CASE REPORT PROF-1190

BRAIN DEATH CRITERIA;
RECONSIDERATION AFTER SNAKE BITE NEED

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INTRODUCTION

Snake bite is an important cause of mortality and morbidity. It is estimated that each year snake bite is responsible for 30,000 to 40,000 deaths world wide[1]. Most snake bites are innocuous and are delivered by non poisonous species. There are 15% of the more than 3000 species of snakes, which are considered dangerous to humans[2]. The family Viperidae (Pit vipers) is the largest family of venomous snakes. The sub-family Crotalidae (Pit vipers) is also very common in certain parts of the world and includes rattle snakes, cotton mouths (Agkistrodon) and copper heads (Agkistrodon). Elapidae includes the coral snakes. Ophiophagus Hannah (King Cobra) elapidae (kraits and Cobras), Viperidae (Pitless vipers), crotalidae (pit vipers) are indigenous snakes of Pakistan[3]. Venom is produced by paired glands below the eye. The venom dosage per bite depends on the laps of time since the last bite, and the degree of threat the snake feels.

The nostril pits respond to the heat emission of the prey. Venom is most by water and enzymatic proteins like proteases, collagenase and arginine ester hydrolase and hyaluronidase which promotes spread of venom by tissue destruction[4]. Coral snakes produce potent neurotoxins that later causes respiratory failure from typical systemic neuromuscular blockade. Myonecrosis raises concerns about myogloinuria and renal damage. In clinical practice the snake bite patient either present as haemotoxic or with neurotoxic manifestation.

King cobra (Ophiophagus), India cobra (Naja Naja) and brown cobra (Naja Oxiana) produce predominately neurotoxic symptoms. We hereby report a case of cobra snake bite which totally paralyzed the patient for twenty six days. He recovered defying all the clinical brain death criteria[5].

CASE REPORT

On 5th July 1998 at 0300 Hrs in the morning I was called to attend a patient of 30 years of age who was bitten by a Cobra snake with gradually diminishing chest movements and was drifting towards complete apnoea. He was bitten on right shoulder thrice with triangular fang marks. He was unable to speak and moving his hands to explain something. His both eyelids were twitching and was lying flat and listless and cyanosis was developing. I immediately gave him 1 mg of Dormicum I/V, intubated the patient and with the help of Ambu bag, shifted him to ICU and put him on ventilator. The moment patient was put on ventilator, his own respiratory efforts became zero.

History revealed that he was bitten by a cobra snake while sleeping on “Charpai” out side his village home. When his condition deteriorated, his brother brought him to civilian medical specialist who immediately referred

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him to CMH Sialkot. There was no history of nausea, vomiting, and any bleeding tendency except sinking of heart and breathlessness at home as stated by his brothers.

His blood CP, urine analysis, Coagulation profile (PT, PTTK) BT, serum Fibrinogen level, FDP's platelets count was normal. Chest was clinically clear on auscultation, X-ray chest was normal. Keeping in view the neurotoxic nature of the venom, he was initially given intravenous 1 ml/Kg (50 cc) of polyvalent snake antivenom in 100 ml of N/salinc, injection Neostigmine 1 mg I/V stat and 0.5 mg four hourly . Inj Solucortef 200 mg, intravenously stat and 100 mg intravenous 8 hourly for next two days. Injection T.T 1 ml intramuscularly was given to the patient. Antiseptic dressing was applied at the site of bite.

DINAMAP was applied to monitor his blood pressure and oxygen saturation. Next morning the patient was seen by two senior medical specialists who repeated the polyvalent snake anti venom dose. On examination, the patient was an esthetic built, lying flat on bed. His systolic blood pressure was fluctuating between 80mmHg to 120 mmHg and was on ventilator with assist/control mode. There was no spontaneous respiratory effort. On examination both pupils were fully dilated and were non reactive to light. Corneal reflex was also absent on both eyes. On fundoscopy there was no papilloedema and retina was normal. Oculocephalic reflex (Doll's head eye phenomenon) was also absent, caloric test to 50 ml of ice cold water was also negative. Superficial and deep sensation and tendon reflexes were also absent.

There was also (Doll's hand eye phenomenon) was also absent, caloric test to 50 ml of ice cold water was also negative. Superficial and deep sensation and tendon reflexes were also absent. There was also autonomic instability and his blood pressure was fluctuating. He was given intravenous parental nutrition with 10% dextrose water, aminovil and lipofundin-N 20%. On third day, the physical status of the patient remained the same and was declared verbally as brain dead patient.

I myself was convinced because clinically he was fulfilling all the clinical criteria of brain death. But as EEG facility to look for flattening of type IV, and digital subtraction angiography was not there in CMH Sialkot and keeping in view his young age, I thought it appropriate to ventilate the patient till the time, the patient would maintain his blood pressure. The patient remained on ventilator for twenty five (25) days under supervision of OT staff round the clock. Patient's pupils remained fixed and dilated for twenty two (22) days and there was no respiratory effort. On twenty third day patient recovered and there was no sensorimotor loss. Mild subglottic granuloma was operated upon by ENT specialist and patient fully recovered.

DISCUSSION

Sporadic cases of snake bite are quite common in our country and need specific therapy. Polyvalent snake antivenom is more effective against haemotoxic effects of viper than against cobra snake bites which bind with acetylcholine receptor of neuromuscular junction[6]. Solucortef 200 mg stat and 100 mg 8 hourly was given to this patient to counter any adverse effects due to polyvalent antisnake vaccine. Adrenaline 0.25 ml of 1:1000 can be used for this purpose.

In the field care the goal should be to support the patient until they arrive at an emergency department. Firm pressure bandage and immobilization of the part substantially delays spread of the venom[7]. Do not harm the patient by giving incision, mouth suctioning or tourniquets. Adhere to the tenants of emergency life support. Monitor vital signs and establish one large bore intravenous canula. Keep a close watch on air way and ventilation.

For Pit vipers observe for 8 to 10 hours and for Coral snake for a minimum of 24 hours. Evaluate to rule out compartment syndrome. Injection Neostigmine at least 0.5 mg 4 hourly should be given to neurotoxin producing snakes. Antibiotics should be given to these patients as the months of snakes contains anaerobes.

This was a classical case of Cobra snake bite that paralyzed the nervous system and impulses for breathing. The patient was given anticholinestrase (neostigmine) for initial three days and no anticholinergic was given to this patient but his pupils remained dilated and fixed for complete twenty two(22) days which were

not even responsive to light.

The lesson to learn from this case is that the brain death criteria either should be changed as this patient was fulfilling all the clinical criteria of brain death or at least

should not be applied to victims of neurotoxin producing snake. My opinion is that perhaps all the victims of neurotoxin producing snakes can be saved if timely ventilator support is being provided to these patients before hypoxic injury to brain and kidney occurs due to respiratory arrest. The neurotoxin at times is far more potent and at times requires ventilatory support for prolong period of time and resuscitative effort should not be given up prematurely.

REFERENCES

1. Cold BS, Barish RA, venomous **snake bites.** Emergency Med clin North Am; 1992;1249.
2. Snake bite; update; by Brain James program director, university of Tennessee school of medicine.
3. A guide to the snake of Pakistan, Mohammad Sharif Khan, Edition Chimaira Frankfurt am main 2002 (www.chimaira.de)
4. Warrell DA. **Injuries, envenoming, poisoning and allergic reactions caused by animals.** In; weatherall DJ, ledingham JGG. Warrell DA, 3rd edition. Oxford test book of medicine volume 1, oxford 1996: 1124-51.
5. **Basis of intensive therapy by;** Professor Mehdi Hassan Mumtaz 1994; Brain death 807.
6. JP, **snake bite in:** Bennet J, Plum F, Text book of medicine.eds.cecil WB Saunders, Philadelphia. 1996; 1951-53.
7. A A H Lawson: **Acute poisoning.** In: Davidson's principles and practice of medicine Churchill living stone 1995; 1144-45.