CASE REPORT PROF-767

MANAGEMENT OF COBRA SNAKE BITE;
THE ROLE OF ANTICHOLINESTERASE

DR. MANZOOR AHMED FARIDI, MBBS, MCPS, FCPS
Senior Classified Anaesthetist.
Department of Anaesthesiology
Combined Military Hospital, Jhelum Cantt.

DR. TALAT BASHIR, MBBS
Medical Officer,
5-Independent Field Ambulance,
Jhelum Cantt.

DR. MUHAMMAD FAROOQ, MBBS. FCPS
Senior Classified Medical Specialist,
Department of Medicine,
Combined Military Hospital, Jhelum Cantt.

Correspondence:
Dr. Manzoor Ahmed Faridi
Senior Classified Anaesthetist
Department of Anesthesiology
Combined Military Hospital, Jehlum Cantt
Tel: +92-541-696162, 696163

ABSTRACT ... talatbashir22@hotmail.com We present the case of a 35-year old male who reported with history of cobra snakebite on the nape of his neck. He developed respiratory failure within hours. He was treated successfully with respiratory support, anti-venom administration and anticholinesterase. Detailed in this report is the reversal of envenomation symptoms following the administration of anticholinestrase, neostigmine methyl sulfate.

Key Words: Cobra Snakebite; Neurotoxic Envenomation; Anti-Cholinesterase.

INTRODUCTION

Approximately 15 percent of total 3000 species of snakes found worldwide are considered to be dangerous to humans[1]. Typically, the victims are male between 17 and 45 years of age. Most of the bites in Pakistan occur between March and October, when snakes are active, and humans are outdoors. These are more common within agricultural workers and military personnel.

Venomous snakes are divided into five families, elapidae, colubridae, hydrophydae, crotalidae and viperidae. Cobra snakes belong to elapidae family and contain neurotoxins, which are basically, polypeptides, needing large doses of anti-venom serum.

These neurotoxins bind to acetylcholine receptors at motor end plates resulting in a clinical picture resembling myasthenia gravis. Anti-cholinesterase drugs may reverse the potentially lethal neurological effects of the venom[2]. Respiratory failure and cardiac arrhythmias are the main causes of mortality and morbidity in victims of cobra snake.

CASE REPORT

A young soldier, 35 years of age, was brought to the hospital with 03 hours history of snakebite on the

neck. The snake was positively identified as a cobra. He complained of pain all over the body. There was no history of bleeding from the wound, mouth or other orifices. He had difficulty in focusing and developed dryness of mouth, difficulty in swallowing and deglutition. On examination, he was restless and had ptosis as well as diminished tendon reflexes. Two fang marks could be seen be seen on the nape of his neck. There was no local bruising or bleeding from the wound.

He developed gradual weakness of muscles and difficulty in breathing with drowsiness. Oxygen was being delivered through facemask but SaO_2 dropped to 40% and patient became semiconscious. Patient was immediately intubated and was put on ventilator. Polyvalent anti-venom serum was started (100 ml diluted in 500 ml in dextrose water, at about 30 drops per minute) after test dose. Considering the resistance of neurotoxic snakebite to anti-venom serum, the dose was increased to 450 ml.

But patients did not show any immediate improvement. Lab investigations were carried out including hemoglobin, serum urea, creatinine, electrolytes, blood glucose, LFTs, PT, PTTK and urine for FDPs. All these were within normal limits.

On second day, injection neostigmine 2mg was given as a trial. Injection atropine was added to counter the muscarinic effects of neostigmine. Patient responded with improved muscle movements and breathing. After successful trial, he was placed on injection neostigmine 1mg hourly. Injection atropine was added to every dose of neostigmine. Gradually patient developed a lot of improvement in reflexes, muscle power and breathing. He was weaned off from ventilator on fifth day. He remained on anti-cholinesterase for next four days. He was being strictly monitored for neurotoxic abnormalities and side effects of the drugs used. Physiotherapy continued and finally he was discharged from hospital after two weeks. He is presently asymptomatic.

DISCUSSION

The cobra snakebite is an important cause of mortality and morbidity. Most cobra are large snakes, 1.2-2.5 m in length. The king cobra, which may reach 5.2m in length, is the largest venomous snake in the world. Cobras live throughout most of Africa and southern Asia. Their habitats vary. Some species adapt readily to life in cultivated areas and villages.

Cobras, like other members of the family, have a pair of short, fixed fangs in the front of their mouths. They attack their victims by bitting, firmly maintaining a hold while injecting venom in a succession of chewing movements. Cobras' envenomation is an extremely variable process. The envenomation of some species causes profound neurological abnormalities. Neurotoxicity characteristically involves ptosis, opthamoplegia with blurred vision or diplopia, dysphagia, dysarthria, flaccid paralysis, loss of deep tendon reflexes, coma and respiratory paralysis[3]. Most of the neurological abnormalities were present in our case.

The main lethal component of the venom is believed to be post-synaptic neurotoxin that binds to the nicotinic cholinergic receptor sites at the neuromuscular junction, producing an effects similar to that seen in curare poisoning [4].Treatment includes respiratory support, anti-venom serum administration and anticholinesterases.

The role of anti-venom serum in the management of neurotoxic snakebite is limited[5]. Treatment often requires administration of large quantities of anti-venom, resulting in high incidence of serum reactions, however a recent study had documented reduction in acute adverse reactions to polyvalent snake anti-venom after use of 0.25 ml of 1:1000 adrenaline given subcutaneously, before start of infusion[6]. In a local study it was seen that available polyvalent snake anti-venom has little to offer in cases of neurotoxic snakebite [7].

In our case although 450 ml of polyvalent anti-venom serum was administered, improvement started only after adding anticholinesterase. Injection neostigmine was used to see the response to anticholonesterase due to non-availability of edrophonium.

Normally edrophonium testing is recommended to predict whether or not a particular case will respond to neostigmine, The patient may then be treated with a longer acting anti-cholinesterase such at neostigmine methyl sulfate (0.5 mg for adults), intravenously, after every 20 minutes[8].

In another study, evidence supports the use of cholinesterase-inhibiting drugs such as edrophonium and neostigmine, as a temporary measure in a situation of severe cobra venom poisoning with significant neurological abnormalities until anti-venom can be obtained [9]. The muscarinic effects of neostigmine are countered by injection atropine simultaneously, Later the patients may be placed on tablet pyridostigmine [10].

Respiratory embarrassment is the main cause of mortality and morbidity in a neurotoxic anakebite, therefore a vigilant look at respiratory status with arrangements for ventilatory support should be ensured. Use of anti-cholinesterase in recommended in cases of neurotoxic snakebite.

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