African spitting cobra (*Naja nigricollis*) bite of the hand

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WM Kuzon, JR Marcus, LD Kerluke, JH Phillips. African spitting cobra (*Naja nigricollis*) bite of the hand. Can J Plast Surg 1994;2(2):90-92. Fifty thousand snakebites occur annually in North America. Of these, about 8000 result in envenomation by poisonous species. In North America, 98% of poisonous snakebites are caused by pit vipers (Crotalidae). This report describes a *Naja nigricollis* (family Elapidae, the African spitting cobra) bite of the hand that occurred in Ontario. This species is common in West Africa and is popular with North American collectors. Its venom is quite different from the venoms of pit vipers and results in significant tissue necrosis. The management of a bite by *N nigricollis* requires aggressive medical and surgical treatment.

Morsure de la main par un serpent à cou noir (*Naja nigricollis*)


In North America, approximately 98% of venomous snake bites are inflicted by Crotalidae – the pit vipers. Included in this group are the cottontooths, copperheads, and massasauga rattlesnakes (1). Whereas most snakebites worldwide are inflicted on the lower extremities of farmers and hunters of the rural tropics, in the countries of North America, many occur on the upper extremity and hand. It is estimated that 21% of these snakebites occur on the digits of the hand and 25% result from the snake being handled (1,2). This report describes a Canadian patient who was bitten on the hand by an African spitting cobra (*Naja nigricollis*).

**CASE REPORT**

A 27-year-old male, previously healthy animal health technician presented to the emergency room at Sunnybrook Health Sciences Centre in Toronto following a snakebite to his right hand. A collector of many rare snakes, he was bitten while handling a 180 cm long *N nigricollis* in his home. His symptoms included severe local pain within minutes of the bite, jaw stiffness, and abdominal tenderness 3h later. He described no neurologic symptoms. On examination, there were visible fang marks on the right ring finger (Figure 1). A small scratch was noted on the right long finger, and a small, innocuous puncture was noted on the right thigh. The right ring finger was edematous but inflammation was not prominent. The ring finger demonstrated diminished moving two-point discrimination (10 mm); sensation on the remainder of the hand was normal. His neurologic exam was otherwise within normal limits. Range of motion in the ring finger interphalangeal joints was diminished due to edema; motion in the remainder of the hand was normal.

Laboratory investigations revealed a slightly elevated partial thromboplastin time (55 s); complete blood count (CBC),

Figure 1) Fang marks on the right ring finger 8 h after injury
electrolyte, blood urea nitrogen (BUN), creatinine and prothrombin time determinations were within the normal range. In the emergency room, he was treated with intravenous (iv) fluids, was administered oxygen, and received ce
cfazolin (continued 1g iv q8h). Following a test dose, seven vials of antivenom (obtained from the Toronto Zoo) were given. The hand was splinted and kept horizontal.

The following day, the ring fingertip was congested and the fingernail was removed. The nailbed was abraded, and heparin soaked gauze was applied continuously. Blistering appeared and local tissue destruction became evident over the following three days with worsening inflammation and edema. Cefazolin was continued, and antimicrobial coverage was expanded by the addition of metronidazole (500 mg iv q8h) and tobramycin (100 mg iv q8h) to the regimen.

By day 5, the ring finger had entirely necrosed (Figure 2). Surgical debridement of the ring finger was performed resulting in amputation at the mid proximal phalanx level. The wound was left open and dressed. Intraoperative cultures grew Morganella and Enterococcus species. The local reaction gradually subsided. Delayed primary closure was performed on day 7, and the patient was discharged to home one week later. The small wounds on the long finger and the right thigh healed spontaneously without significant tissue necrosis. The function of the hand and remaining digits returned to normal.

DISCUSSION

With regard to the site of attack and provocation, this case is an illustration of the typical presentation of a snake bite in North America; however, the offender is far from familiar. Despite being widely distributed in Africa, the spitting cobra (N nigriceps) is not indigenous to any area of North America. It is the most common African cobra, usually coloured black or brown with several light coloured bands on its throat. It can grow up to 220 cm long, although the average length is reported to be 120 cm (2). It is well known for its ability to spit venom with remarkable accuracy into the eyes of its attackers.

Snake venom may contain up to 20 components with various enzymes as the most important constituents (2). The major categories of enzymatic components are: neurotoxins, cardiotoxins, hemotoxins, venotoxins and necrotizing factors. The venom of North American pit vipers is known to cause severe pain, rapid swelling and ecchymosis with local necrosis and sloughing in the area of the fang marks (1,3). With severe envenomation, systemic effects such as coagulation abnormalities, disseminated intravascular coagulation (DIC), pulmonary edema and shock may occur. The venom of N nigriceps causes a different spectrum of toxicity. In the largest published review of N nigriceps snake bites (14) by DA Warrell in Nigeria, the principal physiologic manifestations of the poisoning were local swelling, necrosis and hematologic abnormalities (2). The swelling, dramatic and often massive, is related to the venom’s phospholipase A2 isozymes that cause increased vascular permeability. Tissue necrosis is the most common serious effect of N nigriceps envenomation, and the cytolytic effects of several venom constituents are well characterized. It is this extreme cytotoxicity and often catastrophic local tissue destruction that distinguishes the venom of N nigriceps from all other snakes, including most other species of cobra whose most classic manifestations of poisoning relate to neurotoxicity. The snake’s fangs are relatively short (approximately 3 mm); therefore the extent of necrosis, which is partially dependent upon depth and compartmental invasion, is often superficial. However, further penetration and deeper compartmental destruction is possible. According to the experience in Nigeria, if a bite is neglected, extensive necrosis and secondary infection can develop and then often the entire affected limb must be amputated (2).

The treatment of any snake bite can be broken down into two phases – pre-hospital and hospital. In the field, the patient should be comforted and kept quiet. The snake should be killed and brought with the patient for identification. Patients should be transported on their side to prevent aspiration of vomit. The bitten limb is immobilized by splint or sling and elevated to the level of the heart. Although it may increase the risk of local infection, incision of the fang mark and the application of Sawyer suction cups have been shown to remove up to 50% of controlled injected venom if done up to 30 mins after the bite (1,4). A tourniquet should not be applied to the extremity in cases of N nigriceps bite; because of its severe local toxicity, regional compartmentalization may intensify the local injury. Finally, ice and cooling should not be used in any snake bite, particularly that of N nigriceps, as it has been shown that cooling potentiates the local necrotic effect.

Once the patient is stabilized, having reached the hospital identification of the snake is important. In this case, N nigriceps was identified by the owner, and its identification dramatically changed the pathologic and therapeutic expectations. The patient’s symptoms and signs are carefully evaluated. Laboratory investigations should include CBC with platelet count, prothrombin time, partial thromboplastin time, electrolytes, BUN, creatinine, and type and cross-match. Because of the potential for secondary infection, broad spectrum
antibiotic coverage should be initiated. The oral flora of *N. nigricollis* is known to contain a number of pathogens, including *Enterococcus* species. In addition, the circumference of the extremity should be measured serially documenting proximal migration and progression.

The decision to give antivenom is based upon a set of general indications for its use. Among these are impaired consciousness, hypotension and shock, coagulopathy, evidence of DIC, and evidence of renal failure (1). In the case of the African spitting cobra, any local reaction is an indication for the use of antivenom in light of the potentially disastrous consequences. Once the tissues have become necrotic, the recommended treatment is early and extensive debridement. Blisters should be debrided as they are invariably followed by necrosis in cases of cobra bites (2).

CONCLUSION

Although 98% of snakebites in North America are caused by pit vipers, circumstances can arise in which the accepted treatment performed in typical cases of indigenous snake bite are not applicable. *N. nigricollis*, common in Nigeria and other parts of Africa, is known for its extremely cytotoxic venom. Among North American collectors, it is a popular species. As well illustrated by our patient, the local effects of *N. nigricollis* envenomation are early edema, inflammation and blistering followed by frank tissue necrosis. As tissue destruction progresses, the extremity can become compromised and amputation may be necessary. As with all snake bites, management of *N. nigricollis* envenomation requires early and aggressive medical and surgical intervention.

REFERENCES


CALENDAR OF EVENTS

SEPTEMBER 19-20, 1994
Advanced courses in plastic surgery, skin cancer including melanoma and sarcomas, and malformations of the skin
Royal Marsden Hospital, London, UK
Contact British Association of Plastic Surgeons, 35-43 Lincoln’s Inn Fields, London WC2A 3PN, UK

SEPTEMBER 22-26, 1994
15th World Congress of Dermatologic Surgery
Toronto, Ontario
Contact International Society for Dermatologic Surgery, 930 North Meacham Road, Schaumburg, Illinois 60173-6016, USA. Telephone (708) 330-9830, Fax (708) 330-0050

SEPTEMBER 24-28, 1994
ASPRS/PSEF/ASMS Annual Scientific Meeting
San Diego, California, USA
Contact ASPRS/PSEF Products and Meetings Information, 444 East Algonquin Road, Arlington Heights, Illinois 60005, USA. Telephone 1-800-766-4955

OCTOBER 13-15 1994
Northeastern Society of Plastic Surgeons 11th Annual Meeting
Le Chateau Frontenac, Quebec City, Quebec
Contact Catherine M Beinhauer, 45 Lyme Road, Hanover, New Hampshire 03755, USA. Telephone (603) 643-2325

OCTOBER 15-16, 1994
Twenty-first annual meeting of the Canadian Society for Aesthetic (Cosmetic) Plastic Surgery
Toronto, Ontario (Four Seasons Yorkville)
Contact Mrs P Hewitt, Canadian Society for Aesthetic (Cosmetic) Plastic Surgery, 4650 Highway #7, Woodbridge, Ontario L4L 1S7. Telephone (905) 831-7750.

NOVEMBER 17-19, 1994
Facelifting symposium
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Contact Francine Leinhardt, 116 East 63rd Street, New York, NY 10021, USA. Telephone (212) 838-9200.

DECEMBER 10-15, 1994
Rhinoplasty course
Key Biscayne, Florida, USA
Contact Dr Robert L. Simons, 16800 NW Second Avenue, suite 607, North Miami Beach, Florida 33169, USA. Telephone (305) 651-9903.

MARCH 23-25, 1995
Symposium on Aesthetic Surgery of the Face
Davis Medical Center, San Francisco, California, USA
Contact Sara Burke. Telephone (415) 476-4251