minute whole blood clotting test

Delay in initiation or under-dosing of ASV is a well-established risk factor for severe coagulopathy, AKI, need for kidney replacement therapy, and mortality.[1,3–7]

Indications

Antivenom should be administered promptly in patients with evidence of systemic envenomation, irrespective of the presence or severity of local symptoms. Indications include the following:[3,4,21,26]

- Hemotoxic envenomation.
  - Abnormal 20WBCT.
  - Spontaneous systemic bleeding (gingival bleed, hematuria, hematemesis, intracranial bleed).
  - Laboratory evidence of venom-induced consumption coagulopathy (prolonged PT/INR, low fibrinogen, elevated D-dimer).
- Neurotoxic envenomation.
  - Ptosis, ophthalmoplegia.
  - Bulbar weakness, dysphagia.
  - Respiratory muscle weakness or impending respiratory failure.
- Myotoxic envenomation.
  - Severe generalized myalgia.
  - Dark-colored urine with markedly elevated creatine kinase.
- Cardiovascular or renal involvement.
  - Hypotension or shock attributable to envenomation.
  - Oliguria/anuria or rising serum creatinine consistent with AKI.
  - ASV is not indicated in dry bites or in patients with isolated local swelling without systemic features.[25,27]

Initial Dosing

Indian polyvalent ASV (effective against cobra, krait, Russell's viper, and saw-scaled

viper) should be administered intravenously after reconstitution, preferably within the first 3–6 hours of envenomation.[4,23–25]

- Hemotoxic (viperid) envenomation.
  - The initial dose should be 10 vials IV infusion.
  - In suspected saw-scaled viper bites with mild systemic involvement, 5 vials may be considered initially, with close monitoring.
- Neurotoxic envenomation.
  - The initial dose should be 10 vials IV infusion.
  - Early airway protection should accompany ASV administration in patients with bulbar or respiratory involvement.
- Children and pregnant women.
  - Same dose as adults, as venom load is independent of body weight.
  - ASV should be infused over 30–60 minutes under close monitoring for anaphylaxis, with resuscitation drugs and equipment readily available.[28–30]

Re-dosing of ASV

Re-dosing is guided by clinical response and laboratory parameters, not by fixed maximum dosing.[4,23–25,31,32]

- Hemotoxic envenomation.
  - Reassess coagulation profile (20WBCT, PT/INR, fibrinogen) at 6 hours after completion of infusion.
  - If coagulopathy persists or bleeding continues, administer an additional 2–6 vials, or repeat a full dose depending on severity.
  - Re-evaluation and re-dosing may be required every 6 hours until coagulation normalizes.
  - In rare, severe cases with ongoing life-threatening bleeding, cumulative doses up to 30 vials may be required.
- Neurotoxic envenomation.
  - If neurological deterioration continues or no improvement is seen within 1–2 hours of the initial dose, repeat 10 vials.
  - Further dosing beyond 20 vials is rarely beneficial and should be individualized.

What ASV Can and Cannot Do

Antisnake venom neutralizes only circulating, unbound venom. It does not reverse established tissue damage, including presynaptic neurotoxicity (e.g., krait bites), established AKI, or local tissue necrosis. Therefore, early administration is essential to prevent progression of organ damage.[25,33]

Safety and Adverse Reactions

Early adverse reactions include anaphylaxis, bronchospasm, hypotension, and urticaria. Late reactions include serum sickness (5–10 days post-ASV).[28,29] ASV reactions should be managed promptly with intramuscular adrenaline, antihistamines, and corticosteroids as per institutional protocols. A history of allergy is not a contraindication to ASV when clinically indicated.[28–30]

Special Considerations

Indian polyvalent ASV is ineffective against sea snake venom and many pit vipers; management in these cases is primarily supportive.[23,34,35] ASV dosing should not be reduced in patients with AKI or CKD.[8,36] Documentation of ASV batch number, dose, timing, and response is essential for pharmacovigilance and quality assurance.[4]

Coagulopathy/Bleeding Care

Coagulopathy following snakebite envenomation, most commonly due to VICC, is a major contributor to morbidity and mortality and a key driver of snakebite-associated AKI.[7–9] VICC is characterized by rapid depletion of clotting factors due to procoagulant toxins in venom, leading to prolonged clotting times, hypofibrinogenemia, elevated D-dimer levels, and a nonclotting 20WBCT. Importantly, VICC is a toxin-mediated process rather than classical disseminated intravascular coagulation (DIC), and management principles differ accordingly.[37–39]

Primary Therapy

Prompt and adequate administration of ASV is the cornerstone of the management of VICC.[37,38] ASV neutralizes circulating venom and halts further consumption of clotting factors but does not immediately correct existing coagulopathy. Re-dosing of ASV should be guided by clinical bleeding and repeat coagulation parameters (20WBCT, PT/INR, fibrinogen), typically assessed every 6 hours in hemotoxic envenomation. Delay or underdosing of ASV significantly increases the risk of severe bleeding, AKI, and the need for dialysis.[37,39,40]

Role of Blood and Blood Products

Blood products should never replace ASV and should be used only as adjunctive therapy.[41] Indications for transfusion include life-threatening or ongoing active bleeding (e.g., intracranial, gastrointestinal, pulmonary, retroperitoneal) or preparation for urgent invasive procedures (surgery, dialysis catheter insertion). The recommended blood products are fresh frozen plasma (FFP) to replace clotting factors and cryoprecipitate for severe hypofibrinogenemia (<100 mg/dL).[41–45] Platelet transfusion should only be attempted in the presence of significant thrombocytopenia with active bleeding. Routine prophylactic use of blood products in isolated laboratory coagulopathy without bleeding is discouraged, as it does not improve outcomes and may worsen volume overload, particularly in AKI.

Dialysis and Procedures in Coagulopathic Patients

In patients requiring KRT, use heparin-free dialysis or regional citrate anticoagulation (where expertise exists).[7–9] Dialysis catheter insertion should be delayed until partial correction of coagulopathy whenever feasible.[46] If urgent access is required, transfuse FFP/cryoprecipitate after ASV and use ultrasound guidance to minimize complications.[47]

Thrombotic Microangiopathy and Bleeding Overlap

A subset of patients may develop a TMA phenotype (microangiopathic hemolytic anemia, thrombocytopenia, and AKI) following viper envenomation.[48] These patients may have persistent thrombocytopenia and renal dysfunction despite normalization of coagulation tests. Routine plasma exchange is not recommended for snakebite-associated TMA. It should be reserved only for cases with strong suspicion of true thrombotic thrombocytopenic purpura (TTP), supported by clinical features and very low ADAMTS13 activity.[48,49]

Additional Considerations

Heparin is contraindicated in VICC and should not be used for the correction of coagulopathy.[38,39] Antifibrinolytic agents (e.g., tranexamic acid) are not routinely recommended and may be considered only in selected cases with life-threatening bleeding after correction of venom activity. Close monitoring for recurrent coagulopathy is essential, as VICC may recur due to delayed venom absorption from tissue depots.[38–40]

Monitoring

Repeat coagulation profile (PT/INR, fibrinogen, D-dimer, or 20WBCT) every 6 hours until normalization, then daily until stable. Monitor hemoglobin, platelet count, urine output, and signs of occult bleeding.

Rhabdomyolysis and Pigment Nephropathy

Rhabdomyolysis and pigment-induced nephropathy are important contributors to SAKI, particularly following envenomation by sea snakes and certain elapid species, and less commonly with viperid bites.[50–53] Venom-induced myotoxicity leads to widespread muscle necrosis with release of myoglobin, creatine kinase, potassium, and phosphate into the circulation, resulting in direct tubular toxicity, intrarenal cast formation, and renal vasoconstriction. Clinically, patients present with severe generalized myalgia, muscle tenderness, dark-colored urine, and markedly elevated serum creatine kinase levels, often accompanied by hyperkalemia and metabolic acidosis.[52,53] Management is primarily supportive and includes early recognition, careful volume resuscitation with isotonic crystalloids in patients without fluid overload, close monitoring, and prompt treatment of electrolyte abnormalities—especially hyperkalemia—and avoidance of nephrotoxic agents.[52] Urine alkalinization may be considered in selected patients with significant myoglobinuria, though evidence remains limited. Early nephrology involvement and timely initiation of KRT are essential in cases with refractory metabolic complications or progressive renal failure.[52,53]

AKI Management and KRT

All patients with snakebite envenomation should be actively screened for AKI using KDIGO criteria,[17] based on serum creatinine trends and urine output. Early recognition of AKI allows timely implementation of renoprotective strategies aimed at limiting further kidney injury. Core management principles include optimization of hemodynamics with judicious use of balanced crystalloids, avoidance of hypotension and hypoxia, strict monitoring of fluid balance, and elimination of nephrotoxic exposures such as nonsteroidal anti-inflammatory drugs (NSAIDs), aminoglycosides, and iodinated contrast.[54,55] Drug dosages should be adjusted according to estimated glomerular filtration rate (eGFR). Electrolyte disturbances—particularly hyperkalemia, metabolic acidosis, hypocalcemia, and hyperphosphatemia—should be anticipated and corrected promptly.[54,55] Early nephrology consultation is recommended for patients with KDIGO stage 2 or 3 AKI, oliguria/anuria, or rapidly rising creatinine levels.

Kidney Replacement Therapy

Kidney replacement therapy should be initiated based on standard clinical indications rather than serum creatinine alone. Indications include refractory hyperkalemia, severe metabolic acidosis, fluid overload causing pulmonary edema or hypoxemia, uremic complications (encephalopathy, pericarditis), and persistent anuria or oliguria with progressive azotemia.[17,56] The choice of modality should be individualized according to hemodynamic stability, availability of resources, and institutional expertise. Intermittent hemodialysis (IHD) is appropriate for hemodynamically stable patients, while sustained low-efficiency dialysis (SLED) or continuous kidney replacement therapy (CKRT) is preferred in patients with shock or multiorgan dysfunction.[56] In resource-limited settings, acute peritoneal dialysis remains a viable option.[57,58] Given the frequent coexistence of coagulopathy, anticoagulation strategies should be carefully tailored, with preference for heparin-free dialysis or regional citrate anticoagulation where feasible. Early initiation of KRT in appropriately selected patients is associated with improved metabolic control and may reduce complications.[6–8]

Kidney Biopsy

Kidney biopsy is not routinely required in snakebite-associated AKI but may be considered in selected patients with atypical clinical courses or delayed renal recovery. Indications include persistent dialysis dependence or lack of renal recovery beyond 2–3 weeks, unexplained hematuria or heavy proteinuria, suspicion of acute interstitial nephritis (AIN), TMA, or ACN, and diagnostic uncertainty after exclusion of prerenal and obstructive causes.[7–9] Biopsy should be performed only after correction of coagulopathy and stabilization of the patient, using ultrasound guidance to minimize complications.[59] Histopathological findings in SAKI commonly include acute tubular necrosis (ATN), with less frequent findings of AIN, TMA, and CAN.[50,51] Biopsy results may aid prognostication, guide supportive care, and inform long-term follow-up strategies.

Discharge and Follow-up

Snakebite-envenomation-associated AKI survivors remain at significant risk for acute kidney disease (AKD) and subsequent progression to CKD, even after apparent clinical recovery.[6,7,10,18] Therefore, discharge planning should incorporate renal risk stratification, patient education, and a structured follow-up strategy. Patients should be discharged only after hemodynamic stability, resolution or stabilization of coagulopathy, recovery of urine output, and a clear trend toward improvement or stabilization of renal function. Those requiring ongoing dialysis should be referred to a nephrology service with clear documentation of the anticipated recovery trajectory.

Postdischarge follow-up should be protocolized and time-bound. Renal function (serum creatinine, estimated GFR), urine examination (dipstick for hematuria/proteinuria and urine albumin-to-creatinine ratio where feasible), blood pressure, and electrolyte status should be reassessed at 2–4 weeks, 3 months, and 12 months following discharge. Patients with incomplete renal recovery, persistent proteinuria or hematuria, hypertension, or reduced eGFR (<60 mL/min /1.73 m^2) should be managed as high-risk and followed more closely under nephrology care. Recovery from dialysis dependence should be documented, and vascular access should be removed only after sustained renal recovery.

Patient counseling is a critical component of AKD/CKD prevention.[60,61] Patients should be advised regarding adequate hydration, avoidance of nephrotoxic medications (including over-the-counter NSAIDs and herbal remedies), prompt medical evaluation for febrile illness or reduced urine output, and lifestyle measures to control blood pressure and metabolic risk factors.[61] Documentation of SAKI in discharge summaries is essential to alert future healthcare providers. At a systems level, linkage to primary care and nephrology follow-up, especially in rural populations, is vital to reduce long-term renal morbidity and prevent progression to CKD and ESKD.[62]

Implementation and Quality Improvement

Effective reduction in morbidity and mortality from SAKI requires not only evidence-based clinical management but also consistent implementation, audit, and quality improvement across healthcare facilities. Variability in access to ASV, dialysis services, laboratory support, and trained personnel contributes significantly to outcome disparities, particularly in resource-limited settings.[25,34] To promote uniform standards of care, the authors recommend the use of measurable quality indicators for critical care centers managing SAKI.

Table 4: Quality indicators for critical care centers managing SAKI

Indicator	*Numerator/denominator*	*Target*
Time from arrival to first ASV infusion	Patients receiving ASV within 60 minutes / all envenomed	≥ 80%
Appropriate ASV re-dosing by lab results/ bleeding	Cases with documented q6h coagulation profile guiding re-dose/ hemotoxic bites	≥ 90%
Early nephrology involvement	SAKI with KDIGO stage ≥2 seen by nephrology within 12 hours/ all stage ≥2	≥ 90%
KRT timeliness	SAKI meeting absolute indications dialyzed within 4 hours/ all indicated	≥ 95%
Avoidance of nephrotoxins	SAKI admissions without NSAIDs/ aminoglycosides/ contrast unless justified	≥ 95%
Postdischarge kidney follow-up	SAKI survivors with 3-month kidney review/ SAKI survivors	≥ 80%

ASV, antisnake venom; NSAIDs, nonsteroidal anti-inflammatory drugs

Table 5: SAKI toolkit for snakebite-associated AKI

Domain	*Key recommendations*	*Clinical tools*
Recognition	Maintain a high index of suspicion in all snakebite cases with oliguria, hematuria, edema, or systemic envenomation	Bedside urine output chart; dipstick for hematuria/proteinuria; point-of-care creatinine
Risk stratification	Identify risk factors for AKI and poor outcomes	Hemotoxic envenomation (Russell's viper, Echis); delayed ASV (>6 hours); hypotension/shock; coagulopathy/disseminated intravascular coagulation; rhabdomyolysis
Supportive therapy	Initiate early measures to prevent the progression of AKI	Prompt ASV as per national protocol; adequate hydration guided by central venous pressure/urine output; avoid nephrotoxins (NSAIDs, aminoglycosides, contrast); early management of hyperkalemia and acidosis
Kidney Replacement Therapy (KRT)	Early initiation improves survival; individualize based on resources	Indications: refractory hyperkalemia, severe metabolic acidosis, pulmonary edema, and uremic symptoms Preferred modality: IHD; CKRT for hemodynamically unstable patients, where available Peritoneal dialysis in resource-limited settings
Adjunctive measures	Provide comprehensive care beyond kidney support	Manage complications: bleeding, sepsis, and compartment syndrome; correct coagulopathy with FFP/cryoprecipitate; monitor for thrombotic microangiopathy and secondary sepsis
Follow-up and long-term care	Ensure renal recovery and monitor for CKD	Weekly creatinine for 1 month, then monthly up to 6 months; counsel patients on hydration and avoiding nephrotoxins; screen for CKD progression (eGFR, urine ACR)

ACR, albumin–creatinine ratio; AKI, acute kidney injury; ASV, antisnake venom; CKD, chronic kidney disease; CKRT, continuous kidney replacement therapy; DIC, disseminated intravascular coagulation; eGFR, estimated glomerular filtration rate; FFP, fresh frozen plasma; IHD, intermittent hemodialysis; NSAIDs, nonsteroidal anti-inflammatory drugs; SLED, sustained low-efficiency dialysis

These indicators are designed to monitor the timeliness of antivenom administration, appropriateness of renal support, avoidance of preventable nephrotoxicity, and continuity of postdischarge kidney care. Regular audit against these benchmarks can identify gaps in care, facilitate targeted interventions, and support institutional accreditation and capacity-building initiatives (Table 4).

Summary of Recommendations

SAKI remains a significant cause of morbidity and mortality in India, especially in rural and resource-limited settings.[6–9] Early recognition, risk stratification, and standardized supportive care are critical to improving outcomes. These recommendations propose a structured "SAKI Toolkit" that provides a stepwise framework covering recognition, risk assessment, supportive therapy, KRT initiation, and follow-up (Table 5). This toolkit serves as a practical bedside reference for general physicians, nephrologists, intensivists, and emergency physicians.

Conclusion

Snakebite envenomation remains a significant yet preventable cause of AKI in India, particularly in rural and resource-limited settings. These recommendations emphasize a structured, evidence-informed approach—encompassing rapid assessment, timely administration of antisnake venom, meticulous supportive care, early recognition of AKI, and appropriate use of KRT. The SAKI Toolkit aims to empower clinicians to streamline decision-making, minimize complications, and improve patient survival. Implementation of these recommendations requires not only clinical vigilance but also strengthening of referral networks, training of healthcare personnel, and public health measures for prevention and early presentation. With multidisciplinary collaboration and adherence to standardized protocols, morbidity and mortality from snakebite-associated AKI can be significantly reduced.

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