

Extracorporeal Hemoperfusion as an Adjunctive Therapy in Neurotoxic Snakebite Envenomation: A Case Report

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ABSTRACT

Snakebite envenomation is a medical emergency with potentially fatal systemic complications. We report a case of a 32-year-old male patient who presented to the Emergency Department with a complaint of a snakebite on his right ear. On arrival, he didn't have signs of bleeding, fever, or swelling. Two hours after admission, the patient developed cardiac arrest and required mechanical ventilation. Although resuscitation was successful, the patient's condition deteriorated, with progressive neurological and motor deficits, despite unremarkable findings on head CT scans and chest X-rays. Given progressive neurological and motoric deficits, hemoperfusion was initiated six hours post-envenomation as an adjunctive therapy. The procedure lasted six hours using a standard hemoperfusion cartridge. The patient demonstrated significant neurological recovery within 22 hours post-procedure, and he was successfully extubated on day 4. This case highlights the potential role of hemoperfusion as an adjuvant treatment in managing venomous snakebite envenomation, especially in places with limited species-specific antivenom availability. Early recognition of systemic complications and timely initiation of hemoperfusion may improve neurological outcomes in critically envenomed patients.

Introduction

Snakebite envenomation (SBE) is a significant public health issue, particularly in rural areas of tropical and subtropical countries, and mostly in low and middle income countries especially in Asia, Africa, and Central, and South America [1]. Globally, SBE affects an estimated around of 1.8 to 2.7 million people annually, with mortality ranging from 80,000 to 138,000 deaths. The Southeast Asia region accounts for 70% of global snakebite-related deaths [2]. In Indonesia, data from 2017 reported 12,739 to 214,883 cases of

snakebite with 11,581 people deaths because SBE [3]. The higher number of cases is related to various varieties of venomous snakes, especially from the Elapidae (57 species) and Viperidae (22 species) families. Both families are of major medical importance due to their widespread distribution and their significant contribution to morbidity and mortality rates [4].

Serum Anti Bisa Ular (SABU) often known as antivenom, is the main treatment for SBE. However, because of its high cost, limited availability and possibility for negative reactions, its usage is only advised if the benefits outweigh the dangers [1,5]. These conditions have prompted the development of alternative therapies, including extracorporeal therapies. That

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method allows for elimination of specific toxins from the bloodstream and has shown promising outcomes. Hemoperfusion is one of the techniques in extracorporeal therapies. Hemoperfusion is a process in which blood is passed through a specialized cartridge capable of adsorbing toxins [6]. In this report, we present a case of a snakebite patient who was successfully managed with hemoperfusion therapy.

Case Report

The patient was a 32-year-old man arrived at our Emergency Department with a complaint of a snakebite on his right ear while working in a rice field about an hour earlier. The patient was unable to identify the snake species but can describe morphology as brown with a pattern. He complained of pain, but there was no bleeding, fever, or tightness. After two hours, the patient experienced nausea, vomiting, and loss of consciousness and subsequently experienced cardiac arrest. Return of spontaneous circulation (ROSC) was achieved following successful resuscitation. Physical examinations show the patients' level of consciousness was sopor. The patient had no history of hypertension or diabetes mellitus. Kidney function examination showed elevated urea at 82 mg/dL and creatinine at 1 mg/dL, indicating renal impairment suggestive of nephrotoxicity. Hemostasis evaluation revealed a prothrombin time (PT) of 16.2 sec, an activated partial thromboplastin time (APTT) of 25.2 sec, an international normalized ratio (INR) of 1.29, and a D-dimer level of 1950 ng/mL FEU. That result supports the diagnosis of coagulopathy due to hematotoxins. Based on the clinical and laboratory examination, the patient was assessed as having grade IV envenomation with diagnosis of neurotoxicity, myotoxicity, cardiotoxicity, nephrotoxicity, and shock.

On the first day, to manage cardiac arrest, specifically when the pulse cannot be felt, cardiopulmonary resuscitation (CPR) is used, along with 0.1 mg epinephrine and 0.3 mg norepinephrine. Immediate intubation was carried out once vital signs stabilized (BP 120/80 mmHg, pulse 115 bpm, and oxygen saturation 98-99%), and the patient was placed on mechanical ventilation. The patient was transferred to the Intensive Care Unit (ICU), where the following treatment regimen was initiated. 10 ampoules of polyvalent antivenom (SABU) diluted in 500 mL of 0.9% NaCl, administered over 1 hour and repeated every 6 hours, KAEN 1 B 1000 cc/24 hours, Cefotaxime 2x1 gram IV, Metronidazole 3x500 mg IV, Omeprazole 1x40 mg IV, Paracetamol 3x1 gram IV, Dexamethasone 3x5 mg IV, Anti-tetanus serum (ATS) 2500 IU, and Tetanus toxoid (TT) 0.5 mL. For sedation, the patient received a continuous infusion of ketamine (30 mg/hour), propofol (20 mg/hour), and fentanyl (20 mcg/hour).

On the second day, the patient received sedation containing of propofol at 40 mg/hour and fentanyl at 20 mcg/hour. Sedation was administered for only four hours, followed by an evaluation of patient awareness. The patient was found to be compos mentis, unable to move all four limbs, unable to swallow saliva, experiencing tetraparesis, unable to breathe spontaneously (Table 1). After then, a CT scan and chest X-ray was taken (Figure 1), and the results showed no sign of cerebral infarction.

This confirmed the finding that blood toxins, not brain injury, were the source of the patient's motor impairment. Ten ampoules of SABU in 500 mL of 0.9% NaCl, given over an hour and repeated every six hours; IVFD Clinimix 1000 cc/24 hours; a clear liquid diet of 6x100 cc; Cefotaxime 2x1 gram IV; Metronidazole 3x500 mg IV; Methylprednisolone 2x125 mg IV; Citicoline 2x1 gram IV; and Omeprazole 2x40 mg IV. The total amount of antivenom administered over the two-day period was 90 ampoules.

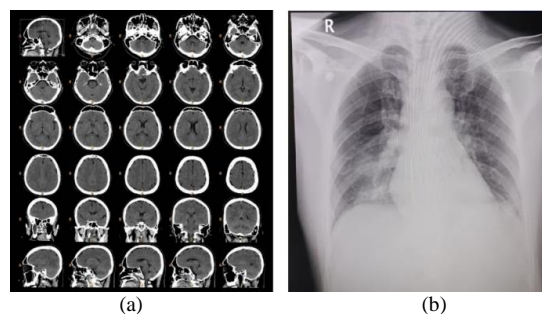


Figure 1- Examination result on patients: (a) CT scans; (b) Chest X-ray

On the third day, based on previous supporting examinations, hemoperfusion therapy was initiated. The procedure was performed using a veno-venous (v-v) hemoperfusion technique with a Jafron HA 230 cartridge filter (Jafron Biomedical Co., LTD, China) [7] for a duration of 6 hours at a flow rate of 80 mL/hour. The development of the patient's hemodynamic status before, during, and after hemoperfusion is shown in Figure 2. The results show that the patients' blood pressure remained stable and within normal limits. The development of the patient's neurological status can also be seen in Table 1, which shows improvement from before and after hemoperfusion. Within the first 24 hours post-hemoperfusion, the patient's neurological status improved (Table 2) with increasingly stable hemodynamic status (Figure 2). The patient showed notable clinical improvement the next day. Extubation readiness was determined by assessing consciousness and respiratory state. The patient's condition improved enough to be transferred from the intensive care unit to an inpatient facility with 48 hours of extubation. The patient's condition gradually improved over the inpatient monitoring period, and no new problems were noted. By the ninth day since admission, the patient was deemed

clinically stable and discharged with instructions for outpatient follow-up.

Table 1- Neurologic status before, during and after hemoperfusion (HP)

Hours/ Condition	Before HP	During HP	+ 1	+ 4	+ 7	After HP (hours)						
Swallowing*	-	-	+	+	+	+	+	+	+	+	+	+
Motoric Status**												
RA/LA/A	1/1/1	1/1/1	2/2/2	2/2/2	3/3/3	3/3/3	3/3/3	3/3/3	4/4/4	4/4/4	5/5/5	
RL/LL/L	1/1/1	1/1/1	2/2/2	2/2/2	3/3/3	3/3/3	3/3/3	3/3/3	4/4/4	4/4/4	5/5/5	

*The ability to swallow: being (+) able to swallow; (-) not being able to swallow.
**Motoric status: (RA) Right Arm; (LA) Left Arm; (A) Arm; (RL) Right Leg; (LL) Left Leg; (L) Leg. Motoric scale: (0) No contraction; (1) Trace muscle contraction; (2) Poor muscle contraction; (3) Muscle contraction; (4) Good muscle contraction; (5) Normal muscle contraction. Adapted from Roman et al. (2022) [8].

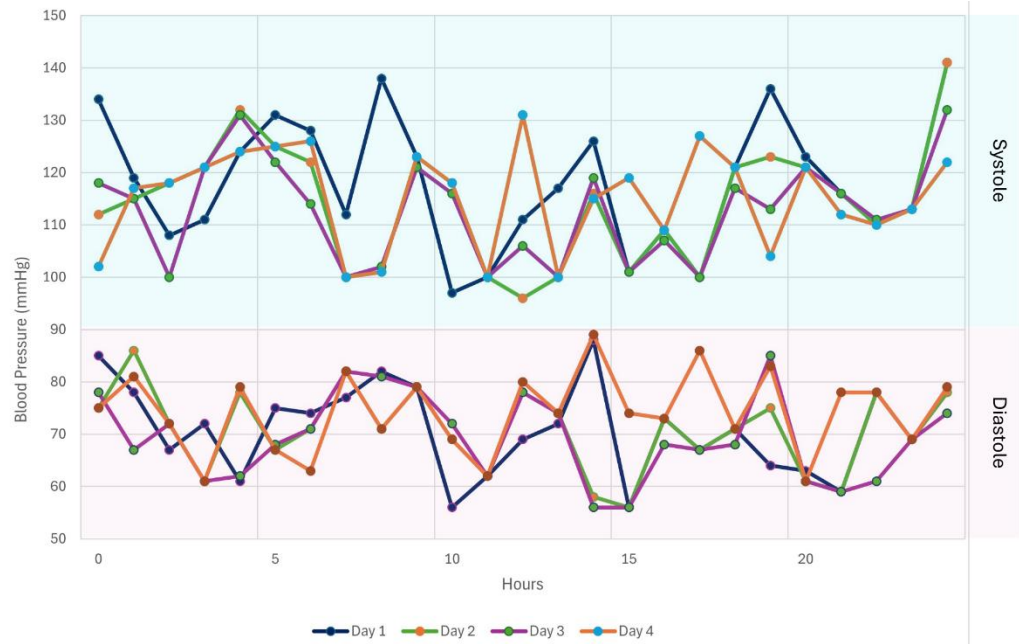


Figure 2- Hemodynamic status (systole and diastole)

Discussion

Snakes can switch from mechanical to chemical strategies for self-defense and subduing prey thanks to an evolutionary breakthrough called venom. Venom originally developed from enzymatic protein found in snakes’ digestive system. However, these proteins can cause significant harmful reactions when they are introduces into the body of another organism. Around 90% of venom composition make up by proteins, including enzymes, non-enzymatic polypeptide toxins, and non-toxic proteins [4,9].

Each component of snake venom exhibits distinct toxic effects on the body. Zinc metalloproteinases can induce spontaneous bleeding during the systemic phase, while procoagulant enzymes trigger blood clotting. Phospholipases A2 contribute a mitochondria damage destruction of blood cells, nerve ending skeletal muscle, and vascular endothelium, Acetylcholinesterase

compound provokes fasciculations, whereas hyaluronidase increases tissue permeability and facilitates tissue degradation. Proteolytic enzymes cause localized edema, blistering, bruising and necrosis at the site envenomation. Additionally, venom polypeptide toxins stimulate acetylcholine release at neuromuscular junctions and impair subsequent neurotransmitter release [4-5,9]. The toxic effects of these components manifest clinically as both local and systemic envenomation.

Both local and systemic envenomation can result from snakebite envenomation. Increased vascular permeability, which causes swelling and bruising at the bite site, is a characteristic of local envenomation. This condition is primarily triggered by the activity of venom endopeptidases, metalloproteinases, hemorrhaging, membrane-damaging polypeptide toxins, phospholipases, and endogenous autacoids that release histamine, 5-HT (serotonin), and kinins. In contrast,

systemic envenomation may induce neuromuscular toxicity, hemotoxicity, and myotoxicity [9].

Management of SBE is the administration of antivenom. In Indonesia, antivenom is commonly referred to as SABU. Antivenom was still the best treatment for neutralizing snake venom, and the World Health Organization (WHO) recognized it as the most effective therapy for that [9]. However, there are several obstacles to its usage, such as problems with formulation, manufacture, distribution, safety and clinical efficacy. Only three snake species are currently covered by Indonesia's polyvalent antivenom: the java cobra (*Naja sputatrix*), the banded krait (*Bungarus fasciatus*), and the Malayan pit viper (*Colloselesmas rhodostoma*). As a result, numerous more poisonous snake species continue to lack antivenom coverage. Apart from the restricted species coverage, SABU is still not widely available. There are now only about eight brands available, and because of their inconsistent availability, many healthcare facilities lack sufficient inventory [9]. Given the comparatively high frequency of poisonous snakebite incidents in Indonesia, the scenario poses a significant concern. That situation creates an urgent need to develop adjuvant therapies that can overcome this limitation, one of which is through the approach of extracorporeal blood purification.

Extracorporeal blood purification is an alternative therapy that works by separating dissolved substances based on molecular mass through blood filtration outside the body, with the main objective of eliminating toxins and specific dissolved substances [10-11]. The therapy differentiated based on membrane separation, mechanisms of action, and absorption capacity for the solute [11]. One of the most frequently performed techniques is hemoperfusion.

Hemoperfusion is a method of cleansing the blood of medium to large molecular weight, lipophilic molecules that bind strongly to proteins. The process is based on the principle of absorption using sorbents and does not cause cell damage. Sorbents are made from natural materials (carbon) and synthetic materials (polymers) that are generally in the form of granules, pellets, flakes, fibers, spheres, and cylindrical pellets [12]. These sorbents are coated and packaged in cartridges to be integrated into an extracorporeal circuit, allowing the blood sufficient contact time to eliminate toxins [11]. Hemoperfusion procedures are generally performed using a hemodialysis (HD) machine or continuous renal replacement therapy (CRRT) [6]. The therapeutic workflow, mechanism, and components of hemoperfusion are illustrated in (Figure 3).

Hemoperfusion is frequently used as an adjunctive therapy to filter toxins in the blood. In this case, the hemoperfusion device adopted was the HA series cartridge from Jafron Biomedical, made of double cross-linked styrene-divinylbenzene copolymers. Drug overdoses and biotoxins, such as snake venom and other poisonous substances, can be effectively managed with this absorbent material [7]. In this instance, pharmacological and pathophysiological mechanisms specifically, the procedure's capacity to lower the venom toxin burden in the blood, hence reducing damage to essential organs-were the main factors considered while performing hemoperfusion [11].

Clinical reports from the patient demonstrated significant improvement, marked by a neurological status progression from grade one to grade five within just 22 hours following hemoperfusion. These results provide credence to hemoperfusion's possible use as an adjuvant treatment for SBE.

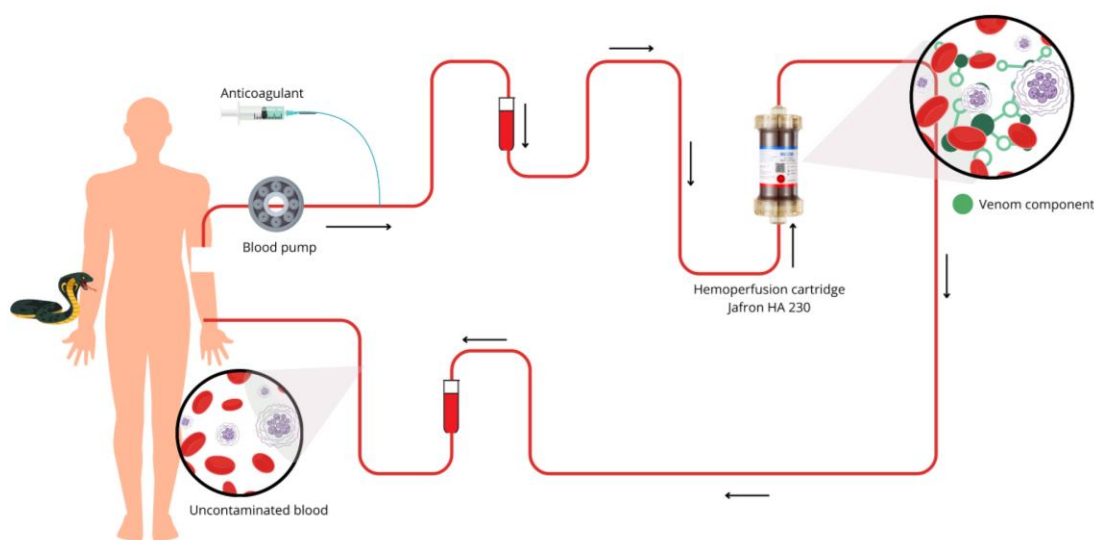


Figure 3- Mechanism of action and therapy flow of hemoperfusion in SBE (created by authors)

These clinical results align with findings from other case reports and experimental investigations. Oliveira et al. (2020) reported that in a trial involving four groups of rats injected with snake venom, the group treated with both venom and hemoperfusion showed outcomes comparable to the group treated with venom and antivenom [13]. These results reinforce the potential of hemoperfusion as an alternative therapy for snakebite envenomation. In line with this, Yoshida et al. (2016) found that a modified hemoperfusion was able to precipitate toxic proteins in snake venom [14]. This study further strengthens the scientific basis for the use of this therapy. In addition to snake envenomation, the effectiveness of hemoperfusion therapy is also evident in other cases of envenomation. Four out of six drug poisoning patients showed clinical improvement after hemoperfusion therapy, according to Mahesh et al (2020) [6]. Similar results were also reported by Li et al. (2020) in patients with multiple organ failure due to wasp stings, where all patients treated with a combination of hemoperfusion and continuous veno-venous hemofiltration experienced improvement and recovery [15].

Based on this evidence, hemoperfusion as an alternative therapy can be considered in envenomation cases, even though it's not yet included in official guidelines. This report expands the literature by demonstrating the benefits of hemoperfusion in human patients with venomous snake envenomation, while highlighting its relevance in Indonesia, which still faces limitations and availability of antivenom in the management venomous animal envenomation

Conclusion

This case illustrates that hemoperfusion may offer clinical benefits in patients with venomous snake envenomation, particularly when antivenom availability is limited and the patient's deterioration is primarily due to the toxic components circulating in the bloodstream. The rapid improvement in the patient's neurological status following the procedure further supports the potential of hemoperfusion as an adjuvant therapy. However, due to the limited evidence from case reports and experimental studies, this therapy cannot yet replace the primary role of antivenom and should be considered cautiously, based on clinical conditions and available resources. Further research is needed to evaluate the efficacy and safety of hemoperfusion in the broader management of SBE.

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