CASE REPORT

Naja Kaouthia Snake Bite: Case Report

Bahtiar Ahmad[✉*], Nancy Margarita Rehatta[**]

[*]RSUD Hj. Anna Lasmanah Banjarnegara, Jawa Tengah, Indonesia
[**]Departmen of Anesthesiology and Reanimation, Faculty of Medicine, Airlangga University/ Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

[✉]Correspondence: bahhyamor27@gmail.com

ABSTRACT
Background: Snakebites prevalent globally, pose a severe threat, especially in resource-limited regions. The impact is substantial, affecting millions annually, with a significant number of fatalities.
Case: A 37-year-old man experienced deterioration leading to cardiac arrest after being bitten by a Naja Kaouthia snake. Adequate management in the intensive care unit (ICU), including antivenom administration, restored the patient's condition to return of spontaneous circulation (ROSC).
Discussion: Snake venom causes various symptoms, from tissue damage to breathing and heart issues. Treating with antivenom, specifically from horses, can neutralize the venom's effects, as shown in studies. This treatment is effective for Naja Kaouthia bite.
Conclusion: Quick treatment with antivenom partially neutralized the venom, showing how important antivenom is for treating snakebites.

Keywords: antivenom; envenomation; Indonesia; Naja Kaouthia; snakebite

INTRODUCTION

Snakebites have a significant impact on health and survival worldwide, especially in regions with limited resources. There are more than 600 known species of venomous snakes across the globe, with the majority belonging to the Viperidae and Elapidae families.[1] Snakebites are most commonly associated with occupational exposure, affecting individuals like farmers, hunters, and even tourists exploring the outdoors.[2] When a person is bitten by a snake, the initial damage primarily occurs at the bite site and can progress to systemic toxicity, depending on the snake species involved. Estimates suggest that between 1.2 to 5.5 million people worldwide experience snakebites annually, with as many as 94,000 of these incidents resulting in fatalities.[3]

A case report of snakebite by the Naja Kaothia species holds significant novelty in the medical field as it delineates a patient's journey from snakebite to experiencing cardiac arrest and subsequent recovery. Snakebites often present complex medical challenges, and this case report provides additional insights into effective clinical management of similar conditions.
The purpose of this case report is to share experiences in handling snakebite cases that progress to critical conditions, emphasizing the importance of appropriate management and successful interventions to address serious complications such as cardiac arrest.

CASE

Previously a 37-year-old male patient presented at the hospital with a complaint of being bitten on his left leg by a Naja Kaouthia snake. The patient had been bitten approximately 30 minutes before arriving at the hospital and had a history of a snakebite six months earlier. His complaints included pain at the bite site, nausea, one episode of vomiting, blurred vision after one hour, and feelings of shortness of breath and confusion two hours into observation. The patient experienced a cardiac arrest but was successfully resuscitated after being transferred to the ICU.

On physical examination, the patient appeared confused with high blood pressure and tachycardia. Oxygen saturation was 99% with 2-3 lpm nasal cannula. Urine output was within normal limits. Laboratory tests for complete blood count, liver and kidney function were revealed within normal limits (Table 1).

Figure 1. Naja Kaouthia snake

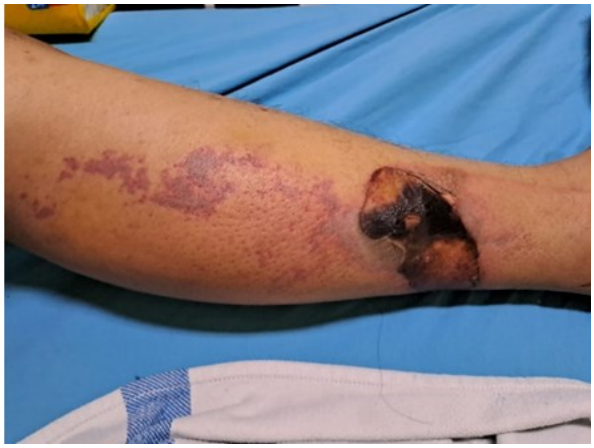

Figure 2. Necrotic wound caused by snake bite

Table 1. Laboratory examination results

Parameters	Unit	Day 1	Day 2 (morning)	Day 2 (night)
Hemoglobin	g/dL	16.30	19.2	19.4
Hematocrit	%	49.00	53.5	51.6
Erythrocytes	/uL	5.46	6.02	5.78
MCV	fl	89.70	88.6	89.3
MCH	pg	29.80	31.9	33.5
MCHC	g/dl	33.20	36.1	37.5
RDW	%	14.20	13.2	13.3
Leucocytes	10^3(uL)	8.93	24.69	26.51
Platelet	10^3(uL)	282	362	283
SGOT	U/L	23	185	65
SGPT	U/L	25	132	97
Ureum	(mg/dL)	28	27.7	42.5
Creatinine	(mg/dL)	0.93	0.78	0.8
PT	Seconds	8.40	9.40	9.3
PT INR		0.92	1.03	1.02
aPTT	Seconds	25.20	26.30	22.90
D-Dimer	ng/mL	467		
Natrium	mmol/L	138		
Kalium	mmol/L	4.40		
Clorida	mmol/L	107		

The patient was given two vials of snake antivenom, 1 gr ceftriaxone IV every 12 hours, 30 mg ketorolac IV every 8 hours, 500mg tranexamic acid IV every 8 hours, 50 mg ranitidine IV every 12 hours, 40 mg omeprazole IV every 24 hours, 500mg paracetamol IV every 8 hours and 5mg dexamethasone IV every 8 hours.

During the next 3 hours, the patient became coma and continued with cardiac arrest. The patient was successfully resuscitated after three cycles of cardiopulmonary resuscitation (CPR) following intubation and mechanical ventilation. The patient was then transferred to the ICU for further observation and stabilization, with total bed rest and nasogastric tube placement.

The mechanical ventilator setting was synchronized intermittent mandatory ventilation (SIMV) mode at a rate of 14 breaths per minute, with Tidal Volume of 400 milliliters. The patient is receiving a Positive end-expiratory pressure (PEEP) of 4 cm H2O with an FiO2% of 90% and the ratio of inspiration to expiration time is 1:2.

The next day, the laboratory test revealed an elevated white blood cell count, platelet count, and decreased hematocrit and hemoglobin levels. Therefore, the patient got another antivenom that was specific to this case, 1 vial of Neuro Polyvalent Snake Antivenom in 500cc NaCl 0.9%/8 h (loading dose 3 vials in the first 24h).

Monitoring included subjective patient assessment, vital signs, and urine output. Laboratory tests were repeated over time, and the patient's condition improved, his vital signs were stable (Figure 3, Figure 4), and there was a trigger for spontaneous breathing.

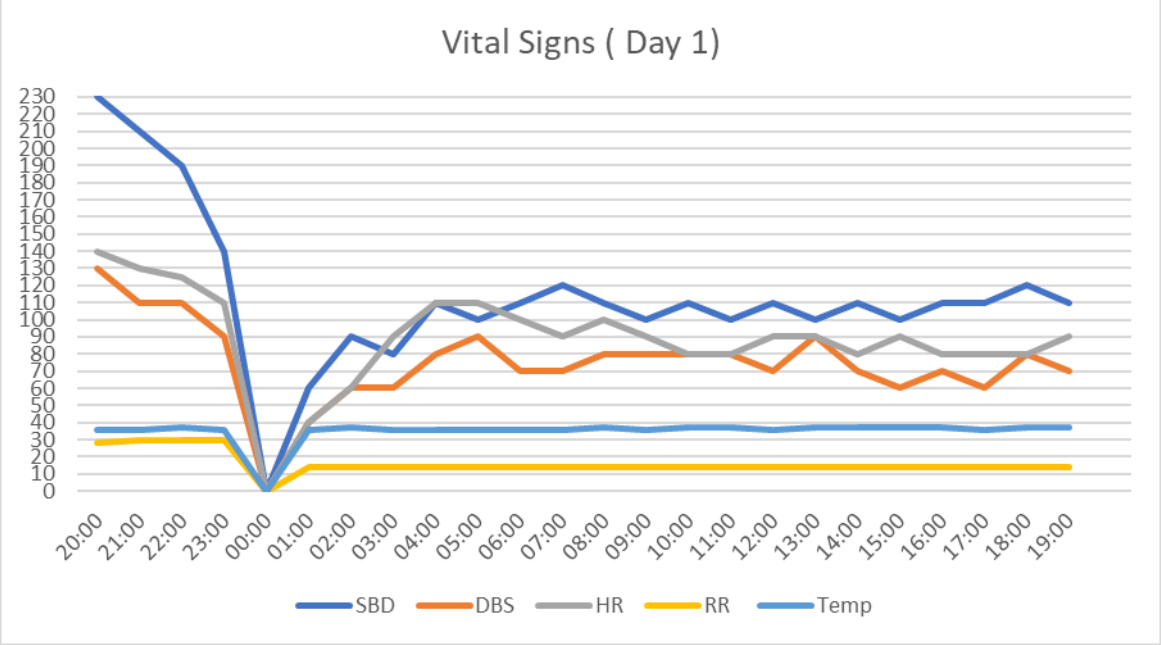

Figure 3. Vital signs patient on day 1
SBD : Systolic Blood Pressure (mmHg) ; DBS : Diastolic Blood Pressure (mmHg) ; HR : Heart Rate (x/minute) ; RR : Respiration Rate (x/minute) on Mechanical Ventilation, Temp : Temperature (℃)

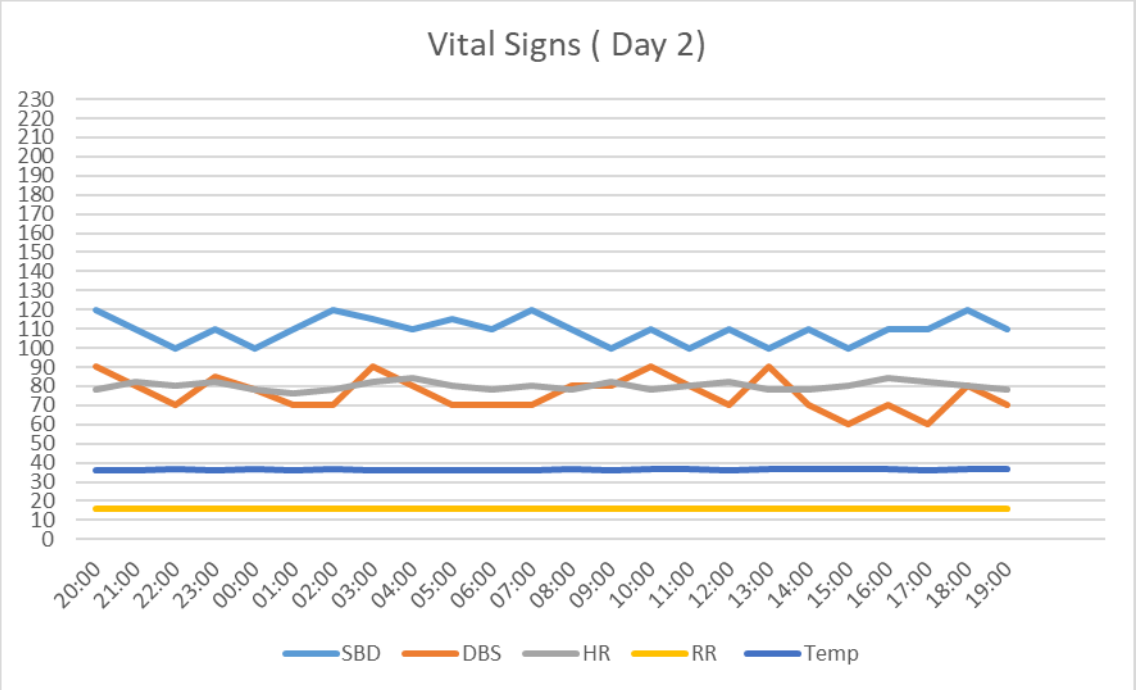

Figure 4. Vital signs patient on day 2
SBD : Systolic Blood Pressure (mmHg) ; DBS : Diastolic Blood Pressure (mmHg) ; HR : Heart Rate (x/minute) ; RR : Respiration Rate (x/minute) on Mechanical Ventilation, Temp : Temperature (℃)

The mechanical ventilator setting was changed to Spontaneous mode at a rate of 16 breaths per minute, with a tidal volume of 400 millilitres. The patient is receiving a PEEP of 4 cm H2O with an FiO2% of 50% and the ratio of inspiration to expiration time is 1:2.

This case report illustrates the effective management of snakebite-induced complications, including cardiac arrest, underscoring the importance of proper intervention and successful measures in handling serious snakebite cases.

DISCUSSION

The symptoms resulting from snakebites are mainly attributed to the toxic components found in snake venom. The composition of venom varies from species to species and can range from causing localized tissue damage to severe coagulation disorders.[2] The clinical impact on humans is influenced by both the potency and the quantity of venom injected during the snakebite. A single species of venomous snake can have a venom composed of up to 100 different toxic elements.[4]

Phospholipase A2, a common component of snake venom, also contributes to local tissue damage and has systemic effects on blood vessels and nerve endings. Snake venom contains various proteins and polypeptides with toxic effects, including neurotoxins and hemotoxins. Neurotoxins primarily damage the neuromuscular junction, affecting both the presynaptic and postsynaptic terminals. This can lead to generalized weakness, ptosis, ophthalmoplegia, facial muscle paralysis, and, ultimately, respiratory failure due to diaphragm paralysis.[5,6] Hemotoxic effects may be evident through significant bleeding at the bite site, nosebleeds, or spontaneous bleeding. Snakebite-induced shock can result from venom-induced vasodilation, hypovolemia, or even anaphylactic reactions in some cases. In certain snake species, such as Vipers and Elapids, cardiotoxic effects may manifest as arrhythmias, bradycardia, tachycardia, or hypotension.[7,8]

Cobra venom's cardiotoxin, while less potent than its neurotoxin, directly affects cell membranes, leading to various effects on muscles, nerves, and neuromuscular junctions, ultimately contributing to circulatory and respiratory paralysis and cardiac asystole. This is due to the irreversible depolarization of cell membrane transport mechanisms and asystolic cardiac arrest, possibly resulting from the release of calcium ions from the myocardium's surface membrane.[9]

Envenomations resulting from Naja Kaouthia (N. kaouthia) bites are typically characterized by localized tissue damage and a variety of neurotoxic effects, such as weakness in respiratory and skeletal muscles. While hematologic toxicity and rhabdomyolysis are infrequent, they have been documented in some cases.[10] Notably, cardiovascular symptoms are atypical for N. Kaouthia envenomations. The underlying cause of bradycardia and hypotension in the first patient remains unclear, with one possible explanation being a vasovagal response.[11] In our patient, the presenting complaints include pain at the bite site, nausea, vomiting, blurred vision, shortness of breath, and confusion. Subsequently, the patient was transferred to the ICU and experienced a cardiac arrest, followed by successful return of spontaneous circulation (ROSC). On physical examination, the patient appears weak, and there are evident hematological

abnormalities, including leukocytosis, trombocystosis, decrease in haemoglobin and hematocrit. Furthermore, there are signs of acute liver injury that can be known from an abrupt increase in the serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT).

The specific treatment for snake envenomation is antivenom; Certain cobra venoms contain alpha toxins that function postsynaptically to counteract nicotinic cholinergic receptors.[12,13] Cholinesterase inhibitors like neostigmine and edrophonium elevate acetylcholine levels at the neuromuscular junction, allowing the neurotransmitter to compete with toxins. Successfully employed in envenomations caused by N. Kaouthia and other neurotoxic snakes, these inhibitors can effectively postpone or potentially avert the necessity for mechanical ventilation.[9] Our Patien received Polyvalent equine immunoglobulins, specifically anti-snake venom serums, commonly utilized in the management of snakebites. The agent obtained from horses and include immunoglobulin Fab fragments. Scientific investigations have validated the effectiveness and safety of polyvalent F(ab')2 equine antivenom in the treatment of snake bites, with a particular emphasis on species like Echis ocellatus (viper).[14,15] In Zheng study, the polyvalent antivenom demonstrated partial neutralization of both biochemical and biological activities at a 1:10 ratio (venom: polyvalent antivenom). Complete neutralization was achieved when the dose of the polyvalent antivenom was increased to tenfold. The partial inhibition is likely attributed to the antibodies against Naja naja proteins present in the polyvalent antivenom, which recognize the proteins in the Naja Kaouthia venom.[10] This study provides evidence that, under in vitro conditions, the polyvalent antivenom can effectively neutralize certain tested biochemical and biological activities of Naja Kaouthia venom.

CONCLUSION

The patient, post-Naja Kaouthia snakebite, exhibited initial localized symptoms escalating to severe manifestations, including cardiac arrest. Timely medical intervention involved the use of polyvalent antivenom, resulting in partial venom neutralization at a 1:10 ratio and complete neutralization at a tenfold increase. The partial inhibition was attributed to antibodies against Naja naja proteins present in the antivenom.

This highlights the crucial role of antivenom in addressing snakebite complications. The study findings underscore the significance of ongoing research to refine treatment protocols, enhancing our ability to manage the diverse and potentially life-threatening consequences of snake envenomation.

REFERENCES

1. Meyers SE, Tadi P. Snake Toxicity. 2023.
2. Warrell DA. Venomous Bites, Stings, and Poisoning. Infect Dis Clin North Am. 2019 Mar;33(1):17–38. Doi:10.106/j.idc.2018.10.001
3. Bawaskar HS, Bawaskar PH. Snakebite envenoming. The Lancet. 2019 Jan;393(10167):131. Doi:10.1016/S0140-6736(18)32745-4
4. Tednes M, Slesinger TL. Evaluation and Treatment of Snake Envenomations. 2023.

5. Mehta S, Sashindran V. Clinical Features And Management Of Snake Bite. Med J Armed Forces India. 2002 Jul;58(3):247–9. Doi.10.1016/S0377-1237(02)80140-X
6. Charles J Gerardo, et al. Does this Patient Have a Severe Snake Envenomation?" The Rational Clinical Examination Systematic Review. JAMA surgery. 2019 Apr 1 ; 154(4):346-354. doi:10.1001/jamasurg.2018.5069
7. Virmani S. Cardiac Involvement In Snake Bite. Med J Armed Forces India. 2002 Apr;58(2):156–7.
8. Aditya John, Ajay K, et al. Cardiovascular manifestations and patient outcomes following snake envenomation : a pilot study. Tropical doctor 2019 ; 49(I) 10-13. doi : 10.1177/0049475518814019
9. Greene SC, Osborn L, Bower R, Harding SA, Takenaka K. Monocled Cobra (Naja kaouthia) Envenomations Requiring Mechanical Ventilation. J Emerg Med. 2021 Feb;60(2):197–201. doi : 10.1016/j.jemermed.2020.10.014
10. Zeng L, Hou J, Ge C, Li Y, Gao J, Zhang C, et al. Clinical study of anti-snake venom blockade in the treatment of local tissue necrosis caused by Chinese cobra (Naja atra) bites. PLoS Negl Trop Dis. 2022 Dec 16;16(12):e0010997.
11. Averin AS, Utkin YN. Cardiovascular Effects of Snake Toxins: Cardiotoxicity and Cardioprotection. Acta Naturae. 2021 Nov 15;13(3):4–14. doi : 10.32607/actanaturae.11375
12. Williams KL, Woslager M, Garland SL, Barton RP, Banner W. Use of polyvalent equine anti-viper serum to treat delayed coagulopathy due to suspected *Sistrurus miliarius streckeri* envenomation in two children. Clin Toxicol. 2017 May 28;55(5):326–31. Doi: 10.1080/15563650.2017.1284334.
13. Rupeng Mong, et al. Safety profile of snake antivenom (use) in Hong Kong- a review of 191 cases from 2008 to 2015. Clin Toxicol. 2017 Dec ; 55 (10):1066-1071. doi :10.1080/15563650.2017.1334916
14. Das D, Urs N, Hiremath V, Vishwanath BS, Doley R. Biochemical and biological characterization of Naja kaouthia venom from North-East India and its neutralization by polyvalent antivenom. J Venom Res. 2013;4:31–8.
15. Leong PK, Sim SM, et al. Cross neutralization of Afro-Asian cobra and Asian krait venoms by a Thai polyvalent snake antivenom (Neuro Polyvalent Snake Antivenom) PLoS Negl Trop Dis. 2012 ; 6:e1672. doi : 10.1371/journal.pntd.0001672.