

CASE REPORT

Use of neostigmine in black mamba snake bite: a case report

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ABSTRACT

Snake bite is quite common in most of the countries of the world, especially tropics and subtropical areas. The snake venom is usually one of the two types; neurotoxins and hemotoxins or hemolysins. The management of snake bite consists of supportive treatment and anti-snake venom serum. The patients suffering from respiratory problems due to muscular paralysis will require mechanical ventilatory support. We present a case of a victim of black mamba bite, which is one of the most dreaded snakes in Africa. His recovery was slow and marred with coagulation profile derangement. Finally we started neostigmine and atropine and witnessed a dramatic improvement in his muscle power. He rapidly improved and was discharged with complete recovery.

Key words: Snake Venoms; Venoms; Biological toxins; Snake Bites; Neurotoxins; Neostigmine

Citation: Shah SARA, Naqvi SS, Abbas MA. Use of neostigmine in black mamba snake bite: a case report. *Anaesth Pain & Intensive Care* 2016;20(1):77-79

INTRODUCTION

Black mamba (*Dendroaspis polylepis*) is one of the fastest and deadliest snakes on land, having a maximum speed of 12 miles/hour and 100% mortality without treatment. It is basically olive to grey in color, and is called black mamba due to black colored mouth cavity. Transporting the snake bite victim to medical facility promptly is very important as he will collapse within 45 minutes. The clinical representation can vary markedly due to the fact that its venom contains neurotoxins, cardiotoxins and fasciculins. The symptoms range from profuse sweating, salivation, ptosis, bulbar palsy to respiratory arrest and collapse. Early management of the victim can save his life. We present a case of black mamba bite who was brought to our hospital. He required ventilatory support and anti-snake venom, but it was the use of neostigmine that helped him recover the most.

CASE REPORT

A 31 years old man reported to us with half an hour history

of black mamba bite at left shin. At the time of arrival, he had no history of diplopia, dysphagia or bleeding. He was conscious, oriented and hemodynamically stable. There was swelling on his left shin. He was admitted to ICU, and was treated with 100 ml of anti-snake venom injection. Tetanus prophylaxis was administered along with inj ceftriaxone 1 gm BID. Whole blood clotting time (CT) was done, and it failed to clot. So inj vitamin K 10 mg once daily was started.

After 1½ hour, patient became restless, drowsy and developed ptosis. His hemodynamic parameters were stable. He was placed on ventilator using S-CMV mode, with tidal volume of 600 ml, respiratory rate of 12/min, PEEP of 5 cmH₂O and FiO₂ of 0.5. Propofol 100 mg and nalbuphine 5 mg were given IV, and patient was intubated. Propofol infusion was started at a rate of 5 mg/hr, and nalbuphine infusion was started at a rate of 2 mg/hr.

Repeat CT after six hours was 40 min. Complete blood

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count (CBC), urinalysis, serum urea and creatinine, and LFT's were within normal limits. PT and PTTK were 16/14 and 37/34 respectively. 100 ml of anti-snake venom was administered.

After 12 hours of ventilation, there was some improvement in the muscle power of patient (3/5 in all limbs), so a trial of weaning was given. Propofol and nalbuphine infusions were stopped. Patient was placed on PSV (pressure support ventilation) with pressure support of 15 cmH₂O, PEEP of 5 cmH₂O, and FiO₂ 0.4. Pressure support was decreased to 10 cmH₂O after one hour. However, the trial was not successful and the patient became lethargic. Therefore, ventilator mode was changed to adaptive support ventilation (ASV) with minute volume of 100%, FiO₂ of 0.4, PEEP of 5 cmH₂O, and propofol infusion was restarted. Blood CT was measured again, which was still 40 min, with PT 16/14 and PTTK 36/34. D dimers were > 400 < 600. Hb was 12.6 gm/dl, TLC 9.1 X 10⁹/L and platelet count 110 X 10⁹/L. Laboratory tests also showed an increase in CK (3049 U/L), LDH (636 U/L). 100 ml of anti-snake venom was repeated. Inj furosemide 40 mg BD was started. Antibiotic coverage was changed to inj Tazocin 4/5 gm TDS and vancomycin 1 gm BID.

During next 24 hours patient remained on ventilator, his blood CT decreased to 25 min, Hb decreased to 11.8 gm/dl, platelets decreased to 72 X 10⁹/L and TLC raised to 10.5 X 10⁹/L. PT was 18/14 and PTTK 40/34. D dimers were > 400 < 800. But later on CT increased to 50 min, PT 20/14, PTTK 44/34 and platelets dropped to 51 X 10⁹/L. He was given 4th 100 ml of anti-snake venom, and transfused with whole blood.

On the next day, patient was started with inj neostigmine 2.5 mg plus injection atropine 1 mg TDS. Patient's breathing effort improved, and his muscle power increased to 3/5. Weaning trial was started again with PSV. Sedation was stopped. Within one hour, pressure support was stopped and a trial of T-piece was instituted. When his limbs power improved to 4/5, he was extubated.

His CK decreased on the following day to 188 U/L, platelets increased from 55 X 10⁹/L to 86 X 10⁹/L. D dimers became > 400 < 800 and power was measured at 5/5. His condition improved constantly, inj neostigmine was stopped and he was shifted to ward, to be discharged with complete recovery.

DISCUSSION

As per estimates of World Health Organization, more than 4 hundred thousand envenomations occur every year, and 20,000 deaths are resulted, though the figures can be much higher than this.¹ Black mamba is one of the most feared snakes in the world as its venom is highly lethal. It contains different toxins; however, highest quantity is of α -neurotoxins,² which is also called postsynaptic neurotoxin, and it binds to nicotinic acetylcholine receptors, disrupting the neurotransmission at neuromuscular junction.³ This mechanism is considered to be responsible for respiratory paralysis in snake bite victims. Dendrotoxins, on the other hand, act presynaptically; these block potassium channels, increasing release of acetylcholine and producing effect like a depolarizing block.⁴ This actually explains the life threatening muscle paralysis which is found in black mamba bite, which necessitates mechanical ventilation in addition to administration of anti-snake venom. However, symptoms are not just limited to muscle paralysis. Our patient developed derangement in blood CT right from the beginning. An increased CK pointed to rhabdomyolysis, although it was not of clinical significance in our patient.

Use of neostigmine in such patients can be a tricky issue. This is because of the fact that if a patient comes with predominantly dendrotoxin effects, administration of neostigmine can add further damage by increasing levels of acetylcholine. Zavada J and colleagues⁵ presented a case report in which a young adult presented with muscle fasciculation. He was put on mechanical ventilator and given anti-snake venom, however, when muscle relaxation was reduced, his fasciculation increased. He was given muscle relaxants intermittently. And no neostigmine was administered due to the reason that this was predominantly dendrotoxin effect. There were no symptoms of fasciculation in our patients, however, and when injection neostigmine was administered, his muscle power improved markedly.

CONCLUSION

Use of neostigmine can be helpful in some of the victims of black mamba bite, and can reduce time of mechanical ventilation provided the dendrotoxin effects are minimum or can be excluded.

Conflict of interest: None declared by the authors.

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