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Case Reports

REVERSAL OF SEVERE VENOM-INDUCED CONSUMPTIVE COAGULOPATHY FOLLOWING DELAYED ANTIVENOM ADMINISTRATION IN A PEDIATRIC GREEN PIT VIPER ENVENOMATION

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ABSTRACT

Background. Green pit viper (*Trimeresurus* spp.) envenomation can cause venom-induced consumptive coagulopathy (VICC), for which species-specific antivenom is the cornerstone of therapy. The standard Indonesian trivalent antivenom does not cover *Trimeresurus*. This report aims to describe the presentation, antivenom dosing, and hematologic recovery of a child with severe VICC after a green pit viper bite and ineffective trivalent antivenom.
Case. A 3-year-old boy (15 kg) was referred to a tertiary hospital three days after a finger bite, with swelling of the entire right arm. At a district hospital, coagulation was initially normal (PT 13.1 s, platelets 440,000/µL), and trivalent antivenom produced no improvement.
Findings. On referral (Day 0), PT and APTT exceeded 180 s, INR was unmeasurable, and platelets were 6,000/µL. A green snake with a red tail was described, and green pit viper envenomation was diagnosed clinically. Because of limited stock, he received two vials of monovalent green pit viper antivenom on Day 0 (platelets 10,000/µL on Day 1), followed by three vials on Day 1. By Day 2, platelets were 63,000/µL, PT 10.7 s, APTT 15.8 s, and INR 0.99; platelets reached 163,000/µL on Day 4. No transfusion or antivenom reaction occurred, and he was discharged on Day 10.
Conclusion. Coagulopathy resolved after species-specific antivenom despite a 72-hour delay, whereas non-specific antivenom had no apparent effect. This case supports antivenom selection guided by clinical toxinology, adult-equivalent vial dosing in children, and repeat coagulation testing, particularly in resource-limited settings.

Keywords: Antivenom; Green Pit Viper; Pediatric; Snakebite; Venom-Induced Consumptive Coagulopathy.

BACKGROUND

Snakebite envenoming is a neglected tropical disease, with an estimated 5 million bites worldwide each year [1], more than 100,000 deaths, and more than 400,000 cases of permanent disability [2]. In Indonesia, arboreal pit vipers of the *Trimeresurus* complex are medically important west of Wallace's line [3]. Accurate incidence data are scarce because many victims consult traditional healers and are never registered [4]. According to WHO, 1.8 to 2.7 million of these bites result in envenoming, and up to 2 million cases occur in Asia. Indonesia currently has no national snakebite data because no accurate national

reporting system exists. At our institution, Dr. Saiful Anwar General Hospital, Malang, emergency department data for 2025 recorded 97 snakebite cases, of which 14 (14.43%) were in children under 18 years of age. *Trimeresurus* envenomation accounted for 19 cases (19.59%) overall, including 4 pediatric patients.

Green pit vipers of the genus *Trimeresurus* are a common cause of venomous bites in Southeast Asia [5]. Their venom contains procoagulant enzymes, including thrombin-like enzymes, together with hemorrhagic and cytotoxic components [6,7], and typically produces progressive local swelling, tissue necrosis and venom-induced consumptive coagulopathy (VICC) [5,7].

VICC results from procoagulant toxins that activate the clotting cascade and consume clotting factors, and in severe cases clotting times become unmeasurable [8]. Antivenom is the only specific treatment for envenoming [1,2], although high-quality evidence for its clinical effectiveness in VICC is limited and differs between snake species [9]. Antivenoms are generally species-restricted. In Indonesia, the existing antivenom is the trivalent Biosave (SABU), covering *Calloselasma rhodostoma*, *Naja sputatrix* and *Bungarus fasciatus* but not *Trimeresurus* spp., and its misuse for *Trimeresurus* envenoming has been associated with poor outcomes [3]. When the snake is not identified, empirical use of the wrong product can delay effective therapy. Children are at particular risk of severe systemic envenoming because a given venom dose is distributed into a smaller volume [10]. The same antivenom protocol is recommended for children and adults [11].

Several reports have described *Trimeresurus* envenoming. In Thailand, Hutton et al. studied 29 patients bitten by arboreal green pit vipers and reported that five ampoules of antivenom restored coagulability, although venom antigenaemia persisted and coagulopathy recurred in some patients [5]. A pediatric case from Hong Kong described severe coagulopathy after a green pit viper bite that did not respond to a non-specific antivenin but resolved after green pit viper antivenin [12]. Preclinical work showed that Thai Green Pit Viper Antivenom neutralizes venoms of Indonesian *Trimeresurus* species far more effectively than the trivalent product, but its clinical effectiveness awaits investigation [3]. Overall evidence for antivenom in VICC remains limited [9], and pediatric data come mainly from other regions and snake species [11,13,14], with antivenom dosing in children still poorly defined [14]. Advanced monitoring such as thromboelastography has been proposed for VICC but is costly and seldom available in resource-limited settings [15].

The aim of this report was to describe the presentation, stepwise antivenom dosing, and hematologic recovery of a 3-year-old child with severe VICC after green pit viper

envenoming in Indonesia, following ineffective trivalent antivenom and a 72-hour delay. It adds clinical observations to preclinical data on species-specific antivenom [3] and to pediatric reports from other settings [11–14].

CASE PRESENTATION

A 3-year-old boy was referred to our tertiary hospital on Day 0 of his hospital stay, three days after sustaining a snakebite to his right hand. The snake was described by the family as a green snake with a red tail, consistent with a Trimeresurus species (Figure 1). Prior to referral, the patient had sought care at a district hospital where, due to the family's inability to provide specific snake identification, the patient was treated as an undetermined snakebite and administered a trivalent antivenom (likely Serum Anti Bisa Ular/Biosave, which targets Naja sputatrix, Bungarus fasciatus, and Calloselasma rhodostoma). This initial non-specific antivenom therapy failed to yield clinical improvement. At a district hospital in Malang Regency on Day −3 (24 March 2025), PT was 13.1 s and platelet count 440,000/µL.

His local symptoms, characterized by progressive swelling and pain in the right hand, worsened over the next few days. Initially, the middle finger of his right hand was noted to be swollen and bluish, with swelling progressively extending to involve the entire right hand upon presentation to our facility (Figure 2). Upon physical examination in the emergency department, the patient weighed 15 kg. His vital signs indicated mild distress likely secondary to localized pain, presenting with a blood pressure of 100/60 mmHg, a heart rate of 115 beats per minute (moderate tachycardia), a respiratory rate of 24 breaths per minute, a body temperature of 37.2°C, and an oxygen saturation of 98% on room air.

Laboratory investigations upon admission (Day 0) revealed severe coagulopathy, a common and serious manifestation of viper envenomation [8]: Prothrombin Time (PT) and Activated Partial Thromboplastin Time (APTT) were both markedly prolonged to >180 seconds (control values for PT: 11.0-13.0 seconds, APTT: 25.0-35.0 seconds), and International Normalized Ratio (INR) was reported as "No coagulation." Concurrently, the patient exhibited severe thrombocytopenia with a platelet count of 6,000 /µL. A complete blood count also showed anemia (Hemoglobin (HGB): 8.90 g/dL) and leukocytosis (White Blood Cell (WBC): 13,330 /µL). Other baseline parameters included normal electrolytes (Na: 140, K: 3.56, Cl: 103), slightly elevated liver transaminases (AST/ALT: 52/23 U/L), normal albumin (3.85 g/dL), elevated random blood glucose (171 mg/dL), and normal renal function (Urea/Creatinine: 16.8/0.46 mg/dL).

FINDINGS

Upon admission, the patient received comprehensive supportive care, which included intravenous fluid administration, analgesics (Inj. Metamizole 3x150 mg), broad-spectrum antibiotics to prevent secondary bacterial infection, and tetanus immunoglobulin (Inj. Tetagam 250 IU) as per standard guidelines for snakebite management. Given the severe coagulopathy, Inj. Vit. K x 21 mg was also administered to support clotting factor synthesis.

Crucially, throughout the patient's hospital stay and despite severe initial coagulopathy and thrombocytopenia, no platelet concentrates or Fresh Frozen Plasma (FFP) transfusions were administered. This approach follows recommendations that blood products be reserved for active major bleeding unresponsive to antivenom [8,9]. Transfusions carry inherent risks, including allergic reactions, transfusion-related acute lung injury (TRALI), and transfusion-associated circulatory overload (TACO) [9].

The primary intervention for envenomation was specific antivenom therapy. Due to initial institutional limitations in antivenom stock, the first dose of Greenpit Viper antivenom commenced with 2 vials, diluted in 75 cc of normal saline and infused over 1 hours (1-2 cc/minute) on Day 0.

On Day 1, follow-up laboratory tests revealed persistent severe thrombocytopenia (10,000/µL) and likely ongoing coagulopathy (as evidenced by persistent severe thrombocytopenia, with PT/APTT values not re-evaluated or available for this time point), indicating an insufficient initial response to the antivenom. Recognizing the ongoing venom effects and the patient's critical coagulation status, efforts were made to procure additional antivenom. Following successful acquisition of more stock, an additional 3 vials of Greenpit Viper antivenom were administered. This second dose was prepared by diluting the 3 vials in 75 cc of 0.9% NaCl and infused at a rate of 100 cc/hour (1-2 cc/minute). The total dose of antivenom administered was 5 vials. The dosage of antivenom in children is generally the same as in adults, as it depends on the amount of venom to be neutralized rather than body weight. [10,11]



Figure 1. Snake picture provided by patient parent

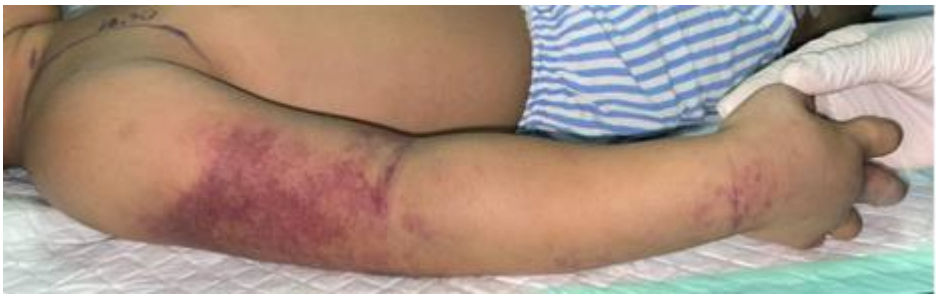


Figure 2. Clinical appeareance in tertiary hospital

The patient's clinical and laboratory parameters were closely monitored, demonstrating a remarkable response to adequate antivenom therapy.

Parameter	Day 0 (Initial)	Day 1 (Post 2 Vials AV)	Day 2 (Post 5 Vials AV)	Day 3 (Continued Improvement)	Reference Range
PT (seconds)	>180	-	10.70	9.90	10.0 - 14.0
APTT (seconds)	>180	-	15.80	18.30	25.0 - 35.0
INR	No coagulation	-	0.99	0.95	0.8 - 1.2
Platelets (103/L)	6,000	10,000	63,000	105,000	150,000 - 450,000
HGB (g/dL)	8.90	8.90	7.90	8.50	11.5 - 13.5
WBC (103/L)	13,330	13,220	10,040	8,660	4,000 - 11,000
Neutrophil (%)	-	72.10	40.70	49.80	40 - 75
Lymphocyte (%)	-	18.70	49.90	40.00	20 - 45

Coagulation Parameters: A dramatic and rapid normalization of PT, APTT, and INR was observed by Day 2, following the complete administration of 5 vials of antivenom. Further improvement was sustained on Day 3, with parameters well within therapeutic range. .

Platelet Count: Platelets showed a significant and progressive increase from a profound nadir of 6,000 103/L to 105,000 103/L by Day 3. This rise, without platelet transfusion, is consistent with neutralization of platelet-depleting venom components by the antivenom [5]. **Other Hematologic Parameters**: While anemia (HGB 7.90 g/dL) and leukocytosis (WBC 10.04 103/L) persisted on Day 2, both parameters showed signs of improvement by Day 3, indicating resolution of systemic inflammatory response and possibly recovery from venom-induced hemolysis or bleeding. The shift in differential white blood cell count (decreasing neutrophils, increasing lymphocytes) further supports resolving acute inflammation. The patient continued to stabilize and was discharged on Day 10, with follow-up scheduled as an outpatient for routine wound care.

DISCUSSIONS

This case shows that severe pediatric envenomation by a green pit viper (Trimeresurus spp.) can evolve over several days into venom-induced consumption coagulopathy (VICC) with unmeasurable clotting times, and that the outcome depended on three decisions: giving species-appropriate antivenom, giving enough of it, and recognizing early that empirical treatment had failed. Snakebite envenoming is a neglected tropical disease with substantial morbidity in rural, low-resource settings, and green pit vipers remain an important cause of hemotoxic snakebite across Asia [1,2,6,16]. Our patient was referred late, had received inappropriate empirical antivenom at the primary-care level, and recovered hematologically only after a cumulative five vials of monovalent green pit viper antivenom.

Pediatric Vulnerability, Venom Load and Antivenom Dosing

Children are more vulnerable to systemic toxicity because a snake does not adjust the amount of venom it injects to the size of its victim. A small child, for example one weighing 15 kg, may receive a venom dose comparable to that delivered to a 70-kg adult, but it is distributed across a much smaller blood volume and body water space. The result is a higher effective circulating toxin concentration and faster local and systemic effects [10,11,13]. This is consistent with the rapid course in our patient, from a normal coagulation profile on Day −3 to unmeasurable PT and aPTT within 72 hours. The smaller limb of a child may also tolerate venom-induced edema poorly, increasing the risk of compromised perfusion and tissue injury. Pediatric data link poor outcomes to delayed presentation, severe swelling and coagulopathy [14], all of which were present in our patient.

For the same reason, antivenom is not dosed by body weight. The therapeutic target is the injected venom load rather than a mg/kg exposure, so a child needs the same number

of vials as an adult [11]. Scaling the dose down to body size risks under-treatment and continued consumption of clotting factors. What can be adapted is the diluent volume, which should be chosen according to the product instructions and local protocol to avoid fluid overload in young children. In our patient, a cumulative five vials were required for hematologic recovery, as discussed below.

Pathophysiology of VICC in Trimeresurus Envenomation

VICC is the principal hematologic syndrome of snake envenoming. It is caused by procoagulant toxins that activate the clotting cascade and consume fibrinogen and other clotting factors, and it should be distinguished from classic disseminated intravascular coagulation (DIC). The term DIC is best reserved for cases with independent evidence of systemic microthrombi and organ failure attributable to DIC [8,17]. Trimeresurus venoms are enriched in serine proteases and snake venom metalloproteinases, including thrombin-like enzymes, with additional toxins contributing to hemorrhage, edema, necrosis, thrombocytopenia and defibrinogenation [6,7]. Thrombin-like enzymes typically cleave only a single fibrinopeptide, producing abnormal fibrin that is rapidly degraded, which explains why prolonged PT and aPTT and low or absent fibrinogen are the central laboratory findings [9,18]. In severe VICC, clotting assays can become unmeasurable and fibrinogen almost completely depleted, as reported in other snake groups [18–20].

Our patient followed this pattern. Coagulation was normal at the district hospital on Day −3, but by Day 0 PT and aPTT exceeded 180 seconds and the platelet count had fallen from 440,000 to 6,000/μL. An initially normal profile therefore does not exclude later VICC, which supports repeated clotting assessment in every child with a suspected viper bite [10,11]. The thrombocytopenia was probably multifactorial, reflecting platelet-active venom components together with consumption during ongoing fibrin formation.

Delayed Antivenom, Dose–Response and Recovery of Clotting

Species-appropriate antivenom is the only specific therapy for systemic envenoming [1,2]. It neutralizes circulating toxins promptly but supplies no clotting factors, so normalization of coagulation depends on hepatic resynthesis, which usually takes 24–48 hours after venom neutralization [4,8,15,21]. Our patient illustrates this principle. Monovalent antivenom was effective even though it was started about 72 hours after the bite, because active systemic envenomation, shown by persistently abnormal clotting times, was still present. Ongoing coagulopathy remains an indication for antivenom irrespective of the time elapsed since the bite [8,13,15].

The dose–response relationship was clear. Because of limited stock, the first dose was only 2 vials, and on Day 1 the platelet count had risen only marginally, from 6,000 to 10,000/µL, indicating incomplete neutralization. After 3 further vials (5 in total), the platelet count reached 63,000/µL within 24 hours (Day 2) and coagulation normalized. Persistent or recurrent coagulopathy after an initial dose signals residual circulating venom and calls for further antivenom [10,11], which is why serial clinical and laboratory reassessment after each dose is essential. Because recovery of clotting factors lags behind neutralization, the partial effect of the first 2 vials cannot be fully separated from the effect of the subsequent 3, a limitation inherent to a single-patient observation.

Comparison with Previous Reports

Our findings parallel earlier observations. In Thai patients bitten by arboreal green pit vipers, five ampoules of antivenom restored coagulability [5], the same cumulative number of vials our patient received, and a Hong Kong child with severe coagulopathy recovered promptly after switching from a non-specific antivenin to green pit viper antivenin [12]. Our report differs in several respects: the patient was 3 years old and presented in Indonesia, where the available trivalent product does not cover *Trimeresurus* [3]; antivenom was started about 72 hours after the bite; and limited stock produced a stepwise regimen that allowed the response to be observed after two vials and after five. Recurrent coagulopathy, described in some patients with persistent venom antigenaemia [5], was not documented in our patient, although monitoring beyond Day 4 is not reported. These observations provide clinical support for preclinical cross-neutralization data on Thai green pit viper antivenom against Indonesian *Trimeresurus* venoms [3], but they do not replace controlled clinical evaluation.

Antivenom Selection and Cross-Neutralization in Resource-Limited Settings

The initial management shows how syndromic, empirical treatment can fail when the biting species is unknown. In Indonesia the standard antivenom is a trivalent product (Biosave/SABU) directed against Naja sputatrix, Bungarus fasciatus and Calloselasma rhodostoma, not against Trimeresurus species. Cross-neutralization among pit viper antivenoms is variable and cannot be assumed from taxonomy alone [1,15]. Experimental work shows that some non-specific or related antivenoms neutralize selected Trimeresurus venoms while others perform poorly [6,15], so local species distribution and product-specific evidence matter more than a generic “viper” label. In our patient the empirical trivalent antivenom was ineffective, which is the expected result when the product does not cover the offending venom.

Clinical clues were available. Rapidly progressive swelling together with severe VICC points to viperid envenomation, and failure to improve after trivalent antivenom should place Trimeresurus envenomation high in the differential diagnosis wherever green pit vipers are endemic. The history obtained at the tertiary center, of a small green snake with a red tail, was compatible with Trimeresurus insularis or T. albolabris, although the species could not be determined with certainty. Continued empirical use of a non-covering product delays definitive care and exposes the child to foreign animal proteins, with a risk of anaphylaxis or serum sickness, without therapeutic benefit. This case supports better toxinology training for primary-care physicians and wider availability of monovalent antivenoms for locally relevant species at regional and district hospitals.

Blood Products in VICC

Replacing clotting factors before venom is neutralized does not address the cause of VICC. Reviews advise that fresh frozen plasma (FFP) is generally unnecessary before effective antivenom and should be reserved for active major bleeding [2,8,9,15]. Factors given while toxins are still active may act as additional substrate and worsen consumption, and rising D-dimer after FFP has been described [9,15,22]. After antivenom, FFP can speed laboratory correction, but trials found no clear clinical benefit such as earlier discharge, and transfusion carries its own risks, including TRALI and anaphylaxis [9]. Pediatric series also report fewer transfusions in children who received antivenom [13]. In our patient, clotting normalized after adequate venom neutralization and endogenous resynthesis, which suggests that the limiting problem was insufficient neutralization rather than a lack of donor clotting factors. Transfusion decisions still need to be individualized to bleeding severity and local expertise.

Study Limitation

This report has several limitations. First, it describes a single patient, so the findings cannot be generalized and causality cannot be established. Second, the data were obtained retrospectively from clinical records, and some measurements were unavailable, notably PT and APTT on Day 1, which limits assessment of the early response to the first two vials. Third, the biting species was not confirmed: identification relied on the family's description and a photograph, without a specimen or venom assay, so *Trimeresurus* envenomation remains a clinical diagnosis. Fourth, fibrinogen and D-dimer were not measured. Fifth, because recovery of clotting factors lags behind venom neutralization, and because vitamin K was also given, the effect of the first two vials cannot be separated from that of the subsequent three. Sixth, spontaneous recovery of clotting factors cannot be fully excluded,

since resynthesis may occur irrespective of antivenom in some envenomations [19], although persistent coagulopathy about 72 hours after the bite and an incomplete response after two vials argue against this. Finally, follow-up was limited to outpatient wound care, so long-term outcomes are unknown, and the evidence base for antivenom in VICC is itself limited [9]. As with most case reports, selective reporting of a favorable outcome may also introduce bias.

CONCLUSION

Pediatric Trimeresurus envenomation can progress within days to severe VICC with unmeasurable clotting times. Early recognition of empirical treatment failure and species-appropriate antivenom in adequate dose with serial reassessment were the key elements of management, and delayed antivenom remained effective while systemic envenomation was active.

Ethical approval and consent Written informed consent for publication of this report and accompanying images was obtained from the patient's father on 13 September 2026. This single-patient retrospective case report used only routine clinical data, and no separate ethical committee approval was obtained. The reporting physician practiced at Dr. Saiful Anwar General Hospital under Surat Izin Praktik Dokter No. 440.03/0296/35.73.406/2023, issued on 9 March 2023 by the Dinas Tenaga Kerja, Penanaman Modal dan Pelayanan Terpadu Satu Pintu, Malang City.

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Conflict of Interest The authors declare no conflict of interest.

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