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ORIGINAL RESEARCH INTENSIVE CARE

Critical care management of snakebite envenomation: An overlooked frontier in the intensive care

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ABSTRACT

Background & objectives: Snakebite envenomation remains a significant cause of morbidity and mortality in tropical and subtropical regions. The existing literature on the critical care management of severe envenomation cases is insufficient, despite the considerable interest in public health strategies and antivenom utilization.

This narrative review aims to provide a comprehensive overview of the pathophysiological characteristics of severe snakebite envenomation requiring ICU admission and to emphasize current evidence-based strategies for critical care management.

Methodology: A narrative review was conducted using literature sourced from PubMed, Scopus, and Web of Science. Search terms included “snakebite,” “envenomation,” “intensive care,” “critical care,” “neurotoxicity,” “coagulopathy,” “renal failure,” and “organ support.” Inclusion criteria comprised clinical trials, observational studies, and expert guidelines focused on ICU-level management of snakebite envenomation. Exclusion criteria included non-English articles, studies unrelated to critical care, and those lacking original clinical data. An informal critical appraisal prioritized high-quality studies based on methodological clarity, sample size, and relevance to both high- and low-resource settings.

Results: Literature indicates that severe envenomation can result in coagulopathy, acute kidney injury, shock, secondary infections, rapid onset of neurotoxicity, respiratory paralysis, and other conditions, all necessitating ICU-level treatment. Timely antivenom delivery is essential; however, it must be complemented by continuous hemodynamic monitoring, mechanical ventilation, vasopressor therapy, renal replacement therapy, and infection control strategies.

Conclusions: Snakebite envenomation poses complex challenges that extend beyond the availability of antivenom. Severe cases necessitate critical care intervention to avert permanent organ damage and reduce mortality. Enhancing outcomes for snakebite victims necessitates a proper integration of the ICU protocols, physician education, and the development of infrastructure, particularly in endemic regions. Future studies should aim to evaluate standardized ICU management pathways, investigate long-term outcomes in envenomated patients, and address regional disparities in critical care capacity to inform more equitable and effective care delivery.

Abbreviations: AKI: acute kidney injury, ARDS: acute respiratory distress syndrome, CK: creatine kinase, DIC: disseminated intravascular coagulation, ICU: intensive care unit, MFO: Multiorgan failure, SBE: Snakebite envenomation, SIRS: systemic inflammatory response syndrome, TSS: Toxic snakebite syndrome, VICC: venom-induced consumption coagulopathy

Keywords: Snakebite Envenomation; Intensive Care; Critical Care Management; Neurotoxicity; Coagulopathy; Organ Support

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1. INTRODUCTION

Snakebite envenomation (SBE) remains a significant public health concern, especially in tropical and subtropical regions. The World Health Organization (WHO) estimates that there are approximately 5.4 million snakebites each year, resulting in 1.8–2.7 million envenomation and 81,000–138,000 fatalities worldwide.[1] Rural and resource-limited settings experience significant challenges due to restricted access to timely medical treatment and antivenom. SBE remains a major cause of morbidity and mortality, highlighting the need for comprehensive management strategies, as classified by the WHO (2023) as a neglected tropical disease.[2]

Severe envenomation may lead to toxic snakebite syndrome (TSS) can lead to a range of life-threatening outcomes, such as neurotoxicity, coagulopathy, acute kidney injury (AKI), and respiratory failure, necessitating admission to an intensive care unit (ICU). Viperid envenomation typically results in coagulopathy and renal impairment;[3] however, neurotoxic effects, such as paralysis and respiratory compromise, are particularly associated with rapid bites. The complexity of these clinical presentations necessitates a multidisciplinary treatment approach that integrates contemporary supportive care strategies with antivenom distribution.[4]

Severe snakebite patients are primarily managed in intensive care; however, there is a lack of research regarding ICU interventions and outcomes. Current research predominantly emphasizes epidemiology, first aid, and antivenom medication, often overlooking the nuances of critical care management.[5] The existing information gap hinders the development of standardized methods and may lead to suboptimal outcomes for critically ill patients.[3]

While first-line therapy and prompt antivenom administration have received considerable attention in the literature, the role of intensive care in managing severe envenomation remains underexplored. A significant proportion of snakebite victims, particularly those exhibiting neurotoxic paralysis, coagulopathy, AKI, or multi-organ dysfunction, require intensive life support and prolonged ICU admission. Research conducted in endemic regions indicates that 20–40% of hospitalized envenomation cases require ICU-level treatment, often in settings deficient in critical care resources.[1,3] Limited comprehensive research has examined snakebite management from an ICU perspective, resulting in a knowledge gap that affects clinical decision-making, training, and resource allocation. This review aims to summarize current knowledge on the critical care management of SBE, focusing on pathophysiological mechanisms, clinical consequences, and ICU-specific therapeutic techniques. This review seeks to delineate optimal practices and highlight avenues for future research within often overlooked domain of critical care medicine, integrating findings from previous studies with contemporary clinical experience.

2. Types of TSS

SBE manifests various clinical symptoms, contingent upon the venom composition, which varies by species and geographical location.[6] The three primary toxicological profiles—neurotoxic, vasculotoxic (hemotoxic), and myotoxic envenomations—are

characterized by distinct pathophysiological mechanisms and clinical effects.[9] A thorough understanding of these conditions is essential for accurate diagnosis, timely antivenom administration, and the selection of relevant critical care interventions.[7]

Neurotoxic envenomation is primarily associated with elapid snakes, including certain Australian elapids, cobras (Naja spp.), and kraits (Bungarus spp.). Neurotoxins present in these venoms, affecting both presynaptic and postsynaptic sites, diminish neuromuscular transmission, leading to progressive paralysis.[8] Patients may present with ptosis, ophthalmoplegia, bulbar weakness, and ultimately diaphragmatic paralysis, which may require mechanical ventilation.[9,10] In certain cases, especially with krait bites, the initiation may be postponed due to the necessity for prolonged observation and preparedness for respiratory assistance.[11] Early respiratory monitoring, ventilatory support, and repeated neurological assessments are critical components of ICU treatment that inform the administration of antivenom.[5]

Vasculotoxic (hemotoxic) envenomation is a characteristic feature of viperid species such as Daboia russelii (Russell's viper), Bothrops spp., and Echis spp.[12] Procoagulants, hemorrhagins, and metalloproteinases present in these venoms cause significant endothelial damage, coagulopathy, and bleeding. Patients may present with thrombocytopenia, disseminated intravascular coagulation (DIC), hypovolemic shock, and spontaneous bleeding.[13] In addition to antivenom, management includes intensive fluid resuscitation, blood product transfusion, and correction of coagulopathy. In critical situations, the ICU may require organ support, including renal replacement therapy and vasopressors.[14]

Myotoxic envenomation, while less recognized, holds clinical significance and is typically associated with various Australian elapids, certain sea snakes, and some vipers.[15]

The venoms primarily consist of phospholipases and cytotoxins, which include myotoxins that induce rhabdomyolysis, muscle necrosis, and subsequent myoglobinuria, potentially resulting in AKI.

Patients may exhibit dark urine, muscle soreness, weakness, and elevated creatine kinase levels.[16] Aggressive intravenous hydration and electrolyte correction, particularly for hyperkalemia, along with the initiation of early renal support upon the onset of AKI, characterize ICU management. The prevention of permanent renal impairment and associated systemic consequences relies on the prompt identification of this syndrome.[17]

3. General Line of Treatment for TSS

The management of TSS, in accordance with advanced emergency care guidelines, begins with a rapid examination and stabilization of the patient. The primary concerns include airway maintenance, respiratory support, circulatory assistance, and management of potentially life-threatening complications.[18] Essential provides a comprehensive account detailing the time, location, and conditions surrounding the bite. While treatment should not be postponed in the absence of snake identification, recognizing the species, if feasible, can assist in determining the appropriate antivenom selection.[17] Minimizing patient movement and immobilizing the affected limb help to prevent systemic venom distribution. Essential components for guiding continuous therapy include early intravenous access, fluid resuscitation, and baseline laboratory studies, which encompass coagulation profile, renal function, electrolytes, and complete blood count.

The primary treatment for systemic envenomation is the prompt administration of antivenom, which neutralizes circulating venom and mitigates adverse effects.[19]

Clinical symptoms such as neurotoxicity, coagulopathy, shock, or rapidly progressive local swelling inform the decision to administer antivenom.[1] Polyvalent antivenoms frequently utilized in regions with multiple venomous species include Dosage is determined by severity; patient weight is not a factor in dosage determination; in severe cases, repeated treatment may be necessary.[20] Premedication with antihistamines or corticosteroids remains a subject of debate; however, individuals with a history of allergies may consider this option. Clinicians must remain vigilant for both early and delayed hypersensitivity reactions to antivenom, such as anaphylaxis and serum sickness, and should be prepared to implement emergency resuscitation techniques.[21]

Supportive care significantly improves the efficacy of antivenom treatment. In cases of neuroparalysis, mechanical ventilation is indicated; in instances of coagulopathic hemorrhage, transfusion of blood products is necessary; in acute renal injury, dialysis is required; and in the presence of local necrosis or infection, appropriate wound care must be administered.[22] Broad-spectrum antibiotics may be indicated if a subsequent infection is suspected, along with tetanus prophylactic agents and analgesics.[23] The absence of stringent monitoring capabilities in resource-constrained settings may necessitate premature referral to tertiary care. Long-term effects such as limb impairment, psychological trauma, and chronic kidney disease necessitate multidisciplinary follow-up. A

systematic, protocol-driven approach that integrates organ support with antivenom treatment improves survival and functional outcomes in snakebite victims.[24]

4. Clinical Indications for ICU Admission in SBE

Admission to the ICU is warranted when SBE presents with symptoms indicative of life-threatening systemic involvement.[4,5] In cases of envenomation by elapids such as cobras or kraits, a significant indicator is the escalation of neurotoxicity. Clinically, the presence of ptosis, bulbar dysfunction, respiratory muscle paralysis, and decreased oxygen saturation necessitates prompt airway assessment and the initiation of mechanical ventilation.[25] Delayed respiratory failure underscores the necessity for ongoing monitoring and the availability of respiratory support, as it may arise even following initial improvement. Early admission to the ICU facilitates rigorous neurological monitoring, prompt intervention for respiratory decompensation, and precise titration of ventilatory support. Bleeding events and coagulopathy are significant indicators for critical care.[26]

Venom-induced consumption coagulopathy from Viperid envenomation is characterized by prolonged clotting times, spontaneous bleeding, thrombocytopenia, and hypovolemic shock. Patients exhibiting overt bleeding, such as gastrointestinal, intracranial, or hematuria, along with significant hypotension or laboratory-confirmed disseminated intravascular coagulation (DIC), require ICU-level management.[27] Intensive care facilitates the administration of repeat antivenom, vigorous resuscitation with blood products, and continuous hemodynamic monitoring when required. Invasive monitoring may be necessary in cases of refractory shock or significant hemorrhage; acute kidney impairment may necessitate renal replacement therapy.[28]

A third significant clue is the involvement of multiple systems, specifically acute kidney damage resulting from rhabdomyolysis-induced antivenom, allergic reactions to antivenom, or subsequent infections such as sepsis.[19] Myotoxic envenomation is associated with a high likelihood of renal failure and cardiac arrhythmias, characterized by significantly elevated creatine kinase levels, myoglobinuria, and hyperkalemia[15]. Candidates for ICU treatment may encompass patients exhibiting altered mental status, systemic inflammatory responses, or persistent metabolic abnormalities.[16] The ICU provides the necessary infrastructure for advanced organ management, continuous monitoring, and prompt escalation of therapy in instances of erratic venom effects. Timely referral to the ICU improves outcomes by enabling proactive management of issues prior to the onset of irreversible organ damage.[15-17]

5. ICU Management of Progressive Neurotoxicity in SBE

Progressive neurotoxicity resulting from bites by kraits (Bungarus spp.) and cobras (Naja spp.) constitutes a significant clinical challenge necessitating admission to an ICU.[29] Primarily targeting the neuromuscular junction—either presynaptically (e.g., β-bungarotoxin) or postsynaptically (e.g., α-bungarotoxin)—these neurotoxins lead to the inhibition of acetylcholine release or receptor blockade. Neurotoxicity typically presents clinically as ptosis, ophthalmoplegia, dysarthria, and bulbar weakness, potentially progressing rapidly to flaccid paralysis and respiratory failure.[30] Timely ICU admission is crucial for effective neurological monitoring and prompt initiation of ventilatory support; notably, diaphragmatic involvement may be subtle and often overlooked in the early stages.[31]

Airway protection and mechanical ventilation are paramount in the ICU setting. Detection of impending respiratory compromise relies on continuous monitoring of oxygen saturation, respiratory rate, and arterial blood gases.[31] Patients with bulbar involvement or deteriorating respiratory mechanics should undergo intubation without delay.[32] Irreversible disruption of acetylcholine release can lead to paralysis in patients experiencing presynaptic neurotoxic effects, such as those from krait bites, lasting for several days. Consequently, even with the administration of antivenom, extended mechanical ventilation remains essential.[33] Weaning from ventilatory assistance requires a meticulous approach involving daily neurological assessments and spontaneous breathing trials. Optimizing sedation techniques can prevent oversedation and enhance neurologic recovery.[34]

The timely and suitable administration of specific antivenom constitutes an adjunctive treatment in the ICU setting; it remains a primary therapeutic approach, although its efficacy may be diminished following irreversible neurotoxic binding to presynaptic targets1. Antivenom administration, especially within the initial hours, may enhance efficacy in instances of postsynaptic blockade, such as cobra envenomations.[20,21] Despite their limited function, which should be guided by species-specific pathophysiology, acetylcholinesterase inhibitors (e.g., neostigmine) are increasingly gaining attention for postsynaptic toxicity.[35] Thromboembolic prophylactic therapy, eye care to prevent corneal ulcers, and the prevention of ventilator-associated pneumonia are integral components of supportive treatment. ICU teams must remain vigilant regarding the consequences

of prolonged ICU stays, which include psychological distress in survivors and critical illness polyneuropathy.[36]

6. ICU Care of TSS with Coagulopathy and Hemorrhage

The envenomation characteristics of viperid snakes such as Daboia russelii, Bothrops spp., and Echis spp. include coagulopathy and hemorrhagic outcomes, which arise from complex venom-induced consumption coagulopathy (VICC). Venom components, such as metalloproteinases and hemorrhagins, disrupt hemostasis by activating or destroying clotting factors, damaging vascular endothelium, and causing platelet dysfunction.[37] These effects may lead to spontaneous bleeding, which can vary from mucocutaneous hemorrhages to severe cerebral or gastrointestinal bleeding, necessitating vigilant monitoring and prompt ICU intervention.[27,28]

Timely diagnosis and management of bleeding diathesis is paramount in ICU settings. Constant hemodynamic monitoring is essential due to the risk of hypovolemic shock resulting from acute blood loss. Fluid resuscitation with crystalloids and blood product transfusion, including packed red blood cells, fresh frozen plasma, platelets, and cryoprecipitate, is often essential for maintaining adequate circulation volume and addressing coagulopathy.[38] Timely delivery and the extent of envenomation are critical factors influencing the efficacy of antivenom, which is the sole definitive treatment capable of neutralizing circulating venom toxins and preventing the progression of coagulopathy. Platelet counts, fibrinogen levels, and serial coagulation profiles inform the determination of antivenom dosage and transfusion therapy.[27,28]

In cases of refractory shock, advanced supportive therapy in the ICU may include mechanical ventilation for respiratory failure due to pulmonary bleeding or aspiration, vasopressor support, and invasive hemodynamic monitoring.[28] AKI due to hemoglobinuria from intravascular hemolysis may necessitate renal replacement therapy.[39] The ICU staff must remain vigilant for complications such as multiorgan failure, secondary infections, and disseminated intravascular coagulation. Collaboration among hematology, nephrology, and critical care specialists enhances patient outcomes.[16,19] Timely ICU referral and adherence to established care protocols significantly reduce morbidity and mortality in patients with coagulopathic snakebites.

7. ICU Care for TSS Presenting with MOF

Multiorgan failure (MOF) represents a serious and potentially fatal consequence of TSS, characterized by significant systemic toxicity and profound physiological disturbances.[40] Multiorgan failure (MOF) typically arises from a combination of venom-induced endothelial damage, coagulopathy, rhabdomyolysis, acute AK), shock, and systemic inflammatory response syndrome (SIRS).[41] Enhancing survival outcomes in critically ill patients relies on prompt identification and intensive supportive care within the ICU.[17]

Targeted organ support tailored to the specific systems involved is fundamental to ICU management in multi-organ failure resulting from snakebite. Respiratory failure resulting from either acute respiratory distress syndrome (ARDS) or neurotoxicity necessitates invasive mechanical ventilation employing lung-protective strategies.[42] Both distributive and hypovolemic shock necessitate rigorous fluid resuscitation, vasopressor therapy, and continuous invasive monitoring (such as arterial lines and central venous pressure) to inform the resuscitation process.[43] Patients experiencing AKI due to fluid overload, electrolyte imbalances, or significant acidosis often necessitate renal replacement therapy (RRT), which may include continuous modalities.[39]

Concurrent treatment involves careful laboratory monitoring, transfusion of blood products, and prompt administration of antivenom to address coagulopathy. Metabolic abnormalities such as hyperkalemia and acidosis resulting from rhabdomyolysis are addressed promptly.[43] The ICU staff must anticipate secondary complications such as sepsis, multiorgan failure, and the consequences of prolonged immobility. Enhancing patient outcomes requires collaborative efforts among intensivists, nephrologists, hematologists, and infectious disease specialists. The prognosis of multiple organ failure following snakebite is cautious, even with optimal supportive care, highlighting the importance of early intervention and prevention through timely administration of critical care and antivenom.[36]

8. Antivenom Use in ICU and Associated Adverse Events

Antivenom therapy remains the exclusive treatment for systemic SBE and is a vital intervention in an ICU setting. Treatment is indicated in cases of neurotoxicity, coagulopathy, cardiovascular instability, or severe local envenomation. Antivenom administration should occur promptly and judiciously, preferably within the initial hours of envenomation, to optimize effectiveness in severely ill patients.[44] Intravenous treatment is favored in the ICU, with dosing determined primarily by the severity of clinical symptoms rather than patient weight. While polyvalent antivenoms are prevalent in regions

with multiple venomous species, monovalent formulations may be preferable when the specific offending species is identified, as they can reduce antigenic load and associated side effects.[45]

During the initial 30 to 60 minutes of infusion, antivenom administration in the ICU necessitates careful preparation, thorough pre-infusion assessment, and attentive monitoring. Some studies suggest limited efficacy in preventing adverse reactions, while others support their use in high-risk patients or situations with suboptimal antivenom purity.[44] The premedication with antihistamines (e.g., chlorpheniramine) and corticosteroids (e.g., hydrocortisone) remains a topic of debate. In the absence of immediate hypersensitivity, the infusion should commence at a slow rate and subsequently escalate gradually. Continuous monitoring of ECG, pulse oximetry, and blood pressure is recommended. The ICU environment facilitates rapid responses to issues related to infusion and allows for treatment adjustments based on clinical and laboratory outcomes.[46]

While antivenom treatment is life-saving, it is associated with side effects primarily concerning early hypersensitivity reactions and delayed serum sickness. Immediate reactions, such as urticaria, bronchospasm, hypotension, and anaphylaxis, occur in 5–25% of cases, depending on the source, protein load, and production technique.[46] These reactions necessitate the initiation of intramuscular epinephrine, intravenous fluids, and antihistamines, along with the prompt cessation of the infusion. The availability of critical care resources facilitates rapid airway control and, if necessary, the administration of vasopressors. Delayed reactions, typically manifesting 5–14 days post-infusion, include serum sickness characterized by fever, rash, arthralgia, and lymphadenopathy. Recording is essential for future treatment; typically, corticosteroids and antihistamines are administered, which do not contraindicate subsequent antivenom use. ICU physicians must remain vigilant regarding these side effects and ensure that prompt management protocols adhere to established standards.[47]

9. ICU Prognostic Markers and Scoring Systems in TSS

Prognostic prediction is essential for risk stratification, resource allocation, and guiding care decisions in ICU patients with TSS. Several laboratory and clinical criteria have been identified as prognostic markers, reflecting the extent of systematic toxicity and potential consequences. Additionally, grading methods adapted from general critical care have been employed to predict outcomes and mortality risk in envenomed patients.

Elevated serum creatinine levels, indicative of AKI (AKI), serve as a significant laboratory prognostic marker and are a robust independent predictor of adverse outcomes.[48] Severe thrombocytopenia, prolonged prothrombin time (PT), hypofibrinogenemia, and elevated D-dimer indicate venom-induced consumption coagulopathy (VICC), which heightens the risk of mortality and hemorrhage.[49] Rhabdomyolysis and impending renal failure are associated with creatine kinase (CK) levels exceeding 5,000 U/L. The increase in lactate, presence of hyperkalemia, and occurrence of metabolic acidosis indicate potential systemic toxicity or tissue hypoperfusion. Monitoring these markers serially is essential for tracking disease progression and guiding treatment decisions.[50]

Clinical prognostic factors include altered mental state at admission, persistent hypotension despite fluid resuscitation, requirement for mechanical ventilation, and multiorgan failure.[48] Increased mortality is associated with the need for renal replacement therapy, persistent coagulopathy despite antivenom administration, and delayed presentation, typically occurring more than six hours after the bite.[51] The extent of cranial nerve involvement and respiratory muscle weakness at admission in neurotoxic envenomation can serve as predictors for prolonged ventilatory dependency and duration of ICU hospitalization.[52]

Scoring systems such as the Sequential Organ Failure Assessment (SOFA) and Acute Physiology and Chronic Health Evaluation II (APACHE II) are frequently utilized for snakebite patients in the ICU and have been broadly validated in critical care settings. Studies indicate that higher SOFA scores at 24 hours post-admission, particularly those exceeding 9, are associated with increased mortality.[53] The modified Snakebite Severity Score (SSS), which incorporates criteria such as neurotoxicity, coagulopathy, renal failure, and local tissue necrosis, has demonstrated potential in patient triage and predicting ICU requirements, despite its current lack of uniformity globally. Dynamic evaluation, through these ratings, facilitates the early identification of high-risk patients, thereby allowing for targeted interventions and family counseling.[54]

10. ICU Challenges in Low-Resource Settings

TSS represents a significant public health challenge in low-income countries, particularly in rural regions of sub-Saharan Africa, South Asia, and Southeast Asia.[55] Systemic constraints frequently hinder the management of snakebite cases in ICU, thereby elevating morbidity and mortality rates. Effective treatment remains significantly hindered by insufficient access to timely

and adequate critical care, despite the relatively common occurrence of severe envenomation.[56]

The absence of adequate facilities and qualified personnel constitutes a primary challenge. Numerous district level or peripheral hospitals are devoid of fully operational ICUs equipped with mechanical ventilators, dialysis facilities, and reliable supplies of oxygen and electricity.[57] Neurotoxic envenomation poses significant risks, as prompt airway protection and ventilatory support are critical for survival. Additionally, personnel in low-resource ICUs may possess insufficient knowledge regarding the management of snakebite sequelae, such as autonomic instability, acute renal injury, or venom-induced coagulopathy.[58] The lack of clinical protocols and monitoring tools, such as invasive blood pressure measurements, arterial blood gases, and real-time coagulation assays, further complicates the ability to deliver safe and effective intensive care.[55,56]

Antivenom supplies and their administration remain inconsistent and problematic. Numerous nations exhibit inadequate control over their supply chains, resulting in stockouts or the availability of substandard or unsuitable antivenom products. Additionally, storage conditions that necessitate refrigeration and the requirement for safe reconstitution and delivery within a controlled environment are often not met.[59] The presence of antivenom does not alleviate concerns regarding potential side effects, particularly anaphylaxis, in resource-limited ICUs lacking resuscitation equipment, which hinders timely administration. Patients frequently incur out-of-pocket expenses for antivenom and ICU treatment, indicating that financial constraints are a significant factor.[60]

A significant issue is the delayed arrival to medical facilities. Patients often present at tertiary centers several hours post-envenomation, by which time complications such as respiratory failure, renal shutdown, or disseminated intravascular coagulation may have already developed. This delay is attributed to insufficient transportation networks, reliance on traditional healers, or a general lack of awareness.[55] The therapeutic window of effective antivenom is constrained by this factor, as is organ support. Targeted care is impeded in relation to ICU due to insufficient pre-ICU stabilization, inadequate documentation of bite incidents, and a lack of laboratory resources for venom identification. Addressing these issues necessitates systematic funding for rural critical care infrastructure, modifications in the supply chain, staff training, and community education.[58]

11. Geographic Disparities in ICU Infrastructure and Staffing

Geographic Variations in ICU Infrastructure and Staffing Outcomes of SBE vary by region, influenced by the availability of critical care infrastructure, staffing levels, and access to life-saving treatments.[56] High-resource ICUs equipped with advanced monitoring, mechanical ventilation, dialysis, and skilled intensivists enhance survival rates and diminish complications. In Australia and the United States, where envenomation is infrequent yet handled with significant clinical precision, the early detection of systemic toxicity, prompt administration of antivenom, and adherence to standardized ICU protocols enhance outcomes, even in severe instances.[3] The infrastructure of ICUs is inadequate in numerous snakebite-endemic areas of sub-Saharan Africa, South Asia, and Latin America. Insufficient availability of ventilators, infusion pumps, and renal replacement therapy may impede or limit the treatment of neurotoxic paralysis, coagulopathy, and AKI.[55-58] Healthcare professionals in rural or district hospitals often lack training in critical care and toxicology, resulting in delays in patient recognition and treatment.[5,17] Studies conducted in India revealed that delays in mechanical ventilation for neurotoxic envenomation were associated with increased mortality, particularly in facilities lacking 24/7 ICU staff.[19,20] The scarcity of ICU beds in sub-Saharan Africa has necessitated the provision of improvised care in standard wards, where close monitoring is unfeasible, resulting in preventable complications that lead to excessive patient mortality.[55]

The inequalities highlight the necessity of strengthening high-burden ICUs. Prioritizing the expansion of infrastructure and enhancement of worker capacity through critical care training, protocol development, and telemedicine support is essential. To mitigate SBE globally and ensure equitable access to life-saving treatment, it is essential to address the resource gap.[60]

12. Future Recommendations

In endemic and resource-limited regions, a multifaceted approach is essential to improve outcomes for critically ill patients suffering from lethal SBE. Enhancing critical care infrastructure is essential; peripheral hospitals must be equipped with necessary ICU resources, including mechanical ventilators, dialysis units, and laboratory diagnostics, to manage coagulopathy, AKI, and respiratory failure. Ensuring consistency and quality in treatment necessitates the development and implementation of standardized clinical protocols for assessment, antivenom administration, and organ support. These protocols should be prioritized and incorporated into public health recommendations. The enhancement of antivenom availability and quality is equally significant. This necessitates a well-regulated supply chain that ensures equitable, subsidized access,

cold-chain maintenance, and effective goods in high-risk areas. Simultaneously, training for healthcare professionals is essential for capacity-building. Prioritizing early detection of severe envenomation, prompt management of antivenom-related adverse events, and appropriate ICU triage is essential to reduce avoidable complications and mortality.

Moreover, early triage and the strategic allocation of intensive care resources through the development and validation of region-specific prognostic scoring systems may improve clinical decision-making. Continuous funding for research and surveillance is essential. This encompasses enhancements in venom proteomics, refinement of antivenom pharmacodynamics, and comprehensive audits of clinical outcomes. Data-driven projects will inform national and international policy frameworks for snakebite control and promote evidence-based enhancements in therapeutic strategies. Addressing the complex issues of SBE in the ICU requires a comprehensive approach that integrates clinical treatment, public health initiatives, and the enhancement of healthcare systems. Outcomes for individuals who have experienced critical envenomation can significantly improve with a coordinated global response and sustained local investment.

13. CONCLUSION / KEY FINDINGS

Particularly in low-income countries, SBE is still a major medical emergency with great morbidity and death. Severe instances presenting with neurotoxicity, coagulopathy, hemorrhagic events, and multiorgan failure need for intensive care therapy. Early antivenom delivery, careful monitoring, and organ support techniques—including mechanical breathing, renal replacement therapy, and hemodynamic stabilization—are underlined in the review as especially important. Envenomations classified as neurotoxic and vasculotoxic necessitate particular ICU treatment catered to the pathophysiological processes of the particular venom. Early risk classification and outcome prediction are supported by prognostic markers like serum creatinine, coagulation profiles, and scoring systems including SOFA and APACHE II. But in resource-constrained environments, infrastructure problems, delayed presentation, and uneven access to potent antivenom seriously impede the delivery of ICU-level treatment.

14. Conflict of interest

All authors declare that there was no conflict of interest.

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16. Authors' contribution

EME: Conceptualization, Supervision, Writing – Original Draft, Methodology

AS: Validation, Writing, Review & Editing, Supervision

WAC: Investigation, Data Curation, Writing – Original Draft

AA: Formal Analysis, Visualization

SSHS: Resources, Project Administration

SAHA: Investigation, Writing – Review & Editing

FFBA: Writing – Original Draft, Literature Review

ASAA: Data Curation, Visualization

SFSA: Editing, Proofreading, Project Coordination

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