FIELD MEDICINE

CLINICAL SPECTRUM OF SNAKE BITE AND THERAPEUTIC CHALLENGES

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ABSTRACT

Objective: To determine the clinical spectrum, therapeutic challenges and outcome of patients of snakebite presenting to Azad Kashmir Combined Military Hospital (AK CMH) Rawalakot.

Study design: It is an observational descriptive study.

Place and duration: The study was carried out at AK CMH Rawalakot from January 2006 to August 2006.

Patients and Methods: Adults above 12 years of age with definitive history of snakebite were included in this study. Detailed history and examination was carried out. Vital signs and peak expiratory flow rate were monitored regularly. Blood complete picture with platelets count, bleeding time, clotting time, and urine for red blood cells were done in all patients. All patients with local or systemic signs of envenomation were administered antisnake venom.

Results: Fifty adult cases of snakebite were studied. Typically, victims were male between 15 and 30 years of age. Thirty-five (70%) of them showed evidence of envenomation and all of them were administered polyvalent antisnake venom and three of them developed hypersensitivity to antisnake venom

Conclusion: Most of the snakes found in this area are venomous and general population has very little knowledge of appropriate first aid measures. Delay in treatment increases mortality and morbidity therefore adequate supply of antisnake venom should be maintained in all remote health facilities.

Keywords: Snakebite, envenomation, antisnake venom

INTRODUCTION

Approximately 15 percent of the 3000 species of snakes distributed worldwide are venomous. Snake envenomation is termed technically as ophitoxaemia. The families of venomous snakes are Atractaspididae, Elapidae, Hydrophidae and Viperidae. These families include. Elapidae, which includes snakes, found in the Indian sub-continent viz., common cobra, king cobra and krait. Viperidae which includes Russell's viper, Pit viper and saw-scaled viper. Hydrophidae which includes the sea-snakes.

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Deaths typically occur in children, in the elderly, and in victims to whom antivenom is not given, given after a delay, or is administered in insufficient quantities [1]. Typically, victims are males and between 17 and 27 years of age [2]. Ninety-eight percent of bites are on extremities, most often the hands or arms. Definitive diagnosis of snake-venom poisoning requires positive identification of the snake and clinical manifestations of envenomation. Snake parts should not be handled directly, since the bite reflex in recently killed or decapitated snakes remains intact, rendering them capable of inflicting a bite [3].

The mortality and morbidity of snake bite depends on species of snake, the amount of

venom that is injected by the bite and bitten persons disposition.

Purpose of this study was to determine the clinical spectrum, therapeutic challenges and out come of patients of snakebite presenting to AK CMH Rawalakot.

PATIENTS AND METHODS

Adults above 12 years of age with definitive history of snakebite were included in study. Detailed history and examination was done in all patients. Vital signs and Peak Expiratory flow rate were monitored regularly. Blood complete picture including platelet count, bleeding time, clotting time, prothrombin time, and urine for red blood cells were done in all patients.

Data had been analyzed using SPSS version 11.0. Percentages were used to describe the data.

RESULTS

Fifty adult cases of snakebite were studied. Virtually all patients with snakebite were apparently apprehensive and frightened. Typically, victims were males between 15 and 30 years of age. Thirty-four (68%) of them were males and 16(32%) were females. Ninety-eight percent of bites were on extremities. Thirty-three (66%) were bitten on feet and ankles, sixteen (32%) were bitten on hands and arms and one (2%) was bitten on scrotum. Majority of patients had applied tourniquets, while an overwhelming majority had applied it incorrectly. Forty-one (82%) patients had applied tourniquets and only five (12%) of them had applied it correctly. Twenty-seven (54%) had visible fang marks, nineteen (38%) had distorted fang marks due to local cuts while four (8%) had no visible bite or cut mark. Thirty-five (70%) of them showed the signs and symptoms of toxicity; 34 of these were having vasculotxicity / hematological derangement. All of these patients had local signs of envenomation, eight of these had enlarged tender regional lymph nodes and twelve had hematological derangement. All patients with enlarged regional lymphnodes had evidence of systemic toxicity. One (2%) patient had severe disseminated intravascular coagulation and died. Only one case proved to be neurotoxic and developed ptosis followed by diplopia. The largest number of snakebites occurred during June-August. Fifteen patients did not show toxicity. All patients with evidence of local or systemic envenomation were administered antisnake venom. Three patients developed hypersensitivity to anti snake venom.

Premeditations used were steroids and antihistamines to prevent anaphylactic reactions to antisnake venom.

DISCUSSION

Snake venom, the most complex of all poisons is a mixture of enzymatic and non-enzymatic compounds as well as other non-toxic proteins, carbohydrates and metals. The variation of venom composition from species to species explains the clinical diversity of ophitoxaemia. Ophitoxaemia leads to increase in the capillary permeability which may cause loss of blood and plasma volume into the extravascular space leading to edema and may be severe enough to compromise circulation.

The venom may also have direct neurotoxic action leading to paralysis and respiratory arrest, cardiotoxic effect causing cardiac arrest, myotoxic and nephrotoxic effect.

The most common and earliest symptom following snake bite is fright and may produce psychological shock and even death. It was seen in all of our patients and reassurance is the mainstay of its management. Anxiolytics and sedatives are best avoided in patients of snakebite. With the possible exception of the psychological trauma of being bitten, local changes are the earliest and commonest manifestations of snakebite [4]. The most common manifestation of envenomation in our study group was local swelling followed by reddish or blue discoloration and blistering. Characteristic fang marks were seen in 54% of our patients while 38% had distorted fang

marks due to local cuts. Regional lymphadenopathy is an early and reliable sign of systemic poisoning. It was seen in eight of our patients and all of them had evidence of systemic envenomation.

Early systemic manifestations usually include nausea, vomiting, perioral paresthesia, lethargy, and weakness. More severe systemic effects include hypotension, tachypnea, tachycardia, and altered sensorium. Bites by rattlesnakes may result in a consumptive coagulopathy manifested by a prolonged or unmeasurable prothrombin time and activated partial-thromboplastin time, hypofibrinogenemia, the presence of fibrin-degradation products, or a platelet count of less than 20,000 per cubic millimeter.

Pit-viper venom increases the permeability of the capillary membranes, resulting in the extravasation of electrolytes, albumin, and red cells into the envenomated site. Renal failure may result from hypotension, intravascular hemolysis, a syndrome resembling disseminated intravascular coagulation, or nephrotoxic effects of components of venom [5]. One of our patients died of severe disseminated intravascular coagulation.

Cobra produces symptoms as early as 5 minutes or as late as 10 hours after the bite. One of our patients presented with ptosis and diplopia that developed within four hours of bite. Ptosis and diplopia persisted despite administration of antisnake venom but they did not progressed. Vipers take slightly longer - the mean duration of onset being 20 minutes. However, symptoms may be delayed for several hours. Sea snake bites almost always produce myotoxic features within 2 hours so that they are reliably excluded if no symptoms are evident within this period. Neurotoxic features are a result of selective d-tubocurarine like neuro-muscular blockade which results in flaccid paralysis of muscles. Cobra venom is however 15-40 times more potent than tubocurarine. Ptosis is the earliest neuroparalytic manifestation followed closely by opthalmoplegia. Generally muscles innervated by cranial nerves are involved earlier. However, pupils are reactive to light till terminal stages. Muscles of chest are involved relatively late with diaphragm being the most resistant. This accounts for the respiratory paralysis, which is often terminal. Reflex activity is generally not affected in ophitoxaemia and deep tendon jerks are preserved till late stages. Onset of coma is variable; however several cases of cobra bite progress to coma within 2 hours of bite. Symptoms that portend paralysis include repeated vomiting, blurred vision, paraesthesiae around the mouth, headache, dizziness, vertigo and signs of autonomic hyperactivity. The onset of neurotoxic effects may be delayed up to 12 hours [6] .Once manifestations appear, it may not be possible to prevent further effects or reverse the changes that have already occurred

Cardiotoxic features include tachycardia, hypotension and ECG changes. Haematological changes - both local as well as systemic - are some of the commonest features of snake bite poisoning. Bleeding may occur from multiple sites including gums, GIT (haematemesis and melaena), urinary tract, injection sites and even as multiple petechiae and purpurae. Subarachnoid haemorrhages cerebral haemorrhage and extradural haematoma may also occur rarely.

Almost every species of snake can cause renal failure. It is fairly common following Russell's viper bite and is a major cause of death.

One patient who had local swelling of index finger and was given antisnake venom had persistent swelling of proximal interphalangeal joint after one month. Delayed onset of signs is rare that occur due to local blebs constituting a venom depot generally inaccessible to antivenom which is suddenly released into the blood stream, especially when the wound is handled surgically.

Three of our patients developed mild hypersensitivity reaction to antisnake venom but none of them had history of previous exposure to antisnake venom. Individuals

bitten twice may develop hives and angioedema within 15 minutes of the second bite.

Naja nigricollis (spitting cobra) can eject venom with considerable accuracy even from a distance of 6-12 feet aiming at the victim's eyes resulting in conjunctivitis and corneal ulceration. We had no patient of toxemia without bite.

After a bite from any venomous snake, the victim should be moved beyond striking distance, placed at rest, kept warm, and transported immediately to the nearest medical facility [7]. The bitten part of the body should be immobilized in a functional position below the level of the heart. None of our patient had immobilized the bitten limb. Previously recommended first-aid measures such as tourniquets, [8] incision and suction, [9] are strongly discouraged. In our study majority of patients had wrongly applied the tourniquets. Paramedical personnel should focus on support of the airway and breathing, administration of oxygen, establishment of intravenous access in an unaffected extremity, and transportation of the victim to the nearest medical facility.

Victims of bites from venomous snakes require aggressive supportive care and sometimes the administration of antivenom. Once airway, breathing, and circulation have been established, a rapid, detailed history should be obtained. Key points include the time of the bite, a general description of the snake, first-aid measures used, coexisting medical conditions, drug and food allergies, allergy to horse or sheep products, and history of snakebite and consequent therapy. The physical examination should be complete, with special attention to the cardiovascular, pulmonary, and neurologic systems. The bite should be examined for fang or tooth marks and scratches, edema, erythema, and ecchymoses. Base-line laboratory studies should include a complete blood count with platelet count, coagulation profile (international normalized ratio (prothrombin time), activated partial-thromboplastin time, and fibrinogen level), measurement of fibrin degradation products, electrolytes, blood urea nitrogen, and serum creatinine, and urinalysis [10]. Laboratory studies should be repeated after each infusion of antivenom. Immunization against tetanus should be administered if indicated by the patient's history.

Since manifestations of envenomation can be delayed, it is recommended that all patients be observed for a minimum of twenty four hours. If no clinical or laboratory manifestations have presented during this time, the patient may be discharged. A mild envenomation syndrome at one hour could progress to a severe syndrome within several hours and, without continuous observation, lead to death. Monitoring in an intensive care unit is recommended for all patients treated with antivenom. There have been no controlled trials to establish the efficacy of pretreatment with epinephrine, histamine H1- and H2-receptor blockers, or corticosteroids but some experts pretreat routinely [8]

Local necrosis and coagulopathy are not seen in persons with coral-snake envenomation. Because coral-snake venom has a potent neurotoxic component, monitoring should focus on neuropathic symptoms. Patients require frequent assessment of oxygen saturation and ventilatory function. Ventilatory support may be required.

It is inadvisable to attempt to correct a coagulopathy until sufficient quantities of neutralizing antivenom have been administered. Treatment with coagulation factors or blood components adds more substrate for unneutralized venom, thus increasing the levels of degradation products, which are also anticoagulant.

Opioid analgesics should be avoided if the venom is known to have neurotoxic components so as to avoid masking neurotoxic effects. Wound infections are rare; therefore, the prophylactic use of antibiotics is not recommended [11].

Severe envenomations by rattlesnakes may be associated with compartment

syndrome and elevation of the bitten body part in conjunction with the administration of an additional four to six vials of antisnake venom over the course of one hour is recommended [12]. Additional antivenom should effectively neutralize the venom components, thereby reducing compartment pressure.

If these measures fail to reduce compartment pressure within four hours and the patient has circulatory compromise, fasciotomy may be required to lower the compartment pressure. [13] Serum sickness is a type III hypersensitivity reaction that may occur 7 to 21 days after the completion of treatment. It is manifested as fever, rash, arthralgias, and lymphadenopathy and responds well to a tapering course of oral prednisone, starting at a dose of 60 mg per day.

While there are many factors influencing the outcome in victims of snake-bite, there is significantly higher mortality among victims who have delayed presentation [14] and develop neurotoxicity. On an average - cobras and sea snakes result in about 10% mortality ranging from 5-15 hours following bite. Vipers have a more variable mortality rate of 1-15% and generally more delayed (up to 48 hours). Only one (2%) of our patients died of intravascular coagulation resulting in consumption coagulopathy.

CONCLUSION

Majority of snakes in this area are poisonous predominantly vasculotoxic. Most of the bites occur on extremities in young individuals during the summer season.

RECOMMENDATION

- Personal protective measures should be ensured at all levels particularly during the summer season.
- No attempt should be made to capture or kill the snake by the bitten person as it may lead to further bites and increase venom absorption.
- Application of tourniquets, cuts and suction should be strongly discouraged as these are more likely to be detrimental than beneficial due to wrong technique.
- Patients of snakebite should be reassured, bitten part be immobilized, and rapidly transported to hospital, vigilant monitoring and timely treatment may be life saving.

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