Clinical manifestations of Cape cobra (Naja nivea) bites

A report of 2 cases

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Summary

Two cases of proven bites by the Cape cobra (Naja nivea) are reported. Both patients developed areflexic flaccid paralysis and required ventilation from 2/4 and 2 1/2 hours after the bite for 4 1/2 and 7 days respectively. Antivenom is of value before the onset of paralysis but of no value once paralysis is established. Both patients developed a non-depolarizing neuromuscular block which was little affected by prostigmine.

Early treatment in identified Cape cobra bites should be the immediate application of an arterial tourniquet, followed by intravenous administration of 80 - 100 ml polyvalent antivenom if signs of neurotoxicity develop. This may need to be repeated. Treatment once paralysis is established is largely supportive, prolonged ventilation frequently being required, following which recovery should be complete.

The Cape cobra (Naja nivea) is one of the most common poisonous snakes in the Cape Province. It is also found in the south-western Transvaal, the Orange Free State, the southern half of SWA/Namibia and southern Botswana. In spite of this, there exist only a few brief reports on proven bites by this snake.1-4 This study reports the clinical manifestations and management of 2 cases of accurately documented Cape cobra bites. One snake was killed and identified by a herpetologist, while the other bit a snake-catcher familiar with the Cape cobra.

Case reports

Case 1

A 53-year-old coloured man was bitten on his right index finger at 20h00 on 9 March 1984 in Darling, CP. Within 5 minutes a tight cloth tourniquet was applied to the arm and the hand began to swell. At 10 minutes his mouth was dry and he began sweating, and a little later he was shaking. At 30 minutes his arm was weak and he had ptosis.

At 1 hour the patient was seen by a doctor, who noted that he was acting as if drunk. Polyvalent SAIMR antivenom (10 ml) was given intravenously and the tourniquet was removed. At 2 1/4 hours he was fasciculating and sweating and had inadequate respiration. A gag reflex was present. Ventilation was assisted by an oral airway, facemask and manual resuscitator.

En route to hospital he had a generalized convulsion but was not incontinent and did not hurt himself. He arrived at Groote Schuur Hospital within 4 hours, where it was noted that he had complete flaccid paralysis. He later claimed that at this stage he had been able to hear and understand but could not move. The pulse rate was 90/min, and the blood pressure 120/70 mmHg; the pupils were normal but a divergent squint was present, and there were coarse crackles at both lung bases.

Before intubation, the following blood chemical results were obtained: pH 6.78, partial arterial oxygen pressure 23 kPa, partial arterial carbon dioxide pressure 23 kPa, and base deficit -13.5. Urea, glucose and electrolyte values were normal. At 6 1/2 hours the acid-base status and blood gas values were normal. Polyvalent antivenom (90 ml) was given intravenously at 7 and 17 hours, with no apparent response. The clotting time on two occasions was normal.

On day 2 at 20 1/2 hours the patient had complete flaccid paralysis with no response to stimuli and no corneal, pharyngeal, plantar or tendon reflexes. The haemoglobin concentration was 13.5 g/dl, the white cell count 14,6 x 10⁹/l, and the platelet count 185 x 10⁹/l. The prothrombin index was 87%, the partial thromboplastin time 40 seconds (control 34 seconds), the fibrinogen value 4.29 g/l, and the fibrin degradation product (FDP) value 80 - 160 mg/l. Urea, glucose and electrolyte values and the results of liver function tests were normal. At 20 hours 5 mg prostigmine plus 1.2 mg atropine was given intravenously. Peripheral stimulation of the ulnar nerve showed a change from no response to that of a post-tetanic facilitation. No pre-tetanic twitches could be elicited. The ECG was normal.

On day 3 the patient moved his tongue and attempted to cough when suctioned. Later that day he opened his eyes and moved his left leg. There was minimal respiratory effort. Nerve stimulation was repeated at 40 hours. There was a 3/4 response to a train of four impulses, with post-tetanic facilitation. After administration of prostigmine 2.5 mg there was a dramatic increase in twitch size, good tetanic movement and an improvement in motor response to command, which persisted for some hours. The response was insufficient to allow discontinuation of ventilation. The CSF was normal. The prothrombin index, platelet count and partial thromboplastin time were normal, the fibrinogen value was 9.01 g/l (normal 2 - 4 g/l) and the FDP value was 40 - 80 mg/l.

On day 4 ptosis was still present, but there was a slight improvement in motor function; the latter had improved still.
further by day 5. On day 6 the vital capacity had increased to 1500 ml and the patient was capable of weak movement of the arms and legs. He was extubated 108 hours after intubation.

Thereafter he continued to improve. At no stage did he show any evidence of coagulopathy, but the course was complicated by a chest infection (day 5) which responded well to antibiotics. This was associated with a minor transient rise in bilirubin, transaminase and alkaline phosphatase values. A small abscess on the bitten finger was drained and culture grew normal skin flora. He was discharged on day 13, almost completely recovered.

Case 2
A 26-year-old black man was bitten on the right thumb while playing with a snake at Aggenays, CP, on 19 January 1984. He was a snake-catcher and knew that it was a Cape cobra.

Early symptoms and signs were not recorded. He arrived at Aggenays Hospital 2 hours after the bite and was given 40 ml polyvalent antivenom, half intravenously and half intramuscularly. At 2½ hours he apparently became unconscious and paralysed. He was intubated and ventilated with a manual resuscitator.

At 4½ hours a further 40 ml antivenom was administered intravenously. This resulted in improvement; the patient was able to open his eyes and move his limbs, but was still weak.

He arrived at Groote Schuur Hospital 10½ hours after being bitten and then had a flaccid paralysis with no reflexes. His pupils were normal, as were blood gas values. At 11½ hours 60 ml polyvalent antivenom and 5 mg prostigmine had no effect. Ventilatory support was continued using constant positive-pressure ventilation by a volume ventilator.

On day 2 the patient's level of consciousness improved and there was some movement of his hands and head. On day 3 there was further improvement, and he was able to communicate, open his eyes, and move his head and shoulders. The bitten hand was swollen.

On day 5 a further 40 ml antivenom was given. This produced rigors and tachycardia and had no beneficial effect.

The patient showed gradual improvement in muscular power and was extubated on day 8, a full 7 days after ventilation commenced. He walked with help on day 9 and was discharged on day 15.

Discussion
South African cobra and mamba (N. nivea, N. haje, N. melanoleuca, Dendroaspis polylepis, D. angusticeps) venom, which produces paralysis, is thought to do so because it contains a postsynaptic neurotoxin. In keeping with this, nerve stimulation after administration of prostigmine showed a non-depolarizing block of the curare type. However, prostigmine was unable to reverse this block completely, even in repeated doses.

Before the onset of paralysis (1 hour after being bitten) patient 1 received 10 ml polyvalent antivenom. While completely paralysed he received two further 90 ml doses at 7 and 17 hours with no response. Patient 2 was given 20 ml antivenom intramuscularly and 20 ml intravenously before the onset of paralysis (at 2 hours). Two hours after intubation intravenous administration of 40 ml produced definite improvement. Once he was completely paralysed further doses of 60 ml at 11½ hours and 40 ml on day 5 had no effect.

Failure of reversal with both prostigmine and antivenom suggests that the toxin is fixed, and that reversal only occurs once it has been naturally degraded. Our patients required mechanical ventilation until the 6th and 8th day respectively.

After bites by the Cape cobra, antivenom is probably of no value once paralysis is well established. After bites by the black mamba (D. polylepis), however, reversal of established paralysis with antivenom begins within minutes and continues unabated provided adequate quantities of antivenom are given.

After Cape cobra bites, antivenom is of value before the stage of established paralysis, as shown in case 2. Grasset showed that sheep injected with 10 mg N. nivea venom were saved with 7 ml antivenom at 30 minutes, but that 40 ml was required if given at 2½ hours. The initial doses of antivenom given in these two cases before complete paralysis were too small. Once it is evident that the patient is becoming weak, a dose of 80 - 100 ml intravenously would be appropriate. This should be repeated if improvement ceases or weakness recurs. It is noteworthy that the pupils of both patients were normal throughout; this is contrary to the opinion that widely dilated pupils are characteristic of muscle paralysis produced by the Cape cobra.

Coagulopathy did not develop. Lymphadenopathy was not noted, but has been reported to occur.

The polyvalent antivenom produced by the SAIRM is recognized as being of excellent quality and is effective against all lethal South African snakes other than the two which produce coagulopathy, namely the boomslang (Dispholidus typus) and the vine snake (Thelotornis spp). A monovalent antivenom exists for the former, but there is no antivenom for the latter. As the Cape cobra is the only snake in the southwestern Cape capable of producing severe neurotoxic paralysis, a monovalent serum would perhaps be of value for use in this area.

Prostigmine appears to be of no benefit in reversing paralysis after Cape cobra bites.

Endotracheal intubation should be performed immediately if necessary and intermittent positive-pressure ventilation should be continued. If no facilities exist for intubation, mouth-to-mouth breathing should be instituted and continued until intubation can be performed. The patient should then be transferred to a respiratory intensive care unit, since ventilatory support may be necessary for up to 7 days. Provided anoxia can be avoided, all victims should survive.

The application of an arterial tourniquet immediately after a bite known to be by a Cape cobra is of proven value. Application of the pressure immobilization technique as a first-aid measure, as described by Sutherland, is of value after bites by Australian elapids, Indian cobras and Eastern diamond-back rattlesnakes. Provided anoxia can be avoided, all victims should survive.

REFERENCES
Summary

A patient with Parkinson's disease developed a non-ketotic hyperosmolar diabetic coma precipitated by chest infection. Initial improvement from treatment with intravenous insulin, ampicillin and fluid therapies was followed by severe deterioration and hypovolaemic shock. Further improvement occurred only when therapy directed against Gram-negative sepsis was added. A barium examination later demonstrated aspiration of oral contents with pulmonary soilage. The differences between the easily recognized early fulminating 'aspiration syndrome' caused by aspiration of gastric contents of low pH and the aspiration of oral contents, which may remain occult for many hours, is highlighted. Life-threatening Gram-negative or anaerobic infection may then occur but remain undiagnosed because the original aspiration of foreign material is unsuspected.

Aspiration of oral contents in Parkinson's disease

A case report

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Hippocrates warned in 400 B.C. that 'for drinking to provoke a slight cough or for swallowing to be forced is bad'. However, the full danger of aspiration of gastric contents with a low pH was not recognized until 1946. Severe acute illness is produced with hypovolaemic collapse, bronchospasm, cyanosis and hypoxia leading to death in 30% of cases. Unconsciousness from any cause is the main predisposing factor, but aspiration of gastric contents also occurs with neurological disorders including extrapyramidal disease. Oropharyngeal dysphagia due to neurological deficit may lead to aspiration of gastric contents but the aspirate is more likely to be of oral origin. The degree of acidity is the main damaging agent in the 'aspiration syndrome', hence the early fulminating illness is unlikely to occur if the aspirate is of oral origin since this is not at a low pH. Instead, bronchopulmonary infection with mixed flora, Gram-negative bacteria and anaerobes causes pneumonia, bronchiectasis, abscess and empyema. Anaerobic infection dominates. A severely ill patient is described in whom pneumonia improved only with antimicrobial therapy directed against Gram-negative and anaerobic infection when underlying aspiration of oral contents was recognized.

Case report

A 49-year-old man, diagnosed 8 years previously as having Parkinson's disease, was admitted to hospital with a 3-day history of progressive thirst and breathlessness. Maturity-onset diabetes had been diagnosed when myocardial infarction occurred 1 year previously. Fluctuating speech and swallowing difficulty with a weight loss of about 13 kg in 2 years had occurred despite levodopa/carbidopa therapy. Fluctuating speech and swallowing difficulty with a weight loss of about 13 kg in 2 years had occurred despite levodopa/carbidopa therapy.

Examination revealed an ill patient with a soft voice, temperature 39°C, pulse 110/min, blood pressure 120/80 mmHg, tachypnoea and basal crepitations. Urinalysis demonstrated glycosuria without ketonuria. Investigations showed the following: serum sodium 156 mmol/l, potassium 3.0 mmol/l, urea 25 mmol/l, glucose 45 mmol/l, haemoglobin 16.1 g/dl and leucocytes 10.8 x 10⁹/l. Blood gas measurements were: partial arterial oxygen pressure (Po₂) 4.8 kPa, partial arterial carbon dioxide pressure (Pco₂) 3.7 kPa, pH 7.44. A chest radiograph demonstrated bilateral basal pneumonia. Non-ketotic hyperosmolar diabetic pre-coma precipitated by pneumonia was diagnosed and treatment started with intravenous insulin, fluids, heparin 40 000 U daily, ampicillin 500 mg 6-hourly and oxygen via mask. There was initial improvement, but 24 hours after admission there was a sudden deterioration and the patient became moribund, with a