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Case report

Severe venom-induced consumption coagulopathy, snakebite-associated thrombotic microangiopathy, and local necrosis following Western bush viper (*Atheris chlorechis*) envenoming in France

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ABSTRACT

Background: *Atheris* spp. are small African vipers whose bites are rarely reported but can result in significant envenoming. There is no specific antivenom available. We describe a case of systemic *A. chlorechis* envenoming presenting with venom-induced consumption coagulopathy (VICC), snakebite-associated thrombotic microangiopathy (TMA), and local necrosis.

Case presentation: A 33-year-old man was bitten on the index finger by a captive *A. chlorechis*. Within 6 hours, early laboratory abnormalities progressed to VICC with markedly prolonged PT/aPTT, undetectable fibrinogen, elevated fibrin monomers, and factor V deficiency. Despite the administration of four vials of Inoserp™ Pan-Africa and repeated transfusions of fibrinogen and fresh frozen plasma, the patient continued to exhibit signs of coagulopathy for 48 hours. Thrombocytopenia, anaemia, schistocytes, and hyperbilirubinemia indicated snakebite-associated TMA, which resolved spontaneously without renal involvement. Progressive local necrosis developed on the finger and dorsal hand, ultimately requiring amputation of the proximal phalanx and surgical debridement.

Conclusion: This case demonstrates that *A. chlorechis* envenoming can produce both local and systemic toxicity. The absence of clinical improvement after Inoserp™ Pan-Africa is consistent with preclinical data showing limited cross-neutralization against *Atheris* venoms. Administration of clotting factors in the presence of unneutralized procoagulant toxins may have contributed to the development of TMA. Therefore, fresh frozen plasma and fibrinogen should be reserved for cases of coagulopathy with active bleeding or when an invasive procedure is being considered, particulary in the absence of a concomitant effective antivenom. The local necrosis highlights the potential for significant local sequelae, necessitating cautious but timely surgical intervention.

1. Background

Atheris species are small and usually arboreal vipers (Stephen and Bill, 2020). Their bites are rarely reported in their native range in sub-Saharan Africa (Chippaux et al., 2024). While they are usually regarded as having limited medical significance, several cases of severe bites to herpetologists and snake keepers have been documented (Hatten et al., 2013; Imperato et al., 2022; Mebs et al., 1998; Ontiveros et al.,

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2022; Robinson et al., 2004; Top et al., 2006; Valenta et al., 2021). A specific antivenom is not commercially available. We report a case of captive *A. chlorechis* envenoming, which resulted in the development of venom-induced consumption coagulopathy (VICC), snakebite-associated thrombotic microangiopathy (TMA), and local necrosis necessitating amputation, despite the administration of several vials of Inoserp™ Pan-Africa (Inosan Biopharma, Madrid, Spain).

Case presentation

A 33-year-old man was bitten by an adult female *A. chlorechis* (a 55cm specimen born in captivity) on the second phalanx of his right index finger while handling the snake during routine care (Fig. 1). The patient was an amateur snake breeder who kept several venomous species at home. He had no significant medical history, including no previous snakebite, and was not taking any long-term medications.

Prehospital management was initiated by a mobile emergency and resuscitation service. At the same time, the regional poison control center was alerted and contacted the antivenom bank. This system, funded by venomous snake breeders, provides access to antivenoms for non-native species stored in several hospitals throughout metropolitan France.

The patient was admitted to the intensive care unit 1.5 hours after being bitten. On arrival, his vital signs were as follows: temperature 37.5 °C, blood pressure 145/39 mmHg, heart rate 82 beats per minute (bpm), respiratory rate, 32 breaths per minute, and oxygen saturation 99% on room air. He complained of severe local pain which required morphine administration. Physical examination revealed a single puncture wound, indicating that the snake had struck with only one fang. Marked edema extended to the wrist, and a hematoma progressively developed on both distal phalanges (Fig. 2). No systemic bleeding was observed.

Initial laboratory tests were within normal limits, except for elevated D-dimer levels (Table 1). Empirical antibiotic therapy with amoxicillin–clavulanic acid was initiated. Since no *Atheris*-specific antivenom was available, Inoserp™ Pan Africa - a polyvalent $F(ab')_2$ antivenom targeting major sub-Saharan African species - was requested from the antivenom bank. Four vials were sent to the hospital. Two vials were administered intravenously 6 hours and 15 minutes after the bite.

Despite this initial treatment, laboratory findings 9 hours and 45 minutes after the bite showed VICC, characterized by prothrombin time (PT) $>$ 70 s, activated partial thromboplastin time (aPTT) $>$ 120 s, and fibrinogen $<$0.4 g/L. Factor assays revealed a marked factor V deficiency (27% activity). The fibrin monomer concentration was elevated at 76.17 μg/mL, and euglobin clot lysis time exceeded 30 minutes, indicating the absence of primary fibrinolysis. Two additional vials of Inoserp™ Pan

Fig. 1. Adult female *Atheris chlorechis* involved in the envenoming.

Fig. 2. Right hand 6 hours after the bite, showing hematoma on the index finger (P2–P3).

Table 1
Laboratory parameters following *Atheris chlorechis* envenoming.

Time after envenoming	Reference range	1h30	9h45	15h10	19h45	29h45	30h45	37h05	45h45	69h45	93h45
Leukocytes (G/L)	4-11	6.65	14.26	9.77	9.16	4.66	9.74		11.24	14.42	13.32
Hemoglobin (g/L)	13.4-16.7	145	132	109	116	59	116		119	123	121
Schistocytes (%)	<0.1					1.3	1.9				
Platelets (G/L)	172-398	278	162	93	76	33	68		64	71	114
Prothrombin time (sec)	12-13	13.9	>70	28.8	>70	>70	>70	28.2	27.1	17.7	15.6
Activated partial thromboplastin time (sec)	20-40	28.2	>120	43.9	91.7	>120	59.2	36.7	38.7	34.2	33.9
Factor II (%)	70-120		89	80	87	77	91	101	93	101	98
Factor X (%)	70-120		80	64	75	60	74	94	79	82	80
Factor V (%)	70-120		27	37	30	35	48	99	70	90	104
Fibrinogen (g/L)	2-4	2.56	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	<0.4	0.79	2.33
D-dimers (µg/mL)	<0.5	>20									
Fibrin monomers (µg/mL)	<6		76.17	91.01	68.26	29.24	52.51	101.43	84.29	133.77	>150
Euglobin clot lysis time (hours)	>2		>2						>2		
Total bilirubin (µmol/L)	<21	8.5	36	31.1	40.7	14.6	24.9		18.7	23.5	
Direct bilirubin (µmol/L)	<5		9.8	8.7	10.7	5.4	7.7		6.7	8.7	
Creatinine (µmol/L)	59-104	87	100	87	103	53	95		79	74	
Urea (mmol/L)	2.8-8.1	5.4	5.5	5	5.9	3.1	4.3		3.1	2.6	

Africa were administered 12 hours after the bite, along with 1.5 g of fibrinogen and 2 units of fresh frozen plasma (FFP). Two additional units

of FFP were administered 1 h later. Coagulation parameters partially corrected but subsequently deteriorated again, with fibrinogen remaining undetectable for the first 48 hours. Fibrin monomers persisted at high levels, while repeat euglobin clot lysis time remained within normal range.

At 24 hours, the patient developed a 10-cm hematoma along the medial side of the right arm and ecchymoses on other fingers. Necrosis appeared on the bitten finger and the dorsal aspect of the hand. On day 2, pain intensified despite morphine. There was no sensory loss, but mobility was limited at the proximal interphalangeal joint and absent at the distal joint. Peripheral pulses were palpable, and there were no signs of compartment syndrome; thus, fasciotomy was not performed to avoid exacerbating the damage caused by the venom. The patient did not have a fever during hospitalization. The patient received 3 g of fibrinogen and vitamin K treatment (10 mg four times a day) was administered for 2 days.

Thrombocytopenia and anaemia developed, reaching their nadir approximately 30 hours post-bite. Elevated bilirubin and schistocytes on the blood smear indicated microangiopathic hemolysis. The patient was diagnosed with snakebite-associated TMA, which resolved spontaneously without renal or other organ involvement. Coagulation gradually normalized from 72 hours after the bite. Locally, multiple blisters appeared on the finger and the back of the hand (Fig. 3). On day 6, the patient was transferred to the orthopedic surgery department.

Amputation was initially considered but deferred in hopes of spontaneous improvement. However, over the following days, the necrotic lesions on the finger and the back of the hand failed to improve. Instead, there was progressive mummification of the finger and retraction at the middle phalanx (P2) level (Fig. 4). On day 14, a trans–proximal phalanx (P1) amputation of the right index finger and surgical debridement of the necrotic dorsal tissue were performed. Ten months after the bite, the examination revealed painful and functional sequelae related to the amputation, and aesthetic skin scarring (Fig. 5).

3. Discussion

This case report highlights a complex and severe manifestation of *A. chlorechis* envenoming, combining VICC, snakebite-associated TMA, and local necrosis, which led to finger amputation. *A. chlorechis* venom contains snake venom metalloproteinases (SVMP), phospholipases A_2 (PLA_2) and disintegrins that are involved in these pathological processes (Wang et al., 2018). These venoms have a marked procoagulant action *in vitro*, potentially responsible for the VICC observed in patients (Chowdhury et al., 2022). To date, there is no functional data regarding the activities of *Atheris chlorechis* venom available. *In vitro*, *A. squamigera* venom exhibits hemorrhagic, platelet-aggregating, and fibrinogen-converting activities; however, fibrin(ogen)olytic activity is negligible (Mebs et al., 1998).

VICC is a well-recognized complication of envenomings by many viper species and some elapids. It is characterized by rapid consumption of clotting factors, prolonged clotting times, hypofibrinogenemia, and elevated D-dimers (without the microthrombi typical of disseminated intravascular coagulation) (Isbister, 2010). VICC promotes and maintains bleeding, including at distant sites, which is a common feature of *Atheris* bites (Hatten et al., 2013; Ontiveros et al., 2022; Top et al., 2006; Valenta et al., 2021). In this case, VICC was initiated by an isolated increase in D-dimer and then occurred within 6 hours. A study of exotic Viperidae envenomings (including *A. nitschei* and *A. squamigera*) found that an increase in D-dimers is the first parameter indicating VICC (Valenta et al., 2019). Fibrin monomers are specific markers of fibrin formation and showed higher sensitivity, specificity, positive and negative predictive value compared to D-dimer when differentiating overt disseminated intravascular coagulation (DIC) from non-DIC (Park et al., 2011). Owing to their short-life, fibrin monomers may be more suitable for monitoring consumption coagulopathy (Shainoff and DiBello, 2003). The sustained elevation of this biomarker suggests ongoing coagulation activation for at least the first four days following envenoming. The only previously published case of an *A. chlorechis* bite also reported VICC occurring within 6 hours of the bite (Top et al., 2006). *A. squamigera* venom does not activate purified prothrombin but contains a factor V and calcium-dependent prothrombin activator (Mebs et al., 1998). In the present case, the decrease in factor V activity as well as the slight effect on factors II and X suggests the presence of a similar activator in *A. chlorechis* venom.

Some patients with VICC progress to develop a snakebite-associated TMA, which is defined as the association of thrombocytopenia and

Fig. 3. Right hand 3 days after the bite, showing progression to necrosis of the index finger and the back of the hand, with blister formation.

Fig. 4. Right hand 13 days after the bite, showing a mummified index finger with retraction at the level of the middle phalanx (P2).

microangiopathic hemolytic anemia (MAHA) with ≥1% schistocytes (Noutsos et al., 2022a). Many of these cases involve *Hypnale* spp., *Daboia russelii* and *Pseudechis* spp. (Noutsos et al., 2020). Snakebite-associated TMA has also been reported with *A. chlorechis* (Top et al., 2006) and *A. nitschei* (Hatten et al., 2013). In the published case involving *A. chlorechis*, an acute kidney injury was present. However, we did not observe renal injury in our patient, suggesting that TMA in this case was an isolated hematologic phenomenon. The pathophysiology of snakebite-associated TMA is unclear, but MAHA may be partially due to activation of the coagulation system, as observed in disseminated intravascular coagulopathy (Heyes et al., 1976; Lesesve et al., 2014). ADAMTS13 levels were not measured in our patient. However, in the few published cases where this analysis was performed, ADAMTS13 levels were always within the normal range (Noutsos et al., 2020).

The lack of a specific antivenom results in *Atheris* envenomings being treated primarily with symptomatic therapy (Hatten et al., 2013; Imperato et al., 2022; Mebs et al., 1998). However, the risk of severe symptoms justifies trying non-specific antivenoms in hopes of a para-specific effect. Four vials of Inoserp™ Pan-Africa were used, though it is not specific for *Atheris*. In an *in vitro* study, none of the antivenoms in a panel of products intended for sub-Saharan Africa (including Inoserp™ Pan-Africa) were able to neutralize the procoagulant effect of *Atheris* venoms, especially *A. chlorechis* (Chowdhury et al., 2022). The lack of preclinical efficacy of Inoserp™ Pan-Africa against *Atheris* venoms is consistent with a published case of an *A. squamigera* bite (Ontiveros et al., 2022). Despite the administration of 8 vials of Inoserp™ Pan-Africa, followed by 10 vials of Antivipmyn® Tri, and 10 vials of Anavip®, the latter 2 of which are antivenoms designed for American pitvipers, the patient experienced progressive worsening of locoregional edema and bruising, as well as thrombocytopenia and fibrinogen consumption, during the first 9 h. Coagulopathy began to improve from the 17th hour after the bite. In the previously published *A. chlorechis* bite case, 6 vials of FAV-Afrique (Sanofi-Pasteur, France) combined with a FFP transfusion corrected the bleeding and VICC within a few hours (Top et al., 2006). Unfortunately, this antivenom has been discontinued, and there is no preclinical data to confirm its efficacy. The case reported here confirms the ineffectiveness of Inoserp™ Pan-Africa in treating envenoming by *A. chlorechis*.

Fig. 5. Right hand 9 months after the bite, showing postoperative appearance of the stump after trans-P1 index finger amputation.

The transient correction of coagulation parameters was likely due to the transfusion of FFP and fibrinogen before the circulating toxins consumed them. In the absence of an effective antivenom against *Atheris* venom, transfusion of clotting factors has been the only available treatment for bleeding proposed in previous reports of *Atheris* envenomings. In this case, the use of FFP and fibrinogen was motivated by the lack of effective antivenom in the presence of VICC with hemorrhagic manifestations (haematomas initially in the hand and subsequently in the arm). However, administration of FFP could also have been deferred given the absence of systemic bleeding, with close clinical and biological monitoring maintained. The persistence of unneutralized procoagulant toxins in combination with FFP administration could be the cause of TMA. The Australian Snakebite Project cohort study showed that initial treatment with FFP infusion was administered more frequently in cases of VICC that subsequently progressed to TMA than in VICC cases that did not (Noutsos et al., 2022b). Notably, the two published cases of *Atheris* envenoming complicated by TMA also received FFP (Top et al., 2006; Hatten et al., 2013). Therefore, FFP should only be administered in cases of active bleeding or when an invasive procedure is being considered, particulary in the absence of a concomitant effective antivenom.

Some of the treatments administered may not have been necessary. Routine antibiotic prophylaxis is not recommended for snakebites (Moody et al., 2025), and amoxicillin–clavulanic acid is unlikely to be effective in cases of infection (Sachett et al., 2017). Antibiotic therapy was initiated by the intensivists during the patient's initial management. When clinical toxicologists were consulted, they did not recommend continuation of this treatment; however, it was maintained. Similarly, vitamin K has no established benefit in the management of VICC. The intensivists decided to administer vitamin K in combination with fibrinogen. This approach had not been validated by the clinical toxicologists, who became aware of it only upon reviewing the medical record.

Local tissue damage from a snakebite may result from the action of direct cytotoxic SVMP and PLA_2. Severe coagulation disturbance and microvascular injury may lead to microthrombi formation and ischemia, which can exacerbate necrosis. Our patient's substantial necrosis requiring amputation highlights the potential for severe local sequelae. Although many snakebite case reports describe ulceration, blistering, or soft-tissue necrosis, full amputation is uncommon in documented *Atheris* cases (Robinson et al., 2004; Waiddyanatha et al., 2019). This case suggests that clinicians must be vigilant for progressive necrosis even when managing systemic coagulopathy. The decision to perform surgery should always be taken with caution in cases of snake bites. Premature surgery can lead to increased bleeding that is more difficult to manage in the event of concomitant coagulopathy (Top et al., 2006). In cases of dry necrosis (i.e., without associated infection), it is advisable to wait several days for the necrosis to become defined before amputation, to

preserve as much healthy tissue as possible (Tanda et al., 2024). However, waiting too long can expose the patient to the risk of local infection or even sepsis (Latz et al., 2022).

4. Conclusion

This case demonstrates that *Atheris chlorechis* envenoming, which is traditionally considered of limited medical significance, can lead to severe and multi-faceted complications. The combination of early VICC, snakebite-associated TMA, and extensive local tissue necrosis progressing to amputation highlights the potential severity of bites from this genus, particularly in the absence of an effective antivenom. TMA may be iatrogenic, resulting from the administration of clotting factors in the absence of prior neutralization of procoagulant toxins. Clinicians should anticipate rapid coagulation disturbances, monitor closely for hematologic complications, and remain vigilant for progressive local necrosis. Optimal management requires early recognition, coagulation support only when necessary, and coordinated multidisciplinary care, including delayed but timely surgical intervention.

CRediT authorship contribution statement

Sébastien Larréché: Writing – original draft, Visualization, Investigation, Conceptualization. **Gael Le Roux:** Writing – review & editing, Resources, Investigation. **Jean-Philippe Chippaux:** Writing – review & editing, Investigation. **Jérémy Hardy:** Writing – review & editing, Resources. **Anne-Laure Fédou:** Writing – review & editing, Resources. **Magali Labadie:** Writing – review & editing, Supervision, Resources, Investigation.

Ethics statement

Written informed consent has been obtained from the patient to publish this case report.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work the authors used ChatGPT (OpenAI) in order to improve the language and readability of the manuscript. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: JPC has been the principal investigator or coordinator of several clinical trials involving antivenoms from different laboratories (Pasteur Mérieux Serums & Vaccines, Sanofi Pasteur, Bioclon, and Inosan Biopharma), without receiving any remuneration or compensation from the manufacturers. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

Data will be made available on request.

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