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Snake Envenomation: Understanding Venom, Danger and Defense

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INTRODUCTION

Snakes are long, slender reptile which are considered as the most venomous. Venomous snakes belong to several families such as Elapidae, Viperidae, Crotalidae, Colubridae and so. Based on toxicity to the host, they are said to be neurotoxic and haemotoxic.

Elapines: They are Cobra family which includes Cobra, Mamba, Kraits and Coral snakes. They have short fangs and tend to hang on and chew venom to victims. They cannot bite through clothes. Their head is of about the same width as that of that of the neck and pupils of eyes are circular. Their venom is mainly neurotoxic.

Viperines: Vipers are of two types namely pitless/true vipers (Puff adder and Russell's viper) and pit vipers (Rattlesnakes and Copperhead). They have long and strong fangs and bite through clothes. Viperine venom is typically haemotoxic, necrotizing and anticoagulant.

TOXIC COMPONENTS

Snake venom is highly concentrated amber coloured digestive juice of the snake. It is a complex mixture of amino acids, proteins, enzymes, biogenic amines, metals and other inorganic substances.

Enzymes: Proteolytic enzymes(proteases) -digest tissue proteins and implicates anticoagulation events, Hyaluronidase -dissolves intercellular gel of connective tissue and helps in rapid spread of venom, Phosphodiesterase – affects cardiac system by lowering blood pressure, ATPases – metabolise ATP and affect victim energy system, Acetylcholinesterase- hydrolyses acetylcholine and interferes with neurotransmission in neuromuscular junction, Phospholipases A, B and C – haemolytic and myotoxic agent contributing cardiotoxicity,

Lipases -hydrolyses lipids, Collagenase- digests collagen, Ribonuclease- acts against ribonucleic acids, Deoxyribonuclease- acts against deoxyribonucleic acids, Ophio-oxidase- autolysis and putrefaction, Thrombin like enzyme- causes clot formation, L-Amino acid oxidase – gives yellow colour to venom.

Non-Enzymes: Low molecular weight peptides or polypeptides which includes neurotoxins, haemorrhagins(vasculotoxic), cardiotoxins, myotoxins (muscle necrosis and myoglobinuria), cytolisins (tissue necrosis) and toxalbumins.

FACTORS

Severity of envenomation depends on the species of snake involved, its age and size, number and depth of bites and total quantity of venom injected. Fatal snake bites are common in dogs due to their relatively small size and their nature of attacking snakes. It is less common in cats. Horses and cattle seldom die. Pigs are less susceptible to venom due to poor absorption of venom through layers of subcutaneous fat. Some bites don't result in envenomation thereby becomes dry bites. 30-70% of the total bites are dry bites. If there has been a previous bite, the victim develops some degree of humoral immunity and is less vulnerable to the toxic effect of the venom.

TOXICOKINETICS

The venom is deposited with the help of fangs into the victim's body. Once deposited, it is rapidly distributed in the body. If the venom is deposited directly into a vein, toxicological effects ensue very rapidly.

Neurotoxicity:

Post-Synaptic Neurotoxins (alpha -bungarotoxin and kappa-bungarotoxin) -primarily bind to cholinergic receptors in the neuromuscular junctions and antagonize the action of acetylcholine on skeletal muscles. They produce curare like effects and cause paralysis. These toxins are found in elapids and sea snakes.

Pre-Synaptic Neurotoxins (beta-bungarotoxin) - inhibit the release of acetylcholine at the myoneural junctions and neurotransmission. These toxins are found in banded kraits(elapids)

Haemotoxicity:

Anticoagulation- have fibrinolytic and fibrinogenolytic factors; activate plasminogen

Procoagulation (in viperine) -Presence of thrombin like enzymes thereby leading to disseminated intravascular coagulation.

Haemolysis- Haemorrhagins cause hemolysis of the blood corpuscles.

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Cardiotoxicity:

Increased capillary permeability and intravascular volume depletion →Reduced cardiac output and mediators like bradykinin, histamine and serotonin causing haemodynamic changes → Cardiovascular shock

Acute Kidney Injury:

Acute tubular necrosis is the most common, but proliferative glomerulonephritis, interstitial nephritis, ischaemic changes, and distal tubular damage

Clinical Signs

Viperines: Pain, restlessness, anxiety, incoordination of movement and lameness, marked discolouration of tissues, blood oozes from fang wound, swelling/oedema, skin-cold, pupils dilated, visual disturbances, dizziness, vomiting, salivation, diarrhoea, convulsions, hypotension with tachycardia, pulmonary oedema, unconsciousness

Elapines: Pain and swelling are minimal but systemic neurological signs predominate. Viscid saliva, Muscular weakness, paralysis, consciousness is usually retained, abdominal pain, convulsions, respiratory paralysis

Death usually occurs within 6 hours in elapine and 2-4 days in viperine.

DIAGNOSIS

- History, Physical examination
- 20WBCT -20 Minute whole blood clotting test – very useful and informative bedside test.

2 ml of freshly sampled venous blood in a small, new, dry glass vessel. Leave undisturbed for 20 minutes at ambient temperature. If the blood is still liquid (unclotted) and runs out, the patient has hypofibrinogenomia ("incoagulable blood") as result of venom-induced consumption coagulopathy.

Laboratory findings:

Haemoglobin concentration/haemocrit: transient increase indicates haemoconcentration resulting from a generalized increase in capillary permeability.

Platelet count: decreased

White blood cell count: an early neutrophil leucocytosis followed by leucopenia

Blood film: fragmented red cells ("helmet cell",schistocytes) are seen there is microangiopathic haemolysis or thrombotic microangiopathy.

Plasma/serum in blood samples: stained pinkish or brownish if there is gross haemoglobinaemia or myoglobinaemia

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Biochemical abnormalities: Plasma creatinine, urea/blood urea nitrogen and potassium concentrations are raised in acute kidney injury.

Snake venom detection kit- to quickly confirm venom presence from 2 drops of blood, preventing unnecessary anti-venom usage.

100% Accuracy in clinical trials.

OTHER INVESTIGATIONS

Radiography: Thorax-pulmonary oedema

Ultrasound: Pleural and pericardial effusion and bleeding into serous cavities

Echocardiography: Detecting reduced left ventricular ejection fraction in hypotensive and shock patients

TREATMENT AND MANAGEMENT

SPECIFIC THERAPY

Antivenom: Monovalent (activity against one species) or polyvalent (activity against two or more species of snakes). Antivenin should be administrated as soon as possible after the bite as delayed administration often diminishes its effect. As antivenins are derived from hyper-immunised horse/mule and contain concentrated purified immunoglobin, so chances of immediate or delayed immune reaction may occur in some patients.

In India, "polyvalent anti-snake venom serum" is raised in horses, using the venoms of the four most important venomous snakes (Indian cobra, Indian krait, Russell's viper, Saw-scaled viper). Antibodies raised against the venom of one species may have cross-neutralizing activity against other venoms, usually from closely related species. This is known as paraspecific activity.

Dog &Cat Dose: Dilute a single vial of antivenom 1:1 with saline and give over 20 minutes.

REACTIONS OF ANTIVENOM

Early anaphylactic reactions- usually within minutes and upto 180 minutes after starting antivenom, the patient begins to itch and develops urticaria, dry cough, fever, nausea, vomiting, abdominal colic, diarrhoea and tachycardia. A minority of these patients may develop severe life-threatening anaphylaxis: hypotension, bronchospasm and angio-oedema. To avoid this reaction, subcutaneous epinephrine (adult dose- 0.25mg), followed by an intravenous anti-histamine such as chlorphenamine.

Pyrogenic (endotoxin) reactions- usually develop 1-2 hours after treatment. If it occurs, antivenom administration must be temporarily suspended.

Late (serum sickness type) reactions- develop 1-12 days after treatment. Clinical features include fever, nausea, vomiting, diarrhoea, itching, recurrent urticaria.

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Storage and shelf-life: lyophilized antivenoms stored at below 25c for 5 years. Liquid antivenoms should be stored at 2-8c and not frozen.

SUPPORTIVE CARE

- Bitten limb or extremity should be immobilized to retard the absorption and distribution of venom in the body.
- Fluid therapy (e.g. lactated ringer's solution) may be required to prevent or control shock especially in pit viper envenomation.
- Corticosteroids are contraindicated.
- Anti-histamines are contraindicated.
- Tetanus antitoxin should be administered to avoid clostridial infection after snake bite.
- Analgesics could be used to control severe bite pain.
- Blood transfusion should be instituted in case of haemolytic or anticoagulant venoms.
- Broad-spectrum Antibiotics should be given to prevent wound infection and other secondary infections. They should be continued until all superficial infections heals.
- Alcohol should not be used to clean the wound because it causes vasodilation and wound promote uptake and spread of venom.

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