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A REPORT ON TWO CASES OF SNAKE BITE IN EAST DELHI (A CASE REPORT)

Medicine

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ABSTRACT

A 16 year old male presented with a history of cobra bite having severe swelling of the left upper limb at the bitten area. Treatment was immediately started with Anti-Snake Venom. The second case was a 19 year old male presented with a history of snake bite without any signs of venom exposure at presentation. On the third day features of acute renal failure were apparent. He was treated with Polyvalent Anti Snake venom and hemodialysis. The patients were followed up and recovered in due course of time.

KEYWORDS

Cobra, snake bite, envenomation, Anti snake venom, Renal Failure

INTRODUCTION

Snake bite is a widespread and neglected public health problem in tropical and subtropical countries, where rural populations are mainly affected. It is a common occupational hazard mainly in farmers, plantation workers, herders and laborers leading to significant morbidity and mortality that remains largely unreported.[1] The most affected region in the world is South East Asia because of dense population and extensive agricultural practices. The WHO has included snake bite in its list of neglected tropical conditions in 2009.[2]

India is reported to have the highest number of snake bites (81,000) and deaths (11,000) per year.[3] However, the geographical distribution and statistics are variable in the country due to gross underreporting, resulting in massive statistical disparity. Estimates of death due to snake bite range widely from 1,300-50,000.[4] A nationally representative snake bite mortality survey in India (2001-2003) has highlighted 45,900 deaths annually, with the highest mortality rate in the state of Andhra Pradesh.[5]

Snake bite is a common occurrence in the Eastern Delhi Region with considerable morbidity and mortality. Therefore, it is of utmost importance to identify the features of poisonous snake bites and treat them promptly. The common poisonous snakes found in India are King Cobra, Common Cobra, Krait (Elapidae family) and Russell's Viper.[1]

Case 1

A 16 years old male patient attended the Emergency Department with history of snake bite around 4 hours back on the left hand .On presentation, Ptosis was present and fang marks were seen over the left wrist. The single breath count was 24 with no history of breathing difficulty, dark brown discoloration of urine, nausea, vomiting with local tenderness at the site of bite. There was no bleeding from any site. There was a tight tourniquet at the left forearm. On investigation, 20 minute whole blood clotting test was normal, as were other blood counts. The limb was immobilized, tourniquet was removed and the limb was kept elevated. She was given 10 vials of Anti Snake Venom along with 5 ampules of Neostigmine. Other Supplementary drugs used were Atropine, Intravenous Antibiotics, Anti-inflammatory agents, Tetanus Toxoid and Intravenous fluids. Ptosis disappeared after few hours but he developed intense pain and swelling of the left forearm, upper arm and wrist with necrosis around the bitten area which lasted for few days. Wound debridement was done after an interval period. The recovery was uneventful and he was discharged from the hospital after 8 days.

Case 2

A 19 year male presented with a history of snake bite around 10 hours back. He presented with pain and oedema of the left leg. There was a tourniquet at left thigh. On examination, fang marks were clearly visible without any systemic symptoms and a normal Systemic examination. There was no bleeding from any site, no ptosis, fatigue or respiratory distress with a normal urine output. All relevant investigations were within normal limits. Tourniquet was removed and limb kept immobilized and elevated. Conservative treatment was started. Within a few days, he started complaining of decreased urine output as well as dark discoloration of urine. He was found to have haemoglobinuria , with increased AST, ALT. Serum creatinine was 5.8 mg/dl, platelet count was 88,000/mm3. Anti-Snake Venom was started immediately and supplemented with I.V Prednisolone considering a vasculotoxic snake bite. First haemodialysis was done on the 5th day. Subsequently, patient developed Anuria, Pulmonary Oedema and Hyperkalaemia. His Creatinine was raised upto 10.8 mg/dl within seven days. He underwent Haemodialysis four times within a span of ten days. He was discharged after one month with a normal urine output. Subsequent follow up visits were uneventful.

DISCUSSION

Snake bite is a common medical emergency, where timely treatment can reduce morbidity and mortality and save precious human lives. Poor access to health care services, difficult transportation and consequent delay in ASV administration result in high fatality. The time elapsed after the bite is of vital importance, because with the passage of time more venom gets bound to the tissues and is thus less manageable for neutralization by ASV.[1] Moreover there is a lot of uncertainty in the doses of ASV, though National Protocol on Snake Bite Management formulated by the Ministry of Health & Family Welfare, Government of India is in place besides the WHO Guidelines[6,7]. Further, the peripheral health care facilities are not well equipped and there is shortage of ASV and emergency drugs necessitating a trip to well-equipped tertiary care hospitals, where treatment may become unaffordable for many. High mortality can be attributed to loss of crucial time and lack of proper treatment.[1]

Snake Venom

Snake venom contain Toxins which are mostly peptides and proteins of low molecular weight; enzymes like proteinases, hydrolases, transaminases, hyaluronidase, cholinesterase, phosopholipase, ATPase, ribonuclease, deoxyribonuclease, and miscellaneous agents like agglutinins and proteolysins.[8] There are more than hundred different proteins in each venom; with elapid and viperid venoms constituting 25-70% and 80-90% of enzymes respectively. Some non-enzymatic polypeptide toxins and non-toxic proteins are also present.[9] The venom enzymes include hydrolases, hyaluronidase, kininogenase. Other enzymes include phosphomono-and diesterases, 5'-nucleotidase, DNAase, NAD-nucleosidase, l-amino acid oxidase, phospholipase A2(PLA2), peptidases and zinc metalloproteinase hemorrhagins.[1] The snake venoms are mainly characterized as Neurotoxic (elapids), Haematotoxic (Vipers) and Myotoxic (sea snakes). The neurotoxic venoms act at molecular level, by disrupting the neuromuscular junctions, limiting muscle activity while hemotoxic venoms cause tissue destruction in body systems besides their effect on circulatory system. Blood clotting may be stimulated by serine proteases and other pro-coagulant enzymes present in some Elapid and Viper venoms.

Certain venoms contain toxins (Russell's viper) that activate factors V, X, IX and XIII, fibrinolysis, protein C, platelet aggregation, anticoagulation and hemorrhage. Widespread damage to

mitochondria, red blood cells, leucocytes, platelets, peripheral nerve endings, skeletal muscle, vascular endothelium, and other membranes is caused due to phospholipase A2, the most widespread enzyme present in the venom. Hyaluronidase aids in venom dissemination from the bite site through tissues. Most elapid venoms contain acetylcholinesterase, which could cause tetanic paralysis. Among the polypeptide toxins are postsynaptic (α) neurotoxins that bind to acetylcholine receptors at the motor endplate. Presynaptic (β) neurotoxins release acetylcholine at the nerve endings at neuromuscular junctions and damage the endings, interfering with its release.[3]

Clinical Presentation

Clinical features can be divided into local, general and systemic manifestations. Local symptoms and signs include fang marks, pain, bleeding, bruising, lymph node enlargement, inflammation, blistering, infection, necrosis etc. General manifestations include nausea, vomiting, malaise, abdominal pain, weakness, drowsiness and prostration. The Elapidae mainly causes Neurological manifestations. The viper venom involves many systems causing Cardiovascular, Renal and Bleeding and Clotting disorders. Russell's viper venom is also associated with Neurological manifestations.[8]

20 minute whole blood clotting test is very useful and informative bedside test requiring very little skill. Other investigations include CBC, peripheral blood film, aminotransferases, muscle enzymes, serum creatinine, BUN, serum potassium, ABG, oxygen saturation and urine examination for haemoglobin, myoglobin, protein and microscopy.[6] However, viper bites should be observed for recurrent coagulopathy as occult coagulopathy can recur up to 2 weeks after the bite.[7]

The Polyvalent Anti-snake venom

The most effective antidote against snake venom is the anti-snake venom. It is usually pepsin refined F (ab) fragments of IgG purified from the serum or plasma of a horse or sheep that has been immunized with the venom of one or more species of snakes. In India, horses are hyper immunized against the venom of four common poisonous snakes the "Big Four" (Cobra, Krait, Russell's viper and Saw-scaled viper), to produce polyvalent anti snake venom. The venom is mostly procured from Chennai in South India. There are seven pharmaceutical laboratories in India that produce ASV against four medically important Indian snake species.[10,11]

Management of Snake Bite

In India, the high morbidity and mortality due to snake bites could be attributed to traditional, harmful first-aid measures like application of tight tourniquets, suction, application of herbal and medicines and above all the usual delay in carrying the patient to the nearest health care facility and providing appropriate medical care. In view of multiple treatment modalities followed by treating physicians in the country, the Ministry of Health & Family Welfare, Government of India has drafted the National Snake Bite Management Protocol to provide guidelines for proper management of snake bites.[7]

The foremost thing for a bitten victim is reassurance and immobilization with a splint or a sling followed by lightly wrapping a bandage. If possible identification of the snake and exact time of bite may help in determining the progression of impending neurotoxic or hemotoxic effects.

A brief history of the bite and the progression of local and systemic symptoms is mandatory. The management in hospital involves the care of airway and breathing, maintaining circulation and preventing shock. Examination of local signs and symptoms like fang marks, local pain, swelling, bleeding from the site or blister formation can also give some clues about the species of biting snake. Hemostatic abnormalities may be ascribed to vipers and neurotoxic manifestations principally to cobras and krait.

The necessary investigations include the 20 WBCT and usual hematocrit, biochemistry and arterial blood gases. The corner stone of management is administration of Anti-Snake Venom which is raised against the four common species of snakes found in India. ASV is given only in patients with evidence of systemic envenoming (coagulopathy, neurotoxicity) or severe local envenomation. Generally administration of 8-10 vials of ASV is recommended and further dosing depends on response to the initial dose[7]. For victims reporting late after several days, the presence of coagulopathy or neurotoxic symptoms determines the ASV administration. Since snake identification is generally not easy and the presentation can always be confusing, venom and antibodies detection in blood can be helpful. The adverse reactions are usually managed with antihistamines, adrenaline and late serum sickness with prednisolone and antihistamines.

Complications of Anti-snake venom

The main issues with ASV in actual clinical practice are species specificity, difficulty in availability, affordability and ideal storage conditions. One of the principal drawbacks of the immunotherapy is the issue of specificity.[1] There is a huge species variation with current taxonomy identifying one, four and eight species of Russell's viper, cobras and kraits, respectively. Russell's viper venom has also shown regional variation.[12] Venom variation, low potency, bites by other species could be responsible for the reported failure of polyvalent ASV in countering the venom effects in India.

Various logistic, marketing and economic issues also plague the production and supply of ASV. Undersupply of the venom is the main cause of insufficient production of ASV to meet the national requirement. The process of development is time consuming, requiring ideal storage conditions. Production in lyophilized form is costly, and there can be physiochemical changes in the product by lyophilization. The liquid form requires cold chain. There needs to be rapid technical advancement in production.[13] The other drawbacks with ASV therapy are the adverse reactions such as pruritus, urticaria and potentially fatal anaphylaxis as well as serum sickness in few cases.

CONCLUSION

The cases described above were from the 10-18 years age group and the sites of bite included extremities in both cases. A Central Healthcare policy needs to be formulated for Snake bite cases and implemented to ensure prompt availability and effective use of Anti-Snake Venom in the rural areas of the country. Compulsory training of young doctors is absolutely crucial in the overall diagnosis and management of the majority cases of snake envenomation.

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