TRAINING MODULE FOR MANAGEMENT OF SNAKE BITE & COMMON POISONS

Department of Health & Family Welfare
Government of West Bengal
Handling common poison cases and snake-bites are well-known medical emergencies across the country including West Bengal, especially in rural areas.

Management of snake bite cases and patients affected by common poisons puts the Medical Officer at Emergency OPD in rural, periurban and rural settings to a challenge. There is a need to update & follow the current recommendations and ensure timely implementation. It is expected that the revised “TRAINING MODULE FOR MANAGEMENT OF SNAKE BITE & COMMON POISONS” will act as a quick reference guide.

This module will also enable the Health Department in achieving reduced mortality from snake bites and common household poisons, thereby, delivering quality treatment as per international standards in a local context.

I hope that these guidelines will help all Medical Officers and Nursing staff to improve the management of snake-bites & poison cases, especially in the peripheral health services. It will be useful in saving human lives and mitigate misery.

The efforts of the Director of Health Services, Public Health Division of the Directorate of Health Services, Directorate of Medical Education, IEC & Training Branch of Health Directorate, Experts from Medical Colleges & WHO NTD division (WB) in developing and editing the book is highly appreciated.

(Anil Verma)
Snakebite is a truly Neglected Tropical Disease (NTD) and is in priority list amongst NTDs in South East Asia. It was observed that in West Bengal, there is a rising trend of snake bite incidence and reported deaths over the year.

Management of snake bite cases and patients affected by common poisons is always challenging specially in peri-urban, remote and rural settings. With limited knowledge and exposure in both the fields, the young medicos often face the challenges to save the life of the victim in a poor resource set up. Both these matter are not covered in the academic curriculum in a way to develop desired level of competency. Medical Officers posted at primary health care institutes thus often feel uncomfortable and tend to refer the patient at higher center without providing proper and specific initial treatment. This leads to increased mortality and morbidity as the initial golden hours of management are lost.

Since my joining in a rural Block Primary Health Center near Sundarban area about three decades back, I have faced this challenge as the incidence and mortality were high for both snake bite and poisoning in that area. Since then, I felt this training need for the Medical Officers to build up their confidence to take care in the resource crunched primary set up. For Snake bite management, the current recommendations were updated considering the necessity. Preparing a new module with overview of all spectrums of poisoning, quick assessment and immediate management protocol in the primary set up was a challenging task which my Colleagues at Medical College have done excellently in a very short period of time. I am expecting that this combined “TRAINING MODULE FOR MANAGEMENT OF SNAKE BITE & COMMON POISONS” will act as a quick reference guide, build up capacity and confidence among the Doctors specially serving in isolation. This effort is expected to save many young and productive lives by delivering quality treatment as per international standards in the local context.

We are most grateful to all the contributors without whose active support it was not possible to prepare this module in such short time.

DATE : 23.05.2018

(Dr. A.K. Chakraborty)
<table>
<thead>
<tr>
<th>Chapter</th>
<th>Page No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foreword</td>
<td>iii</td>
</tr>
<tr>
<td>Preface</td>
<td>v</td>
</tr>
<tr>
<td><strong>MANAGEMENT OF SNAKE BITE CASES</strong></td>
<td></td>
</tr>
<tr>
<td>List of contributors</td>
<td>2</td>
</tr>
<tr>
<td>Introduction</td>
<td>3</td>
</tr>
<tr>
<td>Learning objectives</td>
<td>3</td>
</tr>
<tr>
<td>Epidemiology of Snakebite</td>
<td>3</td>
</tr>
<tr>
<td>Classification of Snake Bites</td>
<td>4</td>
</tr>
<tr>
<td>Death from Snake Bite</td>
<td>8</td>
</tr>
<tr>
<td>Contributing factors</td>
<td>8</td>
</tr>
<tr>
<td>Time between snake-bite and death</td>
<td>8</td>
</tr>
<tr>
<td>First Aid Treatment</td>
<td>8</td>
</tr>
<tr>
<td>Diagnosis of Snakebite &amp; Envenomation</td>
<td>9</td>
</tr>
<tr>
<td>Signs &amp; symptoms of Snake bite</td>
<td>9</td>
</tr>
<tr>
<td>Bite Mark</td>
<td>9</td>
</tr>
<tr>
<td>General signs and symptoms of Viper envenomation (Hemotoxic)</td>
<td>10</td>
</tr>
<tr>
<td>General signs and symptoms of Neurotoxic envenomation</td>
<td>11</td>
</tr>
<tr>
<td>Late – onset envenomation</td>
<td>12</td>
</tr>
<tr>
<td>Clinical features of a compartmental syndrome</td>
<td>12</td>
</tr>
<tr>
<td>Diagnosis and testing</td>
<td>12</td>
</tr>
<tr>
<td>20 Minute Whole Blood Clotting Test (20WBCT).</td>
<td>13</td>
</tr>
<tr>
<td>Management of Snakebite</td>
<td>14</td>
</tr>
<tr>
<td>Important don’ts</td>
<td>14</td>
</tr>
<tr>
<td>Patient Assessment</td>
<td>14</td>
</tr>
<tr>
<td>Management of Pain</td>
<td>15</td>
</tr>
<tr>
<td>Handling Tourniquets</td>
<td>15</td>
</tr>
<tr>
<td>Management of Swelling</td>
<td>16</td>
</tr>
<tr>
<td>Anti-snake Venom Serum (AVS) Treatment</td>
<td>16</td>
</tr>
<tr>
<td>Criteria for Administration of AVS</td>
<td>17</td>
</tr>
<tr>
<td>Doses and administration</td>
<td>17</td>
</tr>
<tr>
<td>Signs of recovery</td>
<td>18</td>
</tr>
<tr>
<td>Repeat doses of AVS</td>
<td>18</td>
</tr>
<tr>
<td>Management of Neurotoxicity</td>
<td>18</td>
</tr>
<tr>
<td>Management of Hemotoxicity</td>
<td>20</td>
</tr>
<tr>
<td>General Management (Antibiotics &amp; Fluid)</td>
<td>23</td>
</tr>
<tr>
<td>Management of Adverse reactions to ASV</td>
<td>23</td>
</tr>
<tr>
<td>Discharge</td>
<td>24</td>
</tr>
<tr>
<td>Follow Up</td>
<td>24</td>
</tr>
<tr>
<td>Flow Chart</td>
<td>25</td>
</tr>
<tr>
<td>Snakebite management in Primary/ Community/ Bedded Health centers</td>
<td>26</td>
</tr>
<tr>
<td>Basic Minimal &amp; Essential Drugs/ Equipment profile for primary care</td>
<td>28</td>
</tr>
<tr>
<td>After snakebite occurs Do’s and Don’ts</td>
<td>29</td>
</tr>
<tr>
<td>Appendix</td>
<td>30</td>
</tr>
<tr>
<td>Clinical Situations for Snakebite Training Module</td>
<td>32</td>
</tr>
<tr>
<td>Management of the cases</td>
<td>34</td>
</tr>
<tr>
<td>MANAGEMNET OF COMMON POISONS IN A HOSPITAL</td>
<td>37</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>----</td>
</tr>
<tr>
<td>List of contributors</td>
<td>38</td>
</tr>
<tr>
<td>Learning Objectives</td>
<td>40</td>
</tr>
<tr>
<td>Management of poisons</td>
<td>42</td>
</tr>
<tr>
<td>Medicolegal Aspects of Poisoning</td>
<td>44</td>
</tr>
<tr>
<td>Poison Specific Management</td>
<td>47</td>
</tr>
<tr>
<td>Aluminium phosphide</td>
<td>47</td>
</tr>
<tr>
<td>Organophosphorus</td>
<td>49</td>
</tr>
<tr>
<td>Organochlorine</td>
<td>51</td>
</tr>
<tr>
<td>Paraquat</td>
<td>52</td>
</tr>
<tr>
<td>Corrosive ingestion</td>
<td>53</td>
</tr>
<tr>
<td>Mushroom</td>
<td>55</td>
</tr>
<tr>
<td>Kerosene</td>
<td>57</td>
</tr>
<tr>
<td>Benzodiazepine</td>
<td>58</td>
</tr>
<tr>
<td>Dhatura</td>
<td>58</td>
</tr>
<tr>
<td>Rodenticide</td>
<td>59</td>
</tr>
<tr>
<td>Copper sulphate</td>
<td>60</td>
</tr>
<tr>
<td>Alcohol</td>
<td>61</td>
</tr>
<tr>
<td>Methanol</td>
<td>62</td>
</tr>
<tr>
<td>Paracetamol</td>
<td>63</td>
</tr>
<tr>
<td>Inhalant fumes</td>
<td>63</td>
</tr>
<tr>
<td>Scorpion sting</td>
<td>64</td>
</tr>
<tr>
<td>Iron poisoning</td>
<td>65</td>
</tr>
<tr>
<td>Poisoning by Household insecticide</td>
<td>67</td>
</tr>
<tr>
<td>Ingestion of disc/ button battery</td>
<td>67</td>
</tr>
<tr>
<td>Naphthalene</td>
<td>68</td>
</tr>
<tr>
<td>Assessment of a person who has attempted suicide by poisoning</td>
<td>70</td>
</tr>
<tr>
<td>Case studies</td>
<td>73</td>
</tr>
</tbody>
</table>
Management of Snake Bite Cases
List of contributors

Professor (Dr.) Krisnagshu Roy
Director, Institute of Health & FW, Govt. of West Bengal

Professor (Dr.) Akhil Bandhu Biswas
Faculty Member (Community Medicine), IHFW, Govt. of West Bengal

Dr. Dayal Bandhu Majumder
Senior MO (Grade - II), Calcutta National Medical College, Kolkata - 14

Dr. Shibendu Ghosh
Assoc. Professor, Raipur Institute of Medical Science, Raipur, Chhattisgarh

Dr. Ajay Chakraborty
Director of Health Sciences, Govt. of West Bengal

Dr. Pritam Roy
WHO Coordinator (Neglected Tropical Diseases)

PH & CD Branch
Directorate of Health Science, Govt. of West Bengal
Snakebite is an acute life threatening time limiting medical emergency. It is a preventable public health hazard often faced by rural population in tropical and subtropical countries with heavy rainfall and humid climate. It may result in death or chronic disability in active younger people. Snake bite is a well-known occupational hazard amongst farmers, plantation workers, and other outdoor workers and results in much morbidity and mortality throughout the world. The WHO has declared Snakebite as a “Neglected Tropical Disease” in the year 2009. The WHO SEARO office in New Delhi has published Guideline for management of Venomous Snakebite cases in South East Asian countries; last updated in 2016. The Government of India also published revised National Guidelines in 2016.

There is a huge gap between the number of snakebite deaths reported from direct survey and official data. Only 7.23% snakebite deaths were officially reported. Accurate statistics of the incidence of snakebite and its morbidity and mortality throughout the world does not exist; however, it is certain to be higher than what is reported. This is because even today most of the victims initially approach traditional healers for treatment and many are not even registered in the hospital.

Only 22.19% of the snakebite victims attended the hospitals. Nearly 65.7% of the snakebite deaths were due to common krait bite, most of them occurring in the months of June to September. As more and more cases of Common Krait (CK) bite are being diagnosed, it is now proved that, snakebite is not restricted to some outdoor occupations only, anybody from any occupation and any age group can also become a victim of CK bite at home (indoor).

According to the Bureau of Health Intelligence, Govt. of India, only 185625 snakebite incidence & 1064 snakebite deaths were recorded in the country in 2016. West Bengal is one of the states of India reporting high Snakebite incidence besides Tamil Nadu, Maharashtra, Uttar Pradesh, and Kerala. As per records of Public Health Branch of Govt. of WB, Snakebite incidence for the years 2014, 2015, 2016 and 2017 are 20975, 27754, 28265 & 29885 while reported deaths of snakebite cases for the years 2014, 2015, 2016 and 2017 are 170, 210, 213 & 240 respectively. Over the years, there is an increasing trend of both cases & deaths in West Bengal.

Learning objectives

- To identify snakebite cases and manage them accordingly following International & National Guidelines of Snakebite Management.

Epidemiology of Snakebite

As a Tropical disease, Snakebite has Snakes as Agent and Human being as Host. Snakes are found in most countries of the world except in the North and South poles and some small islands. More than 250 species of Snakes are found in India; of which around 60 species are venomous. Out of these 60 venomous species, about 50 species are Sea snakes which cause very little bite accidents.

In India, four species are responsible for 99% of the venomous bites; they are called “Big Fours”. Big fours are, 1) Spectacle Cobra (Naja naja), 2) Russell’s Viper (Daboia russelli), 3) Common Krait (Bungarus caeruleus) and 4) Saw scaled Viper (Echis carinatus).
Though Saw scaled vipers cause 75% of the venomous snakebites in India (4), they are not found in WB. Besides the Big fours some other new venomous species are now being identified in India and West Bengal also. (Details of Clinically significant snakes of WB are given in “Classification of Snakebites” section).

As snakes are natural habitats of rural areas, more than 97% snakebites happen in the rural areas. Incidence of Snakebite depends on frequency of contact between snakes and human. Snakes are usually elusive and reclusive. Snakebites occur when human move to the habitat of snakes like paddy field, tea, rubber and coffee plantations, bushes for open field latrine, and besides the water bodies during fishing. Bites may be inflicted at home by peri-domestic species which lives in roof space or under floor like cobras and Common Kraits (CK).

Seasonal peak of snakebite is noted in summer and rainy seasons. Increase in agricultural activity or heavy rain leading to flooding of the natural habitats of snakes increase the chance of snake human contact. During flood there may be epidemic of snakebite. Males are bitten more than females as outdoor activity after sunset is predominantly done by males. Peak age of bite is 15 – 45 years. Most of the snakebites are inflicted on feet and ankles.

Walking bare foot or wearing only sandal either in dark or in undergrowth increases chance of snakebite. Bites occur with Common Kraits when they come to homes for prey or probably by attraction of human sweat, and someone sleeping on floor rolls over the snake.

In recent years, few cases of Snakebite and fatality were recorded in unscientific and casual handlers of venomous snakes by snake rescuers and snake charmers.

---

**CLASSIFICATION OF SNAKE BITES**

- **Venomous snake Bites**
  - Neurotoxic
    - Cobras
  - Haemotoxic
    - Kraits
  - Myotoxic
    - Russell’s Viper (some neurotoxicity with viper’s Haemotoxic venom).
  - Pure haemotoxic
    - a) Humpnose Pit
    - b) Saw-scaled viper

- **Non venomous Bites**
SNAKE BITE: WEST BENGAL 2017

Cases Reported per lakh Population

- 0 to 9 Cases
- 10 to 29 Cases
- 30 to 59 Cases
- ≥ 60 Cases

Number of Deaths per 1000 Cases Reported

- 0 to 9 Cases
- 10 to 19 Cases
- ≥ 20 Cases
NEUROTOXIC

Cobras:

i) Spectacle Cobra (Naja naja), local names: Gokhro, Kharish, Goma.

ii) Indian Monocled Cobra (Naja kaouthia); local names: Keute, Samukhbhanga.

iii) King Cobra (Ophiophagus hannah). Bengali name Sankhachur.

Kraits:

i) Common Krait (Bungarus caeruleus); Local names: Kalach, Kalachiti, Domnachiti, Seorchanda.

ii) Banded Krait (Bungarus fasciatus); Bengali Sankhamuti.

iii) Black Krait (Bungarus niger).

iv) Wall’s Sind Krait (Bungarus walli).

Coral Snakes (Calliophis maculiceps): Nonhooded, cobra like venom. (Not found in WB).
HEMOTOXIC

Rusell’s Viper (*Daboia russelii*); Bengali Chandrabora. mainly hemotoxic, with some neurotoxic venom.

Saw Scalled Viper (*Echis carinatus*)
Pure Haemotoxic (Bengali name Fursha; very rare in W B).

Pit Vipers

i) **Humpnose Pit viper** (*Hypnale hypnale*): Pure Hemotoxic (Only in Western Ghats; Kerala and TN).

ii) **Green Pit vipers** (*Trimeresurus gramineus*): mild venom, causes local swelling only. (Gechhobora).

iii) **Mountain Pit Viper** (*Ovophis monticola*); found in Darjeeling Hills of WB. (Gurbe).

MYOTOXIC: All flat tail Sea snakes.

Common Nonvenomous snakes of WB:

Non Snakes: (These are called as snakes; but they are lizards)

1) Go Saap (Monitor Lizard), 2) Takshak (Chamellion).

Some patterns are helpful in identifying the types of snake, like the longitudinal rows of large, dark-rimmed, pale-centered spots are indicative of the Russell’s vipers or mountain pit viper snakes—commonly hemotoxic. The alternating black and yellow/white circumferential bands of the body are indicative of Kraits—commonly neurotoxic. Flat tails are usually sea snakes. It is to be noted that there are a few variations and exceptions to these.

However, it is stated that identification of type of snake only by appearance can be misleading and is not an essential step in snake bite management. Current guidelines do not promote killing of snake nor bringing dead or alive snake into health facility.

Death from snake-bite

Contributing factors

Factors identified as contributing to a fatal outcome included problems with antivenom use, delayed hospital treatment resulting from prolonged visits to traditional healers and problems with transportation, death on the way to hospital, inadequate artificial ventilation or failure to attempt such treatment, failure to treat hypovolaemia in shocked patients, airway obstruction, complicating infections, and failure to observe patients closely after they were admitted to hospital.

Time between snake-bite and death

Although very rapid death after snake-bite has rarely been reported (e.g. “a few minutes” after a bite by the king cobra), it is clear from studies of large series of snake-bite deaths that many hours usually elapse between bite and death in the case of elapid envenoming, and several days in the case of viper envenoming. Usually all the cases of death within one hour of snakebite were not directly due to venom effect, but probably due to some associated diseases like old heart diseases.

First Aid Treatment

In view of the limitations both tourniquets and ‘Pressure Immobilization Method’ (PIM) are rejected for use in India. PIM requires a skilled medical or paramedical person to be present at the site of accident which is rarely possible. The first aid recommended is based around the mnemonic: “Do it R.I.G.H.T.” It consists of:

- **R. = Reassure.** This is vital. Whenever and whatever snake bites a person, he/she becomes panicked. This panic may lead to a cardiac attack also. If the patient gets panicked his heart rate would increase which in turn would spread the venom rapidly. Try to reassure the patient. Tell him that seventy per cent of all snakebites are from non-venomous species. Only 50% of bites by venomous species actually envenomate the patient.
• **I. = Immobilize.** Immobilize the bitten limb in the same way as a fractured limb. Use bandages or cloth to hold the splints, not to block the blood supply or apply pressure. Do not apply any compression in the form of tight ligatures, they do not work and can be dangerous particularly in case of Russell’s Viper bite. If the bite is on the trunk, carry the patient in supine position on a stretcher or country cot. Children can be carried on shoulder.

• **G.H. = Go to Hospital immediately.** There is no alternative. Traditional remedies have NO benefit in treating snakebite. Most of the vital time is lost at the chamber / house of traditional healers. Refer the case to a health centre / hospital where AVS is available. For rapid transport in rural areas “Motor bike Ambulance” is ideal.

• **T – Tell the doctor** of any progress/new symptoms such as ptosis that manifest on the way to hospital.

Diagnosis of Snakebite & Envenomation

1.1.1. Bite Mark

A bite from a venomous snake may show one or more punctures, a small abrasion and perhaps a linear laceration. **Bite marks to determine whether the biting species was venomous or non venomous are of no use.** The pattern of fang marks is, however, of no help in ascertaining the amount of venom injected, severity of systemic poisoning and nature of poisoning – Elapidae or Viperidae venom. Very fine bite marks of Common Krait snakes are almost invisible (particularly in dark complexions). So, searching for bite marks in a case of CK bite is always misleading.
1.1.2. General signs and symptoms of Viper envenomation (Hemotoxic):

- Swelling and local pain.
- Tender enlargement of local lymph nodes (as large molecular weight Viper venom enter the system via the lymphatics).
- Bleeding from the gum and other orifices.
- Epistaxis
- Vomiting (may be blood stained or not).
- Acute abdominal pain (which may suggest gastro-intestinal or retro peritoneal bleeding).
- Hypotension (resulting from hypovolaemia or direct vasodilation).
- Low back pain, indicative of an early renal failure or retroperitoneal bleeding, (although this must be carefully investigated as many rural workers involved in picking activities complain of back pain generally).
- The skin and mucous membranes may show evidence of petechiae, purpura, and ecchymosis.
- The passing of reddish or dark-brown urine or declining or no urine output.
- Lateralising neurological symptoms and asymmetrical pupils may be indicative of intra-cranial bleeding.
- Parotid swelling, conjunctival oedema, sub-conjunctival haemorrhage
1.1.3. General signs and symptoms of Neurotoxic envenomation:

- Descending paralysis, initially of muscles innervated by the cranial nerves, commencing with *ptosis*, inability to maintain upward gaze, diplopia, or ophthalmoplegia. The patient complains difficulty in focusing and the eyelids feel heavy.

- Progressive swelling and local pain (Cobra).

- Local necrosis and / or blistering (Cobra).

- Paralysis of jaw and tongue may lead to upper airway obstruction and aspiration of pooled secretions because of the patient’s inability to swallow (pharyngeal palsy).

- Stomach pain suggesting submucosal haemorrhage in the stomach (Krait).

- Krait bites often present in the early morning with paralysis that can be mistaken for a stroke.

- Early morning “Pain Abdomen” is the commonest presentation in Krait bite.

- Numbness around the lips and mouth, progressing to pooling of secretions, bulbar paralysis and respiratory failure.

- Hypoxia due to inadequate ventilation can cause cyanosis, altered sensorium and coma. This is a life threatening situation and needs urgent intervention.

- Paradoxical respiration, as a result of the intercostals muscles becoming paralysed is a frequent sign.

Neuroparalytic snakebite patients present with typical symptoms within 30 min– 2 hours in case of Cobra bite and 3 – 24 hours for Krait bite; however, *ptosis in Krait bite have been recorded as late as 36 hours after hospitalization.*
These symptoms can be remembered as 5 Ds and 2 Ps.

- 5 Ds – dyspnea, dysphonia, dysarthria, diplopia, dysphagia
- 2 Ps – ptosis, paralysis

In chronological order of appearance of symptoms

Furrowing of forehead, Ptosis (drooping of eyelids) occurs first, followed by Diplopia (double vision), then Dysarthria (speech difficulty), then Dysphonia (pitch of voice becomes less) followed by Dyspnoea (breathlessness) and Dysphagia (inability to swallow) occurs.

All these symptoms are related to 3rd, 4th, 6th and lower cranial nerve paralysis. Finally, paralysis of intercostal and skeletal muscles occurs in descending manner.

Other signs of impending respiratory failure are diminished or absent deep tendon reflexes and head lag.

Additional features like stridor, ataxia may also be seen. Associated hypertension and tachycardia may be present due to hypoxia.

Bilateral dilated, poorly or a non-reacting pupil is not the sign of brain death in elapid envenoming.

Late-onset envenoming

The patient should be kept under close observation for at least 24 hours. Many species, particularly the Krait and the Hump-nosed pitviper (not present in WB, only present in Western Ghat), are known for the length of time it can take for symptoms to manifest. Often this can take between 6 to 12 hours. Late onset envenoming is a well-documented occurrence. [N.B. Bilateral ptosis was noted after 42 hours of admission with sore throat in a krait bite patient, admitted in the ENT dept. of Aliporeduar SD Hospital in 2013]

Clinical features of a compartmental syndrome (mostly in Russel viper bite)

Compartment syndrome is diagnosed with 5 ‘P’ –

- Pain (severe) on passive movement
- Pallor
- Paraesthesia
- Pulselessness
- Paralysis or weakness of compartment muscle.

Signs & symptoms of Snake bite

<table>
<thead>
<tr>
<th>Features</th>
<th>Cobras</th>
<th>Kraits</th>
<th>Russel’s viper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local progressive pain/ tissue damage</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Ptosis/ neurological sign</td>
<td>Yes (early)</td>
<td>Yes (late)</td>
<td>Rare</td>
</tr>
<tr>
<td>Hemostatic abnormality</td>
<td>No</td>
<td>In black krait</td>
<td>Yes</td>
</tr>
<tr>
<td>Renal complication</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Response to neostigmine</td>
<td>Yes</td>
<td>+ / -</td>
<td>No</td>
</tr>
<tr>
<td>Response to ASV</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>
1.2. Diagnosis and testing

- Carry out a **simple medical assessment** including history and simple physical examination – local swelling, painful tender and enlarged local lymph glands, persistent bleeding from the bite wound, bleeding (gums, nose, vomit, stool or urine), level of consciousness, drooping eyelids (ptosis) and other signs of paralysis.

- The Glasgow Coma scale cannot be used to assess the level of consciousness of patients paralyzed by neurotoxic venoms.

- **Monitor the patient closely** and repeat all above, every 1-2 hourly.

- Check for and monitor the following: **Pulse rate, respiratory rate, blood pressure and 20 minutes Whole Blood Clotting Test (20 WBCT)** every hour for first 3 hours and every 4 hours for remaining 24 hours.

- Check distal pulses and monitor if there is presence of gross swelling. The presence of a pulse does not rule out compartment syndrome. Pain on passive movement, pallor, pulseless limb, hypoesthesia over the sensory nerve passing through the compartment is **suggestive of compartment syndrome**.

- Severe local symptoms are defined as swelling rapidly crossing a joint or involving half the bitten limb, in the absence of a tourniquet. Once the tourniquet has been removed for more than one hour, if the swelling rapidly continues, this should be viewed as venom generated and not due to the continuing effect of the tourniquet. **Progressive local swelling is the commonest sign of envenomation**. There would be local pain along with swelling [Particularly in case of Russell’s Viper and Cobra bites].

- **Neurological signs and symptoms** are Ptosis, hoarseness of voice (due to pharyngeal and palatal palsy, then progressing to respiratory failure (in both Cobras and Kraits).

- In case of viper bites [haemotoxic like Russell’s Viper or ‘Chandrabora’] in addition to local pain and swelling there would be signs of coagulopathy. If you suspect coagulopathy, do not wait for red colouration of urine, but do the **20 Minute Whole Blood Clotting Test (20 WBCT)** which is adopted as the standard test.
1.3 20 Minute Whole Blood Clotting Test (20 WBCT)

20 WBCT is simple to carry out but essentially requires a clean and new dry glass test tube or glass vial (Must be glass not Plastic). If the vessel used for the test is not made of ordinary glass, or if it has been cleaned with detergent, its wall may not stimulate clotting of the blood sample (surface activation of factor XI – Hageman factor) and test will be invalid.

Draw 2-3 ml of venous blood. Keep this fresh blood in a dry test tube left undisturbed at ambient temperature for 20 minutes [cf. normal clotting time is 8 min maximum] and then gently tilt the tube.

If the blood is still liquid (not clotted) this is evidence of coagulopathy and confirms that the biting species is a Viper. Cobras or Kraits do not cause anti-hemostatic symptoms.

If cobra bite is not surely proved and first blood test is “clotted” the test should be carried out every hourly for four times; after that, if incoagulable blood is discovered, the 6 hourly cycles is then adopted to test for the requirement for repeat doses of ASV.

In coagulopathy, there may be continuous oozing from bite site, gum or old ulcers. Then lead to hemoptysis and hematuria and ultimately renal failure. (In Chandrabora bite there would be Ptosis also).

2. Management of Snakebite:

The following general principles are to be followed:

- Admit all cases with history of bites (Snake or unknown). All patients will be kept under observation for a minimum of 24 hours.
- Deal with any life threatening symptoms on presentation i.e. Airway, Breathing and Circulation.
- Closely observe for any sign of local or Systemic envenomation. In 50% of known venomous snake bite there may not be any envenomation (called dry bite). If there are no indications for administration of AVS, continue the general treatment (plain drip) for 24 hours.
The following local treatments are contraindicated:

- Washing
- Antiseptics.
- Incision.
- Suction.
- Electrotherapy or Cryotherapy.
- Venom Stone.

Important don’ts

1. Do not attempt to kill or catch the snake as this may be dangerous and not essential for management.

2. Do not wash wound and interfere with the bite wound (incisions, suction, rubbing, tattooing, vigorous cleaning, massage, application of herbs or chemicals, cryotherapy, cautery) as this may introduce infection, increase the flow of venom into system by stimulating lymphatic system, increase absorption of the venom and increase local bleeding.

3. Do not apply or inject antivenom venom (ASV) locally.

4. Do not tie tourniquets as it may increase risk of ischemia and gangrenous limbs; increase risk of embolism if used in viper bite.

5. Cutting the biting site in a victim with incoagulable blood increases the risk of severe bleeding as the clotting mechanism is no longer effective and increases the risk of infection. No venom is removed by this.

6. Electrotherapy and cryotherapy should be avoided.

2.1 Patient Assessment

- Where possible identify the snake responsible (not essential).
- Determine if any traditional medicines have been used; they can sometimes cause confusing symptoms.
- Take history of the exact time of the bite. This can be given indications as to the progression of any symptoms.
- Ask questions as to what the victim was doing at the time of the bite. Some activities such as grass cutting or feeding stock animals in the evening can be suggestive of snakebite. Sleeping in the floor at night is also important.

2.2 Management of Pain

- Snakebite can often cause severe pain at the bite site. This can be treated with analgesics such as paracetamol only.
- Aspirin should not be used due to its adverse impact on coagulation. Do not use non steroidal anti-inflammatory drugs (NSAIDs) as they can cause bleeding. This can be particularly dangerous in a patient already having coagulopathy.
• Mild opiates such as Tramadol, 50 mg can be used orally for relief of severe pain. In cases of severe pain at a tertiary centre, Tramadol can be given IV.

• In scorpion bite there would be much pain but almost no swelling.

2.3 Handling Tourniquets

• Never remove tourniquet in emergency room.

• Before removal of the tourniquet, test for the presence of a pulse distal to the tourniquet. Care must be taken when removing tight tourniquets. Sudden removal can lead to a massive surge of venom leading to neurological paralysis, hypotension due to vasodilation etc. Be prepared to handle the complications such as sudden respiratory distress or hypotension. If the tourniquet has occluded the distal pulse, then a blood pressure cuff can be applied to reduce the pressure slowly.

• Pro-coagulant enzymes will cause clotting in distal blood. In addition, the effect of the venom is causing vasodilation presents the danger of massive hypotension when the tourniquet is released.

2.4 Management of Sweling

• Persistent moderate swelling of the limb after viper bite can be successfully managed by repeated Magnesium Sulphate Compresses (in the layers of wet bandage, changed 2 to 3 times a day for 5 to 7 days).

2.5 Anti-snake Venom Serum (AVS) Treatment:

10 vials of AVS dissolved in 100 ml of distilled water and added to 400ml of normal saline. 

Mention date and time of starting infusion.
All including children must get 10 vials of AVS. Each vial of AVS to be dissolved in 10 ml of distilled water and added to an infusion medium such as normal saline (i.e. 10 vials of AVS dissolved in 100 ml of distilled water and added to 400 ml of normal saline; running fluid amount to be reduced in children).

The volume of infusion is reduced according to the body size and the state of hydration of the patient. In oliguric patients restrict fluids and use infusion pump to give full dose of AVS over 30 minutes.

**Pregnant women** are treated in exactly the same way as other victims. The same dosage of AVS is given. Refer the victim to a gynecologist for assessment of any impact on the foetus.

**Children** also are given exactly the same dose of AVS as adults as snakes inject the same amount of venom into children and adult. Liquid or reconstituted AVS is diluted in 5-10 ml/kg body weight of normal saline. However, reduce amount of fluid in running bottle to 200 ml to avoid fluid over load in children.

**ASV dosage in victims requiring lifesaving surgery:** Rarely patient may develop intracranial bleeding for which a lifesaving surgery is required. In such cases before surgery coagulation must be restored to avoid catastrophic bleeding and higher initial dose of AVS (up to 30 vials) can be administered.

### Criteria for Administration of AVS

- AVS should be used with evidence of systemic envenomation or severe progressive local swelling.
- Essentially systemic envenomation will be evident from the 20WBCT or signs of spontaneous bleeding in Viper bite, or by visual recognition of neurological impairment such as ptosis.
- Evidence of coagulopathy- detected by 20WBCT or visible spontaneous abnormal bleeding from gums, bite sites, injection sites, etc.
- Evidence of neurotoxicity- ptosis, external ophthalmoplegia, muscle paralysis, inability to lift the head, etc.

### Doses and administration

**Neurotoxic/ Anti Haemostatic: 10 vials.**

The initial dose is 10 vials for both adults and children as AVS is targeted at neutralizing the venom. Snakes inject the same amount of venom into adults and children.

First of all keep in hand one ampoule of Inj. Adrenaline. **Give 0.25 ML Inj. Adrenaline Subcutaneously as premedication** and keep 0.5 ml aside ready.

**All AVS to be administered over 1 hour at constant speed.** Add 10 vials of Indian Polyvalent Anti Snake Venom Serum (AVS) to the running bottle (200 ml in children) on earliest sign of envenomation. Open the fluid in jet and try to infuse 10 vials AVS in 1st hour. Closely observe for any adverse reaction to AVS, if any, treat accordingly. Pregnant women are treated in exactly the same way as other victims.
Points to note:

- ASV should be administered over one hour. There is no benefit in administering each dose over longer periods.
- To prevent volume overload undiluted ASV can be administered in oliguric or anuric patients.
- The patient should be closely monitored by treating doctor for 2 hours, then by nursing staff or other paramedical staff.
- ASV must NEVER be given by the IM route because of poor bioavailability by this route. Also do NOT inject the ASV locally at the bite site since it is not effective, is extremely painful and may increase intra-compartmental pressure. Take all aseptic precautions before starting ASV to prevent any pyrogenic reaction.

Signs of recovery

If an adequate dose of AVS has been administered, the following responses may be seen:

- Spontaneous systemic bleeding such as gum bleeding usually stops within 15 – 30 minutes.
- Blood coagulability is usually restored in 6 hours (Principal test is 20WBCT).
- Post synaptic neurotoxic envenoming such as the Cobra may begin to improve as early as 30 minutes after AVS, but can take several hours.
- Presynaptic neurotoxic envenoming of Krait bite usually takes a considerable time to improve.
- In shocked patients, blood pressure may increase after 30 minutes.

Repeat doses of AVS

In Viper bites, once the initial dose has been administered over one hour, no further AVS is given for 6 hours. If there is no active abnormal bleeding, 20WBCT test every 6 hours, will determine if additional AVS is required. This reflects the period the liver requires restoring clotting factors. If clotting defect is present, there will be active abnormal bleeding after one hour of 1st dose. Then repeat 2nd dose of 10 vials immediately.

- In viper bites maximum 30 vials of AVS may be needed.

Monitoring:

- Pulse rate, respiratory rate, blood pressure every hour.
- Blood urea, creatinine, and WBC count; potassium level if facility available (in Viper bite).
- Urine output, urine for RBCs (in Viper bite).
- Vomiting, diarrhea, abnormal bleeding.
- Extent of local swelling and necrosis.
- 20WBCT at Referral Hospital (after 6 hours of 2nd dose of 10 vials).

2.6.1 Management of Neurotoxicity

Neostigmine is an anticholinesterase that prolongs the life of acetylcholine and can therefore reverse respiratory failure and neurotoxic symptoms. It is particularly effective for post-synaptic neurotoxins such as those of the Cobra. There is some doubt over its usefulness against the pre-synaptic neurotoxin such as those of the Krait and the Russell’s Viper.
In the case of neurotoxic bites, once the first dose of AVS has been administered, and a Neostigmine test given, the victim is closely monitored. A Neostigmine test is administered using 1.5mg of Neostigmine IM. *Inj. Atropine 0.6mg IV must be given before Neostigmine.* Inj. Myopyrolate (Neostigmine + Glycopyrolate) is an easily available alternative; one shot of injection is to be given @ 1 ml IV for 10 Kg of body weight. The paediatric neostigmine dose is 0.04mg/kg IM and the dose of atropine is 0.05 mg/kg.

Observe the patient closely for 1 hour to determine if the neostigmine is effective. **After 30 minutes, any improvement should be visible by an improvement in ptosis.** Positive response to “AN” trial is measured as 50% or more recovery of the ptosis in one hour. **First sign of improvement is ability to open the eyes (Ptosis improves).**

If the response (AN trial) is positive, **repeat dose of neostigmine 0.5 mg IM every 30 minutes for 5 doses (In children, repeat dose of Inj. Neostigmine 0.01 mg/kg every 30 minutes for 5 doses) with 0.6 mg of atropine IV over an 8 hour period by continuous infusion.**

The following measures are useful objective methods to assess response of AN trial:

1. Single breath count
2. Uncovered area of iris measured in mm
3. Inter incisor distance (measured distance between the upper and lower incisors)
4. Length of time upward gaze can be maintained
5. FEV1 or FVC (if available)

Stop Atropine neostigmine (AN) dosage schedule if:

- Patient has complete recovery from neuroparalysis. Rarely patient can have recurrence, carefully watch patients for recurrence.
- Patient shows side effects in the form of fasciculations or bradycardia (these are signs of neostigmine overdose; give 0.3 mg atropine IV stat).
- If there is no improvement after 3 doses.

**Improvement by atropine neostigmine indicates Cobra bite. Give one dose of “AN” injection before transferring to the higher center** as half hourly regimen is not practical during referral. Rapid deterioration of Cobra bite neurotoxic syndrome may kill the patient on the way to transfer.

If after 1 hour the victim has not improved or has worsened then a second and final dose (of Both Atropine and Neostigmine) should be given. If no improvement occurs even after 2nd dose of Atropine and Neostigmine, the patient will require mechanical ventilation. Repeat 2nd dose of 10 vials of AVS if neurodeficit remains even after 2nd injection of Atropine and Neostigmine.

**Injection Calcium Gluconate in Krait bites**

If there is no improvement after repeat doses of atropine neostigmine (after 1 h), this indicates probable Krait bite. Krait affects pre-synaptic fibers where calcium ion acts as neurotransmitter. **Give Inj. Calcium gluconate 10ml IV (in children 1-2 ml/kg (1:1 dilution) slowly over 5-10 min every 6 hourly for next 24 hours.** Usually neuroparalysis recovers within 5-7 days.
Whenever there is non-response of neurotoxic features, the patient should be advised for mechanical ventilation support.

<table>
<thead>
<tr>
<th>REFERRAL CRITERIA: Neurotoxic Envenomation</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Progressive neuroparalysis - transfer with life support in ambulance for mechanical ventilation. Whilst it is entirely possible to maintain a neurotoxic victim by simply using a resuscitation bag, this should always be used as a last resort; the ideal means of support remains a mechanical ventilator (<em>Battery operated Transport Ventilator</em>) operated by qualified staff.</td>
</tr>
<tr>
<td>➢ PHC and even many referral hospitals are not equipped with mechanical ventilators. The most important factor, therefore, is when to refer a patient to a hospital with a ventilator.</td>
</tr>
<tr>
<td>o The key criteria to determine whether respiratory failure, requiring mechanical ventilation is likely, is the ‘neck lift’ to elicit broken neck sign.</td>
</tr>
<tr>
<td>o Neurotoxic patients should be frequently checked on their ability to perform a neck lift. If they are able to carry out the action then treatment should continue until recovery in the BPHC.</td>
</tr>
<tr>
<td>o Neck lift test is also useful for children except very young children who may not be able to follow commands.</td>
</tr>
<tr>
<td>o Other tests which indicate descending paralysis are declining single breath count, pooling of saliva.</td>
</tr>
<tr>
<td>o If the patient reaches the stage when patient cannot do neck lift, immediately refer the patient to a hospital with a mechanical ventilator.</td>
</tr>
<tr>
<td>➢ Maintain oxygen saturation using Pulse oximetry. Oxygen saturation &lt;90% patient indicates requirement for ventilator support.</td>
</tr>
</tbody>
</table>

*Remember- Antivenom treatment alone cannot be relied upon to save the life of a patient with bulbar and respiratory paralysis. Death may result from aspiration, airway obstruction or respiratory failure.*

### 2.6.2 Management of Hemotoxicity

If there is definite history of viper bites or signs of abnormal bleeding or 20WBCT test comes ‘not clotted’ indicating coagulopathy, refer the case to a Hospital having facility of kidney function test/ dialysis after giving initial dose of 10 vials of AVS.

Repeat another 10 vials of AVS in fluid in jet if active bleeding persists (this should be done before referral).

If required, repeat 20 WBCT test and AVS after 6 hours. After 6 hours of receiving 2nd dose of 10 vials of AVS, repeat 20 WBCT. If the result comes “not clotted”, repeat 10 vials of AVS in fluid in jet. No AVS after 30 vials. If haematuria is present or Kidney function test is abnormal, refer the patient for Dialysis. Discharge patient when Kidney function test is normal.

If viper bite is strongly suspected but 20WBCT comes ‘clotted’, then repeat 20WBCT every hourly for next 4 hours as it may come ‘clotted’ late. If in between test result comes ‘not clotted’, repeat 2nd dose of 10 vials of AVS.
Renal failure, a common complication is contributed by intravascular haemolysis, DIC, direct nephrotoxicity, hypotension and rhabdomyolysis.

Renal damage can develop very early in Russell viper bite and even when patient arrives at hospital soon after bite. Even when ASV is administered within 1-2 hours after bite, it was incapable of preventing ARF.

The following are indications of renal failure:

- Declining or no urine output although not all cases of renal failure exhibits oliguria.
- Blood testing
  - Serum creatinine > 5gm/dl or rise of > 1 mg/day
  - Urea > 200 mg/dl
  - Potassium > 5.6 mmol/l (Confirm hyperkalemia with EKG)
  - Evidence of uremia or metabolic acidosis

Management of renal failure:

- Early intervention with early initiation of ASV
- Control of hypotension
- Control of coagulopathy
- Hemodialysis
- Control of hyperkalemia

Declining renal parameters require referral to specialist nephrologist with access to dialysis facility.

<table>
<thead>
<tr>
<th>REFERRAL CRITERIA: Vasculotoxic envenomation</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ If no ASV is available, transfer to a hospital (where ASV availability is confirmed over the phone).</td>
</tr>
<tr>
<td>➢ If 20 WBCT is “not clotted” after loading dose of 10 vials of ASV as in case of Viper bite.</td>
</tr>
<tr>
<td>➢ If patient is continuing to bleed even after full dose of ASV, transfer to a tertiary care medical college or higher level of health facility.</td>
</tr>
<tr>
<td>➢ Signs of kidney injury or abnormal kidney function test. Transfer to a tertiary care medical college or higher level of health facility having dialysis facility.</td>
</tr>
<tr>
<td>➢ Compartment syndrome</td>
</tr>
<tr>
<td>➢ Progressive septicemia</td>
</tr>
</tbody>
</table>

**Forced Alkaline Diuresis** (in referral hospital)

If the patient has oliguria or dipstick positive for blood give a trial of forced alkaline diuresis (FAD) within first 24 hours of the bite to avoid pigment nephropathy leading to acute tubular necrosis (ATN).

Delayed FAD has no role.
Sequence of FAD in adults is as follows:

- Inj. Frusemide 40 mg IV stat
- Inj. Normal saline 500 ml + 20 ml of NaHCO₃ over 20 minutes
- Inj. Ringer’s lactate 500 ml + 20 ml of NaHCO₃ over 20 minutes
- Inj. 5% dextrose 500 ml + 10 ml of Potassium Chloride over 90 minutes
- Inj. Mannitol 150 ml over 20 min

Whole cycle completes in 2 h 30 min and urine output of 3 ml/min is expected.

If patient responds to first cycle, continue for 3 cycles. FAD converts oliguria into polyuria and avoid ATN and acute kidney injury needing dialysis in more than 75% patients. If there is no response to furosemide discontinue FAD and refer patient immediately to a higher center for dialysis.

**Indications for dialysis are:**

- Absolute value of Blood urea > 130 mg/dl (27 mmol/L) (BUN 100 mg/dl), Sr. Creatinine > 4 mg/dl (500 μmol/L) OR evidence of hypercatabolism in the form of daily rise in blood urea 30 mg/dL (BUN > 15), Sr. Creatinine > 1 mg/dL, Sr. Potassium > 1 mEq/L and fall in bicarbonate > 2 mmol/L
- Fluid overload leading to pulmonary oedema
- Hyperkalaemia (> 7 mmol/l (or hyperkalaemic ECG changes)
- Unresponsive to conservative management
- Uremic complications – encephalopathy, pericarditis.

**For coagulopathy –**

- In case of prolonged CT, PT, aPTT administer fresh frozen plasma (FFP) infusion. Associated low platelets indicates consumption coagulopathy and disseminated intravascular coagulopathy (DIC).
- To confirm fibrinogen level, FDP should be estimated. Low fibrinogen and high FDP will require fibrinogen/FFP supplementation.
- Bleeding leads to anaemia, PCV of 30% must be maintained. Therefore, measure serial PCV every 4 – 6 h depending upon severity of bleeding. If PCV is lower than 30, patient needs blood transfusion/PCV transfusion.
- Avoid intramuscular injections.
- FFP administration after ASV administration results in more rapid restoration of clotting function in most patients, but no decrease in discharge time. Early FFP administration (< 6-8 h) post-bite is less likely to be effective. Administer 10-15 ml/kg of FFP within over 30–60 min within 4 hours of ASV administration. The aim should be a return of coagulation function, as defined by an INR of < 2.0, at 6 h after ASV administration was commenced.
• Non–response to FFP can occur with use of FFP that has low activity of F V and F VIII, because of either poor storage or premature thawing (> 24 hours) prior to administration.

2.7. General Management (Antibiotics & Fluid)

• There are many factors that contribute to potential infection in snakebite, including poor or dangerous first aid, oral snake flora and environmental factors. Routine use of antibiotic is not necessary, although it should be considered if there is evidence of cellulitis or necrosis.

• Where wound infection is suspected give prophylactic broad-spectrum antimicrobial treatment for cellulitis after completion of first 10 vials of ASV with following.
  1. Inj. Amoxicillin + clavulanic acid 1.2 g IV thrice daily for first 7 days then switch to oral therapy Tab. Amoxicillin + clavulanic acid 625 mg three times a day for further 3-7 days; In children, the dose is 100 mg/Kg/day in three divided doses intravenously; for oral therapy, the dose is 50 mg/kg/day in three divided doses.
  2. Inj. Metronidazole 400 mg IV infusion thrice daily for 7 days; in children- 30 mg/kg/day in 3-4 divided doses.
  
  o Alternatively Inj Ceftriaxone 1 g IV twice daily (in children the dose is 100 mg/kg/day in two divided doses) for 7 days. Both Amoxicillin + clavulanic acid and Ceftriaxone are mainly excreted through Kidney. Therefore, in case of acute kidney injury in Viper bites dose of both these antibiotics should be reduced and adjusted according to renal function. Non-nephrotoxic antibiotics should be used (i.e. avoid aminoglycosides such as gentamicin).

• Persistent moderate swelling of the limb after viper bite can be successfully managed by systemic broad spectrum antibiotics and repeated Magnesium Sulphate compresses (in the layers of wet bandage, changed 2-3 times a day) for 5-7 days.

• Volume replacement by IV fluid: Normal saline / Ringer solution.

• In case where generalised capillary permeability has been established a vasoconstrictor such as dopamine can be used. Dose is 5-10 µgm/kg/minute

2.8 Management of Adverse reactions to ASV

Adverse reactions, either anaphylactoid or pyrogenic, have often been identified as reasons not to administer ASV in smaller local hospitals. The fear of these potentially life threatening reactions has caused reluctance amongst some doctors to treat snakebite. However, if handled early and with primary drug of choice, these reactions are easily surmountable and should not restrict doctors from treating snakebite. Early intervention against these kind of reactions has been shown to have more positive outcomes. Patients should be monitored closely as there is evidence that many anaphylactoid reactions go unnoticed.

Common signs of adverse reactions to AVS singly or in any combination are: Urticaria, Itching (particularly Scalp itching), Fever, Shaking chills, Vomiting, Diarrhea, Abdominal cramps, Tachycardia, Hypotension,
Bronchospasm and Angio-oedema. Any new sign or symptoms or unexplained uneasiness after ASV infusion should be taken as indication of reaction.

**Premedication:** With 0.25 ML Inj. Adrenaline S/C, most of the AVS reactions can be prevented.

At the first sign of any of the above mentioned signs,

i. Stop AVS drip temporarily for the time being and

ii. **Give 0.5 ml (0.5 mg of 1:1000) Adrenaline IM over deltoid or thigh.** The pediatric dose is 0.01 mg / kg body weight of Adrenaline IM.

iii. If after 10 to 15 minutes the patient’s condition has not improved or is worsening, a second dose of 0.5 mg of Adrenaline 1:1000 IM is given.

iv. Oxygen

v. Start fresh IV normal saline infusion with a new IV set

vi. 100 mg of hydrocortisone and an H1 antihistaminic, (Pheniramine maleate 22.5 mg IV or Promethazine HCl 25 mg IM, or 10 mg chlorpheniramine maleate IV.)

vii. The dose for children of Pheniramine maleate is 0.5 mg/kg/day IV. Promethazine HCl can be used at 0.3 – 0.5 mg/kg IM or 0.2 mg/kg of chlorpheniramine maleate IV and 2 mg/kg of hydrocortisone IV. Antihistaminics use in pediatric cases must be deployed with caution.

viii. Once the patient has recovered, re-start ASV slowly for 10-15 minutes keeping the patient under close observation. Then resume normal drip rate.

**Treatment of Late (serum sickness–type) reactions**

i. Inj. Chlorpheniramine 2 mg in adults (In children 0.25 mg/kg/day) 6 hourly for 5 days.

ii. In patients who fail to respond within 24–48 h give a 5-day course of Prednisolone (5 mg 6 hourly in adults and 0.7 mg/kg/day in divided doses in children).

2.9. **Discharge**

If no symptoms and signs develop after 24 hours, the patient can be discharged.

Keep the patient under observation for 48 hours if ASV was infused.

2.10 **Follow-up**

A snakebite victim discharged from the hospital should continue to be followed up.

At the time of discharge patient should be advised to return to the emergency, if there is worsening of symptoms or signs such as evidence of bleeding, worsening of pain and swelling at the site of bite, difficulty in breathing, altered sensorium, reduced or increased urine output etc. The patients should also be explained about the signs and symptoms of serum sickness (fever, joint pain, joint swelling) which may manifest after 5-10 days.
FLOW CHART FOR SNAKE BITE MANAGEMENT

Patient attending Emergency Room of any hospital; H/O Bite (Snake or Unknown)

No Referral in venomous snake bite before 10 vials AVS administration

- Respiration & Airway must be restored first of all.
  Use AMBU BAG SOS.

- Always admit and start IV fluid (NS / FNSD); Inj. Toxoid
  Remove any figure
  Rapidly assess for any crisis (Give attention to crisis first)

(0.25ML ADRENALINE S/C before AVS)

- Signs of Envenomation Present
  [Progressive local swelling and pain are sure signs of envenomation.]
  Add 10 vials of AVS in running fluid & Start in jet (less than 1 hr.)
  No Skin Test
  Dose of AVS is same for adult, children, pregnant women

- Neurological Signs Present
  [Ptosis, hoarseness of voice, choking throat are early Neuro Signs.]
  Inj. Atropine 1 amp (0.6mg) IV (must), then Inj. Neostigmine 3 ML (1.5mg) IM.
  Improvement: Neostigmine 0.5 mg IM - 5 doses every 30 min with 0.6 mg atropine IV infusion for 8 hours
  No improvement (in Krait & Viper bite)

- Scalp itching, Urticaria, fall of BP, Pain, Abdomen and vomiting are signs of AVS reaction.
  Restart AVS when ever reaction is controlled.

- If active bleeding persists after one hr, repeat 2nd 10 vials of AVS here & transfer

- If Haematuria present or kidney function abnormal.
  Transfer for Dialysis; No AVS after 30 Vials

- Transfer to Referral Hospital (2nd Hospital)
  (where lab facility for kidney function test & dialysis is present)
  After 6 hrs. of receiving 2nd dose of 10 vials of AVS → Repeat 2 WBCT
  2nd 20 WBCT = not clotted; Repeat 10 vials of AVS in fluid in jet.

- If Haematuria present or kidney function abnormal.
  Transfer for Dialysis; No AVS after 30 Vials

No Swelling in Scorpion bite but tremendous pain

In Krait bite local sign, only Neurological sign & may not be and bite mark, nor any H/O bite; H/O open floor bed is suggestive. May present with pain in throat, abdomen or joints

Definite H/O Viper bite, or signs of bleeding
[Bleeding gum, Haematuria, Blood stained stumtm or bleeding from bite mark or old ulcer]

Do 20 WBCT:  a) clotted → repeat 20WBCT every hourly for next 4 hours →
  if comes not clotted in between, follow below steps
  b) not clotted (after 20 min): viper bite confirmed

Drug chart for causing immediate effect

- Systemic antibiotic if cellulitis is evident

Essential drugs & equipment list

- AVS
- Neostigmine
- Tramadol
- Glass syringe
- Laryngeal tube

- Hydrocortisone
- Atropine
- Antibiotics
- Glass test tubes
- Ambu Bag with mask

- Antihistaminics Inj.
- Paracetamol
- NS / DS
- Oxygen Cylinder

Management of AVS reaction: discontinue AVS temporarily; 0.5mg (children 0.01 mg/kg) of 1.1000 adrenaline IV / IM;
  Adult: 100 mg hydrocortisone & H1 antihistaminics (10 mg chlorpheniramine maleate/ 22.5 mg pheniramine maleate)
  Children: 2mg/kg hydrocortisone & 0.1-0.3 mg/kg of antihistaminics IV

Other drugs according to body weight:
  Adrenaline: 0.01 mg/kg
  Neostigmine: 0.04 mg/kg
  Atropine: 0.05 mg/ Kg.
Snakebite management in Primary/ Community/ Bedded Health centers

**Patient arrival & assessment**

1. **Assess** Airway, Breathing, Circulation and deal with any life threatening symptoms on presentation.

2. Establish **large bore intravenous access** and start normal saline slow infusion. The first blood drawn from the patient should be typed and cross-matched, as the effects of both venom and ASV can interfere with later cross-matching.

3. Before removal of the tourniquet/ligatures, test for the presence of a pulse distal to the tourniquet. In case of **clinically confirmed venomous bite**, tourniquet should be removed only after starting of loading dose of ASV and keep Atropine Neostigmine injection ready. In case of multiple ligatures, all the ligatures can be released in Emergency Room EXCEPT the most proximal one; which should only be released after admission and all preparations.

4. Carry out a **simple medical assessment** including history and simple physical examination.

5. The snake, if brought, should be carefully examined and identified, if possible. (One smart phone photograph of the snake, dead or alive, if available, should be taken for confirmation by an expert). – **NOT AN ESSENTIAL STEP**.

6. **Clotting test ‘20WBCT’** to diagnose vasculotoxic envenomation. Report should be given as Clogged or Not Clogged. Never write Positive/negative.
   a. If clotted continue every 1 hour for the 1st 3 hours from the time of hospitalization and then 6 hourly for 24 hours.

7. Give analgesia

8. If the patient fulfils criteria for antivenom treatment, **give ASV**. If no ASV is available, transfer to a health facility where ASV is available.
   a. 0.25ml Adrenaline s/c before AVS [keep 0.5 ml ready in syringe]
   b. 10 vials of AVS dissolved in 100ml distilled water and added to 400 ml of NS in 1 hour [200 ml in children]

9. Manage according to suspect of Haemotoxic or Neurotoxic envenomation. After initial management other supportive therapy may be initiated.

<table>
<thead>
<tr>
<th>Suspect Haemotoxic envenomation</th>
<th>Suspect Neurotoxic envenomation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. If there is definite history of viper bites or signs of abnormal bleeding or 20WBCT test comes ‘not clotted’ indicating coagulopathy, refer the case to a Hospital having facility of kidney function test/ dialysis.</td>
<td>1. <strong>Neostigmine test (AN trial)</strong> is administered using Atropine and Neostigmine as per dosage described earlier.</td>
</tr>
<tr>
<td>2. Repeat another 10 vials of AVS in fluid in jet if active bleeding persists (this should be done before referral).</td>
<td>2. Observe the patient closely for 1 hour to determine if the neostigmine is effective.</td>
</tr>
<tr>
<td>1. A Neostigmine test (AN trial) is administered using Atropine and Neostigmine as per dosage described earlier.</td>
<td>a. If the response (AN trial) is positive, repeat dose of neostigmine 0.5 mg IM every 30 minutes for 5 doses with 0.6 mg of atropine IV over an 8 hour period by continuous infusion.</td>
</tr>
<tr>
<td>2. Observe the patient closely for 1 hour to determine if the neostigmine is effective.</td>
<td>b. If after 1 hour the victim has not improved or has worsened then a second and final dose (of Both Atropine and Neostigmine)</td>
</tr>
</tbody>
</table>
### REFERRAL CRITERIA:

| **Once AVS is finished and adverse reaction if any is dealt with.** | **Progressive neuroparalysis - transfer with life support in ambulance for mechanical ventilation.** |
| **Active bleeding persist** | **The key criteria to determine whether respiratory failure, requiring mechanical ventilation is likely, is the ‘neck lift’ to elicit broken neck sign.** |
| | **Other tests which indicate descending paralysis are declining single breath count, pooling of saliva.** |

The 6 hour rule ensures that a six hour window is now available in which to transport the patient. If haematuria present or kidney function test result is abnormal- may be transfer for dialysis.

The primary consideration is to be equipped to provide respiration support to the victim. Transfer the patient with a face mask, resuscitation bag and a person, other than the driver of the vehicle, who is trained of how to use these devices. If respiration fails then the victim must be given artificial respiration until arrival at the institute.

### Instructions while referring

- Give prior intimation to the receiving center using available communication facilities.
- Transport in an ambulance equipped with transport ventilator. If ventilator is not available, tight-fitting face mask connected to an anaesthetic (Ambu) bag should be available. However, do not waste time to get an ideal ambulance.
- If ASV is not available at First contact center transfer to the nearest health facility where ASV is available (confirmed by telephone).
- Transfer to a higher health facility (Secondary Care Hospital or Tertiary Care Hospital) where mechanical ventilator and dialysis facilities are available for ventilation and dialysis, if required **after completion of ASV infusion only**.
- During transfer, continue life-supporting measures, insert nasogastric tube and provide airway support with the help of an accompanying staff, if required.

### Points to be mentioned in Referral Note

| Site & time of bite | - Atropine & Neostigmine – doses & time, if given |
| Initial symptoms | - Supportive treatment given including blood |
| 20 WBCT result – initial & subsequent | - Condition at the time of referral and reason of referral |
| Total AVS dose administered with time of each dose |  |
### Basic Minimal & Essential Drugs/ Equipment profile for primary care

#### Drug:
- AV/ ASV (in domestic fridge if liquid)
- Adrenaline ∨ Neostigmine
- Atropine ∨ Hydrocortisone
- Antihistaminics (injectables)
- Analgesics
  - Paracetamol
  - Tramadol (both oral & injectable)
- NS bottles
- Antibiotics
  - Inj Amoxicillin + clavulanic acid
  - Inj Metronidazole
  - Inj Ceftriaxone
- Equipment
  - Syringes
  - IV set
  - Clean new Glass Test tubes
  - Blood pressure monitor
  - AMBU Bag with mask
- Other desirables
  - Oxygen
  - Laryngeal tube with laryngeal mask airway (LMA)
  - Nasopharyngeal Airways (these can be improvised using size 5 Endotracheal tubes cut to the required length)
Do’s and Don’ts after snakebite occurs

Do’s

– Call ambulance and transfer patient to a medical health facility. Arrange transport of the patient to medical care as quickly, safely and passively as possible by vehicle ambulance, boat, bicycle, motorbike, stretcher etc.
– Keep the person calm. Reassure them that bites can be effectively treated in an emergency room. Restrict movement.
– Remove any rings or constricting items, because the affected area may swell.
– Create a loose splint (it should be capable of inserting one finger beneath) to help restrict movement of the area.
– Ideally the patient should lie in the recovery position (prone, on the left side) with his/her airway protected to minimize the risk of aspiration of vomitus.
– If the area of the bite begins to swell and change colour, the snake was probably venomous.
– Monitor the person’s vital signs
  — temperature, pulse, rate of breathing, and blood pressure
  — if possible. If there are signs of shock (such as paleness), lay the person flat, raise the feet about a foot, and cover the person with a blanket.

Don’ts

– Do NOT waste time in traditional first aid methods
– Do NOT allow the person to become over-exerted. If necessary, carry the person to safety.
– Do NOT apply a tourniquet. Do NOT block the blood supply or apply pressure.
– Do NOT apply cold compresses to snakebite.
– Do NOT cut into snakebite with a knife or razor.
– Do NOT try to suck out the venom by mouth or wash the wound.
– Do NOT give the person anything by mouth.
– Do NOT attempt to kill or catch the snake as this may be dangerous. Bring in the dead snake only if this can be done safely.
– Do NOT waste time hunting for the snake, and do NOT risk another bite if it is not easy to kill the snake. Be careful of the head while transporting it - a snake can actually bite for several hours after it is dead (from a reflex).
Venomous snakes have two venom glands inside their mouth, which are modified salivary glands. These two venom glands are connected with long fangs (venom teeth). These teeth may be hollow like hypodermic needles (as in R Viper) or grooved (in Cobras and kraits). When a venomous snake bites; for hunting or for defence (most of the human bites), some amount of venom is injected into the bitten soft tissue. This venom is gradually absorbed from the deposit site mainly via lymphatics; in a small percentage of cases there may be direct venipuncture or intramuscular injection of the venom. Systemic signs of venom are noted after spread from the deposit site.

Fatal dose of venom varies from species to species. These are 42 mg in R Viper, 15 mg in Cobras (Gokhro and Keute), and only one milligram in Common Krait. Fatal dose of venom may not be injected in 50% of venomous snakebites due to different factors.

More than 90% (w/v) of the venom is different biologically active proteins. These proteins are different enzymes (more than hundred types), non-enzymatic polypeptide toxins, and non-toxic proteins such as nerve growth factor.

**Venom enzymes**

These include digestive hydrolases, hyaluronidase, and activators or inactivators of physiological processes. Zinc metalloproteinase haemorrhagins damage vascular endothelium, causing bleeding.

**Procoagulant enzymes:** Venoms of Vipers contain serine proteases and other procoagulant enzymes that are thrombin-like or activate factor X, prothrombin and other clotting factors.

These enzymes stimulate blood clotting with formation of fibrin in the blood stream. Paradoxically, this process results in incoagulable blood because most of the fibrin clot is broken down immediately by the body’s own plasmin fibrinolytic system and, sometimes within 30 minutes of the bite, the levels of clotting factors are so depleted (“consumption coagulopathy”) that the blood will not clot. Some venoms contain multiple anti-haemostatic factors.

For example, Russell’s viper venom contains toxins that activate factors V, X, IX and XIII, fibrinolysis, protein C, platelet aggregation, anticoagulation and haemorrhage.

**Phospholipase A2 (lecithinase):** It damages mitochondria, red blood cells, leucocytes, platelets, peripheral nerve endings, skeletal muscle, vascular endothelium, and other membranes, produces presynaptic neurotoxic activity, opiate-like sedative effects, leads to the autopharmacological release of histamine and anti-coagulation.

**Hyaluronidase:** Promotes the spread of venom through tissues.

Proteolytic enzymes and polypeptide cytotoxins (“cardiotoxins”): Increase vascular permeability causing oedema, blistering, bruising and necrosis at the site of the bite.
Venom polypeptide toxins ("neurotoxins")

Postsynaptic (α) neurotoxins such as α-bungarotoxin and cobrotoxin (in Cobras) bind to acetylcholine receptors at the motor endplate. Presynaptic (β) neurotoxins such as β-bungarotoxin (in Kraits) release acetylcholine at the nerve endings at neuromuscular junctions and then damage the endings, preventing further release of transmitter. Both presynaptic and postsynaptic blockage cause neuroparalytic signs and symptoms. Postsynaptic blockage only (in cobras) is benefitted by Inj. Neostigmine.

Nephrotoxicity:

Nephrotoxicity is a feature of Viper venoms, this is particularly noted in R Viper and Humpnose Pitviper (not found in WB) bites.

Direct enzymatic damage to the renal tubules is the main cause of renal toxicity. Hemoglobinuria, myoglobinuria (in sea snake bites) and deposition of high molecular weight proteins are other causes of oliguria and anuria. Hypovolemic shock is also a cause of oliguria.

Appendix II

Anti-snake Venom Serum (ASV)

Indian Polyvalent Anti Snake Venom Serum (AVS or ASV) is a unique solution to all type of venomous snakebite cases in India. It is called polyvalent as it can be used to treat many a types of snakebites. Species specific “Monovalent” AVS is not available in India. Indian AVS is prepared from horse (mule or donkey) serum. Four types of snakes are used to prepare this AVS. These snakes are 1) Indian Spectacle Cobra, 2) Russell’s Viper, 3) Common Krait and 4) Sawscaled Viper.

Sub lethal dose of venom of all these four snakes are injected to a horse (to hyper immunize). Antibody (Ig G) develops against these snake venoms in the blood of that horse. This horse blood (plasma) is then purified in the laboratories to prepare Polyvalent AVS. So, AVS is basically a horse protein, and has every chance to be rejected by our human body, as a foreign protein. This rejection is called “reaction”. Impurities in the AVS increase the chance of reaction during treatment of a snakebite case.

Prior sensitization to horse serum, as in case of previous use of anti rabies or anti tetanus immunoglobulin (equine) increases the chance of AVS reaction.

Hump nose pit vipers are quite exceptional in India. Their venom is not responsive to common AVS. As these snakes are limited to a small geographic area (Western Ghat hill areas), few laboratories prepare specific Polyvalent AVS including Pit viper snake venom along with other four snakes as mentioned earlier.

Indian Polyvalent AVS is available in 10 ML vials; both in liquid and lyophilized form. Liquid ones have to be kept in the domestic refrigerators (2-8 °C); Lyophilized AVS can be stored in room temperature ( below 25 °C ). No AVS can be freezed.

Lyophilized AVS is to be dissolved into 10 ml of water for injection just before mixing in the IV fluid for infusion. Usually Lyophilized AVS lasts for 5 yrs and liquid ones last for 2 – 3 yrs. Both must be discarded if any precipitate is noted before mixing in the IV fluid.
One vial of 10ml AVS is meant for neutralizing 6 mg of Russell’s Viper venom, 6mg of Cobra venom, 4.5 mg of Common Krait venom and 4.5mg of Saw scaled viper venom.

Indian polyvalent AVS has some cross sensitivity to other species of snakes also; Spectacle cobra venom AVS has good neutralizing effect on Monocled cobra and King cobra venom. Common Krait venom AVS can neutralize Banded Krait venom also. AVS for sea snake venom is not available in India. Some authorities recommend high dose of Indian polyvalent AVS for sea snake bite cases in India.

Many Indian laboratories prepare AVS for commercial use; all of them have to be standardized from the Central Research Laboratory of Govt. of India, situated at Kasauli, Himachal Pradesh.

Is SKIN Test before administering AVS recommended? — NO

AVS test doses have been abandoned. They have no predictive value in anaphylactoid or late serum reactions and may pre-sensitize the patient to the protein. This test is mostly false negative.

Clinical Situations for Snakebite Training Module

1) Case No. 1. A 35 year old male patient, Bikash Hazra was brought to Habra State General Hospital in North 24-Paraganas, at about 4 AM. On examination the patient was unconscious with flaccid limbs, no respiration notable. Pulse was normal. Relatives gave the history of suspected snake bite about 2 hours back while the pt. was sleeping on open floor outside his house. Snake was not seen by patient, but he told about severe pain in left arm. He could speak till 30 minutes before reaching hospital. No medical specialist was available at that time.

a) Can you rely upon the history of Snakebite?

b) Can you start treatment without identification of a snake?

c) Whether to try treating there or to refer to district Hospital 20 km away?

d) What treatment to start with?
e) How long to wait for improvement?

f) When to transfer?

g) If treated at S G Hospital, when to discharge?

2) Case No. 2. Barun Manna, 29 year old male patient, brought to Debra BPHC at 10 AM from a remote village. H/O Russell’s viper bite about an hour back. There were two bite marks near Rt. ankle with swelling. Moderate pain was complained by the patient. There was a tight ligature on Rt. thigh.

a) Try to manage there or to transfer to Midnapur Medical College 35 Km away?

b) What to do with the ligature?

c) Start AVS straight way or to do any investigation?

d) What investigation to do?

3) Case No. 3. A 13 year old boy Tapan Bag brought to one private nursing home at a small town of WB about 50 KM from Kolkata, at about 11 AM with severe convulsions. Pt. went to total respiratory failure within 5 minutes while doctors were examining the patient. Relatives gave history of sore throat at about 8 AM, it was followed by some tablets from a village quack. Complaint of blurring of vision and drooping of eye lids starting at about 8.45 AM. Convulsions started about half an hour before they reached the NH. There was no previous history of convulsions, no other H/O any previous illness.

a) What should be the diagnosis?

b) What other history is important?

c) How this pt. can be managed if attended Tamluk District Hospital?

d) Could you try artificial respiration? How?

e) What other medications can be tried?

f) When and where to transfer?

4) Case No. 4. A 12 year old boy, Jhantu Roy, was brought to Habra S G Hospital of WB at about 11 AM with H/O snakebite about two hours back. On examination the boy was conscious and cooperative. There were multiple tight ligatures starting from middle of the Rt. Leg to upper part of Rt. thigh. There were multiple scratch marks near the Rt. ankle. Part of the leg below the lowest ligature was swollen and there was no complaint of pain.

a) Should we wait for the snake to be brought?

b) What to do with the ligatures?

c) Any investigation to be done?

d) What treatment to be started?
e) How long to wait before referral?

f) When to discharge?

5. Case No. 5. Subhendu Ghosh, a 10 year old boy, was brought to Bishnupur S D Hospital of Bankura in the evening with history of snakebite 2 hrs back. One medium size R. Viper snake was killed and brought along with. There were two bite marks on Rt. Foot of the boy. There was no pain, no swelling.

a) Was the patient manageable at S D Hospital or should be referred to Bankura Medical College?

b) What treatment to start with?

c) What investigation to be done?

d) How long to wait before referral?

e) How long to be kept under observation?

6. Case 6: A 10 year old boy brought to Salboni Rural Hospital with history of common Krait bite in Bed previous night. The boy was gasping on admission.

a) Manageable at Rural Hospital or not?

b) What treatment to start with?

c) Try to refer to higher center or not?

d) What to do before referring?

**Management of the cases:**

All these cases are from our records; see the managements given to each individual case.

**Case No.1.** B. Hazra was successfully managed at Habra S G Hospital in Oct, 2007. He was immediately admitted and IV fluid started. IV infusion of 15 vials of Polyvalent AVS was given within 45 minutes. Inj. Atropin IV and Inj. Neostigmine IM were given simultaneously. The Pt. opened his eyes within 40 mins, could tell his name after 45 mins, could walk to other bed after 75 mins. Inj. Adrenaline (0.5 ml) was given IM as some urticarial rashes were noted on his throat and chest. After completion of AVS drip, he recovered completely and was discharged in the afternoon.

**Case No.2.** B Manna was managed at Debra BPHC in April 2010. He was admitted in the BPHC, and IV AVS was started. 10 vials of AVS were infused in 1 hr. 20WBCT was normal after infusion of AVS. The pt. was not transferred to any higher center as there were no clinical abnormalities; blood test from outside shown normal renal function. Pt. was discharged after 2 days.

**Case No. 3.** T Bag attended a private NH at Mecheda in Sept 2009. Artificial ventilation was started by Ambu bag and a fluid was started. Neurotoxic snake bite was diagnosed by typical history (including floor bed). 10 vials of AVS were infused in the ambulance when the pt. was being transferred to Kolkata. Pt.
survived after artificial ventilation for 5 days in a private hospital of Kolkata. One Common Krait snake was recovered from the room where the boy slept last at home.

**Case No. 4.** This boy was managed at Habra S G Hospital in Nov. 2007. All ligatures were released after IV fluid was started. No progress of local swelling was noted; swelling subsided within one hour. 20WBCT was normal. No AVS was given as it was a case of non venomous snakebite. Pt. discharged after 24 hours.

**Case No.5.** This boy was managed at Bishnupur S D Hospital in March 2012. Pt. admitted and a plain drip started. 20WBCT test was done on admission, and repeated thrice in one hr. Intervals. No clotting abnormality note. Diagnosed as a dry bite, pt. discharged on the next day.

**Case No. 6.** A10 year old boy comes to Salboni Rural Hospital in June 2017. Spending 4 hours at the house of Ojha. The boy was gasping on admission. 10 vials of ASV and AN given rapidly. Intubated and ambubag ventilation started. No ITU bed was available at Midnapore Medical College. Patient was treated at Salboni. Tube was removed after 5 hours as spontaneous respiration started.

**References:**


3) Annual Administrative Report 2015-16; Dept. of H & FW, Govt. of West Bengal.


**Further Reading :**

1) WHO SEARO (New Delhi Office); Guideline for management of Venomous snakebites 2010.

2) Indian Paediatrics; March 2007.

3) JIMA (Journal of Indian Medical Association); June 2007.

4) WHO endorsed A2 Snakebite Management in Asia & Africa; March 2010.

Management of Common Poisoning in Hospital
List of contributors

Professor Sibarjun Ghosh
Head, Department of Pediatric Medicine
Medical College, Kolkata

Professor Biswajit Sukul
Head, Department of Forensic and State Medicine
Medical College, Kolkata

Professor Malay Kumar Ghosal
Department of Psychiatry
Medical College, Kolkata

Professor Arunansu Talukdar
Professor, Department of Medicine
Medical College, Kolkata

Dr. Sujit Sarkhel
Associate Professor, Institute of Psychiatry
IPGMER, Kolkata

Dr. Pramit Ghosh
Assistant Professor, Department of Community Medicine
Medical College, Kolkata
Patient with suspected poisoning

- Check Vitals, Blood Glucose, pupil, consciousness, GCS
- Check ventilation; give Oxygen
- General management to stabilise
- Wide bore IV access (if needed)
- IV fluid (as needed)
- Collect urine & serum for toxin assay (if permitted)
- Inform police/respective legal authority (if possible)

Patient unstable

- Resuscitate the patient
- Hypotension-IV fluid f/b pressor support
- Correct hypoglycaemia; add Thiamine 100 mg
- Control active seizure

Patient stable

Early presentation (3-4 hours)?

- Yes
  - Gastric lavage, Activated charcoal (C/i-Corrosive & Kerosene)
  - Dress change, skin wash
  - Keep Lavage sample, legal proceedings

- No
  - Toxidromic analysis

Toxidromic analysis

Cholinergic
- miosis, diarrhoea
- bradycardia, B/L crepts
- salivation, diaphoresis
- Atropine ± PAM

Anticholinergic
- mydriasis, light non-responsive
- tachycardia, hyperthermia, delirium
- dry skin, urinary retention
- Supportive + Physostigmine

Sympathomimetics
- mydriasis, light responsive
- tachycardia, hyperthermia
- wet skin, no retention
- Supportive + BZD

Opioids
- miosis
- bradycardia, ? RR
- ? BP, coma
- Naloxone

Sedatives
- Stupor
- slurred speech
- apnoea, coma
- Supportive ± Flumazenil

Others

- Paraquat, CuSO₄
- Diarrhoea-CuSO₄
- Bleeding- AIP, ZnP
- Jaundice-Mushroom, CuSO₄, PCM etc

Re-evaluate & dosage modification or change of therapy
At the end of studying this learning unit the learners will be able to –

1. Make rapid assessment including psychological state in a case in the Emergency room presenting with history of poisoning.
2. Suspect a case about possible poisoning in absence of history of such.
4. Follow general principles of management of poisoning including plan for psychiatric management in case of suspected attempted suicide.
5. Categorize and institute poison-specific management.
7. Solve different medico-legal issues relating to a case of poisoning.

Poisoning refers to the development of dose related adverse effects following exposure to chemicals, drugs or other xenobiotics. It is rightly said that it is the dose that makes the poison. Poisoning is the fourth common cause of mortality in rural India. Compared to Western countries where medicinal poisoning is common, the prevalence of pesticides and agricultural poisons is high in West Bengal, especially in rural population. This pattern is also changing as effect of rapid urbanisation. The commonly used poisons can be broadly divided into following groups:–

- Pesticides
- Herbicides
- Corrosives
- Drugs of abuse
- Medicines
- Plant products
- Kerosene
- Others

Approach:

While dealing with poisoning, four sections are to be considered:

1. Identification of the poison-by history/circumstantial evidence or sign/symptoms of the case
2. Differential diagnosis-exclusion of poison mimics
3. Identification of co-morbidities
Clinical Features:

A proper history is elusive because the patients, in some cases, might not be conscious or co-operative. The nature of the poison, dose taken, the route of entry in body, time of exposure and vomiting, if any, are sometimes valuable clue to predict prognosis and to guide the mode of therapeutic action. In some cases, circumstantial evidences in the form of empty bottles/packs or travelling alone may be useful.

Medical management is to be provided urgently if the situation demands. Resuscitative measures are to be initiated at sign of hemodynamic instability. Certain clinical signs can provide valuable clues in etiological analysis:

### Vital signs

<table>
<thead>
<tr>
<th>Vital signs</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bradycardia</td>
<td>Oleander, OPs, CCBs, Opioids etc</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Sympathomimetics, Theophylline, Dhatura etc</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Alcohol, Opioids, Sedatives, OHAs etc</td>
</tr>
<tr>
<td>Hyperthermia</td>
<td>Dhatura, Heroin, Rodenticides, Alcohol withdrawal etc</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Thyroxine, Anticholinergics, Sympathomimetics</td>
</tr>
<tr>
<td>Rapid respiration</td>
<td>OPs, Kerosene, Salicylates, Paraquat</td>
</tr>
<tr>
<td>Slow respiration</td>
<td>Sedatives, Alcohol, Opioid, Marijuana</td>
</tr>
<tr>
<td>Coma</td>
<td>OP, Alcohol, OHAs, Ethylene glycol etc</td>
</tr>
<tr>
<td>Convulsion</td>
<td>OP, Alcohol withdrawal, TCA, Salicylates</td>
</tr>
<tr>
<td>Miosis</td>
<td>OP, Opioids, Sedatives, Clonidine</td>
</tr>
<tr>
<td>Mydriasis</td>
<td>Dhatura, Sympathomimetics</td>
</tr>
<tr>
<td>Diaphoresis</td>
<td>OPs, Salicylates, Sympathomimetics</td>
</tr>
<tr>
<td>Dry skin</td>
<td>Anticholinergic, Antihistaminic</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>Dapsone, Aniline dyes, Nitrate, Ergotamine</td>
</tr>
<tr>
<td>Odour</td>
<td>OP, AlP, Petroleum</td>
</tr>
</tbody>
</table>

Certain toxidromes are also helpful:

<table>
<thead>
<tr>
<th>Syndromes</th>
<th>Symptoms</th>
<th>Common Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholinergic</td>
<td>Bradycardia, bronchorrhoea, miosis, salivation, wheezing, diarrhoea, diaphoresis</td>
<td>Organophosphorus, pyridostigmine etc</td>
</tr>
<tr>
<td>(Muscarinic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholinergic</td>
<td>Abdominal pain, fasciculation, hypertension, paresis, tachycardia, seizures</td>
<td>OPs, Nicotine</td>
</tr>
<tr>
<td>(Nicotinic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anticholinergic</td>
<td>Delirium, mydriasis, tachycardia,hyperthermia, dry skin, urinary retention</td>
<td>Atropine, TCAs, Psychoactive drugs</td>
</tr>
<tr>
<td>Opioids</td>
<td>Miosis, sedation, hypotension, hypoventilation, bradycardia, coma</td>
<td>Opioids</td>
</tr>
<tr>
<td>Sympathomimetics</td>
<td>Mydriasis, tachycardia, hypertension,seizure,hyperthermia</td>
<td>Cocaine,AmphetamineTheophylline, Ephedrine</td>
</tr>
</tbody>
</table>
**Investigations:**
- Routine tests
- Electrolytes & Renal function test
- Liver function test
- Arterial blood gas analysis-HAGMA with increased serum osmolarity in methanol, ethylene glycol, Ethanol
- Gastric fluid analysis-forensic implication
- Urinalysis for toxicity-forensic implication
- Specific measures -
  - Serum acetylcholinesterase-for OP poisoning
  - ECG-for Aluminium phosphide, TCAs, Antiarrythmics
  - Toxicology screen-where the nature of the poison can’t be ascertained from investigations - BZD etc
  - Serum drug level-e.g. Digoxin, theophylline, lithium etc

**Management:**

- **Decontamination:**
  The poison that has not entered the body should be removed immediately. In OP poisoning, the clothes must be changed immediately to prevent additional exposure from cutaneous route. For carbon monoxide poisoning, the patient should be taken to fresh air immediately. Skin & eyes are irrigated with clean water/normal saline after corrosives exposure. Gastric lavage should be done if patient presents within 2-5 hours except in corrosives, kerosene poisoning.

- **Antidotes:**
  Antidotes are agents that counteracts the effect of poison. Activated charcoal acts by absorbing by porosity organic and some mineral poisons. Specific Antidotes be like:

<table>
<thead>
<tr>
<th>Antidote</th>
<th>Toxin(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine,PAM</td>
<td>Organophosphorus</td>
</tr>
<tr>
<td>Naloxone</td>
<td>Opioids</td>
</tr>
<tr>
<td>BAL</td>
<td>Arsenic, Mercury</td>
</tr>
<tr>
<td>Sodium edentate</td>
<td>Lead</td>
</tr>
<tr>
<td>Desferrioxamine</td>
<td>Iron</td>
</tr>
<tr>
<td>D-penicillamine</td>
<td>Gold, Copper, Lead, Mercury etc heavy metals</td>
</tr>
<tr>
<td>N-acetyl Cysteine</td>
<td>Paracetamol</td>
</tr>
<tr>
<td>Physostigmine</td>
<td>Anticholinergics</td>
</tr>
<tr>
<td>Ethanol/Fomepizole</td>
<td>Methanol/Ethylene glycol</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>Tricyclic antidepressants</td>
</tr>
<tr>
<td>Nitrites</td>
<td>Cyanide</td>
</tr>
<tr>
<td>Fab fragments</td>
<td>Digoxin</td>
</tr>
<tr>
<td>Methylene blue</td>
<td>Methaemoglobinaemia</td>
</tr>
</tbody>
</table>
• **Supplemental Oxygen therapy:**

O₂ is needed to correct hypoxaemia as effect of many poisoning like OP, but it is also of primary importance in treatment of inhalation of toxic fumes and vapours like carbon monoxide, asphyxiants etc. In some cases due to medullary centre suppression, ventilation may be necessary for sustainance of life to counter hypoventilation mediated type 2 respiratory failure.

• **Enhanced Toxin Removal:**

Adequate hydration is the cornerstone of management of many poisons as many poisons are excreted through kidneys. Forced diuresis by furosemide and/or mannitol can be done. Forced acid diuresis is employed for amphetamines whereas alkaline diuresis is beneficial for aspirin and barbiturate poisoning. Urinary alkalinisation is done by IV Sodium bicarbonate (1 litre of an 1.26% solution over 3 hours). IV potassium supplementation is necessary for urine alkalinisation. Urinary acidification is done by giving Ammonium chloride 4 gm every 2 hourly through nasogastric tube/Per Oral.

• **Dialysis:**

Small molecules, freely circulating in plasma not binding to proteins are dialyzable, especially those with molecular weight 2000 D or less. Cut off for high flux membrane is 10000 D. Rate of clearance can be increased by increasing the flow rate of blood and ultrafiltration rate. Barbiturates, Theophylline, Lithium, Bromides, Salicylates, Digitalis and Methanol poisoning cases are suitable candidates for dialysis. Hemofiltration, by convective transport can remove molecules up to 40000-D. It is advantageous for metal chelating complexes and aminoglycosides. Peritoneal dialysis is relatively ineffective. Exchange transfusion has also role by removing not only the poison but also red cell fragments and free haemoglobin and also in hydrogen sulphide poisoning.

<table>
<thead>
<tr>
<th>Indications for dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refractory hyperkalemia</td>
</tr>
<tr>
<td>Refractory fluid overload</td>
</tr>
<tr>
<td>Refractory metabolic acidosis</td>
</tr>
<tr>
<td>Uraemic asterixis, pericardial rub/effusion, bleeding, encephalopathy</td>
</tr>
<tr>
<td>GFR below 10 mL/min/1.73 m² body surface area</td>
</tr>
<tr>
<td>Some specific toxins mentioned below</td>
</tr>
</tbody>
</table>

• **Supportive measures:**

- Intensive care support-Arrhythmia, Hypotension etc.
- Seizures-BZD, barbiturates and valproates are choice
- Respiratory failure-Mechanical ventilation
- Renal failure-Dialysis
- Management of hepatic failure
MEDICOLEGAL ASPECTS OF POISONING

- Whatever be the nature of poisoning — every hospital is under legal obligation to treat the victim at its best.
- No case can be refused on the pretext that the hospital concerned is not authorized to handle medicolegal cases.
- If adequate facilities do not exist for proper treatment, the victim should be administered first aid and such other medical or surgical help before referring him to the nearest hospital where required facilities exist.

**Duties of a doctor towards suspected / diagnosed case of poisoning:**

The duties include both medical and medicolegal ones:

**Medical duties**

1. Save life (undertake ABCD of assessing the patient and treat accordingly)
2. Remove from source of poisoning.
3. Advise immediate hospitalization of patient.
4. Identify the poison or at least the type of poison (pharmacological group) consumed.
5. Assess the approximate time of exposure to the poisonous substance.
6. Assess the approximate quantity or amount of the poison.
7. Ascertain route of poisoning.
8. Rule out any head injury.
9. Appraise the psychiatric status.
10. Elicit H/O any suicidal attempt earlier.
12. Verify for any other co-existing disease and/or drug intake history.
13. Take second opinion from a competent professional colleague.

**Medicolegal duties:**

1. The case should be booked as a medicolegal case
2. Note preliminary particulars of patient i.e. name, age, sex, address, date and time of examination, *identification marks* etc.
3. Assist police to determine the manner of death (definite opinion as regards to the suicidal homicidal or accidental intake of poison should be given in a guarded manner not as mere assumption if asked for)
4. Detailed history regarding route of exposure, quantity consumed, time of exposure etc. (if patient is conscious).

5. Collect vomitus, blood, urine and stomach wash sample (if not contraindicated) and submit for chemical analysis (ideally in a clean glass container duly packed, labelled with patient particulars and sealed & signed properly and handed over to the law enforcing authority if asked for).

6. Carefully observe and record the symptoms in relation to food intake history i.e. any change in colour, taste, smell of food and drink.

7. Consult in strict confidence with senior colleague and keep him informed about the case.

8. Admit the patient to hospital.

9. Doctor should keep detailed records of number of his visits, symptoms and signs observed and treatment given time to time.

10. If the patient is an adult who retains his mental faculties, it might be desirable to intimate him about the steps to be taken.

11. Any suspected article i.e. food, excreta etc. should be preserved (ideally in clean glass container duly packed labelled with patient particulars and sealed & signed properly and handed over to the law enforcing authority if asked for).

12. Full or empty bottles, capsule/medicine strips, paper packets or liquid substance around should be collected and preserved noncompliance is punishable under Sec 201 IPC (causing disappearance of evidence of offence, or giving false information to screen offender). If it is proved that doctor did it with intention of protecting the accused.

13. Doctor working in Govt. hospital is required to report every case of poisoning to the police regardless of its nature.

14. All cases of homicidal poisoning (proved or suspected) must be compulsorily reported to the police as per Sec 39 CrPC (public to give information of certain offences). Failure to do so will make him culpable under Sec 176 IPC (omission to give notice or information to Public servant by person legally bound to it).

15. If police require information on any case of poisoning the attending doctor has to disclose it. There is no scope for professional secrecy in such matters Sec 175 CrPC (power to summon person). If information is withheld or wrong information is provided, the doctor becomes culpable under Sec 202 (intentional omission to give information of offence by person bound to inform) and 193 IPC (punishment for false evidence) respectively.

16. Every effort must be made by the attending doctor to collect and preserve evidence suggestive of poisoning. Deliberate omission to do so can cause punishment under Sec 201 IPC.
17. If death of the patient is imminent, dying declaration relating to the circumstances of poisoning can be recorded by the attending doctor in presence of two witnesses, if situation arises.

18. If death occurs or patient is brought dead to the hospital, on duty doctor must inform the police about the medicolegal autopsy to be done. **Death certificate must not be issued in such cases.**

19. Detailed written records should be made with respect to every case of poisoning and kept in safe custody.

20. If a doctor come across a case of **food poisoning from public eatery** (canteen, café, restaurant etc.), he must notify the incidence to **the public health authority** concerned. Such cases may come under the provisions of **Sec 269 IPC** (negligent act likely to spread infection dangerous to life), **272 IPC** (adulteration of food and drink intended for sale) and **284 IPC** (negligent conduct with respect to poisonous substance).

- Consent of a patient is necessary for all diagnostic and therapeutic procedures particularly which are invasive and risky in nature. In case of minor, consent is to be taken from parents or legal guardian.

- A patient who has deliberately consumed a poisonous substance or overdosed on a therapeutic drug is likely to be non-cooperative and may resist all efforts at treating him. But doctor should persuade him for receiving the treatment.

- At times the patient may be heavily agitated or extremely non-cooperative. In such cases, an attempt to remove the poison or drug may place the patient at greater risk of physical harm than the ingestion itself. In such cases, patient should be observed or partially restrained until the patient becomes cooperative. **If the patient has ingested imminently life threatening poison then no effort should be spared in restraining the patient physically or even pharmacologically, if necessary, in order to eliminate the toxin before it exerts its harmful effects.**

- In case of comatose/unconscious patients, consent must be obtained from the next of kin.

- During gastric wash/evacuation by stomach tube (if not contraindicated), explain exact procedure to the patient and **obtain his consent.** If refused, better not to undertake lavage because it will amount to assault, besides increasing risk of complications due to active non-cooperation.

- **However, if it is an emergency and consent is being refused on unreasonable grounds, the physician can go ahead with necessary treatment even in absence of consent.**

**Toxicology and the criminal law**

The following sections of IPC deal directly or indirectly with offence involving poison **Sec 284 IPC Sec 299 IPC Sec 300 IPC Sec 304A IPC Sec 324 IPC Sec 326 IPC Sec 328 IPC** (whoever administer to any person any poison or any stupefying agent or intoxicant or drug with intend to cause hurt to such person is punished with imprisonment up to 10 years with fine). **It deals specifically with Poison.**
Poison Specific Management
ALUMINIUM PHOSPHIDE POISONING

Aluminium phosphide is a solid fumigant pesticide, used for grain preservation. The main toxic product is Phosphine gas (PH₃) which is liberated on contact with moisture. It is available in India in packet and tablet forms-3 gm per tablet and 10 gm per tablet in various names- Celphos, Quickphos, Fumitoxin, Fieldphos, Weevil-cide etc. Each packet contains 56% AlP and 44% ammonium carbonate [(NH₄)₂CO₃].

Mechanism of Action:
The phosphine gas upon liberation from its preparation causes cellular and tissue hypoxia by inhibition of cytochrome c. There is also generation of reactive oxygen radicales as well as inhibition of mitochondrial catalase, extra-mitochondrial release of H₂O₂.

\[
\text{AlP} + 3\text{HCl} \rightarrow \text{AlCl}_3 + \text{PH}_3 \text{(Stomach)}
\]
\[
\text{AlP} + 3\text{H}_2\text{O} \rightarrow \text{Al(OH)}_3 + \text{PH}_3
\]
\[
(\text{NH}_4\text{CO}_3 + \text{H}_2\text{O} \rightarrow 2\text{NH}_3 + \text{CO}_2 + \text{H}_2\text{O} \text{ (To create neutral environment for PH}_3\text{)}
\]

Fatal Dose: 150-500mg (500mg/70 kg body weight)

Clinical Features:
Inhalational exposure:
Airway irritation, cough, breathlessness, pulmonary edema in some cases and severe cases, cardiovascular collapse and shock depending on higher concentration.

Exposure by Ingestion:
The clinical spectrum varies with dose of poison, route as well as freshness of the sample-old sample kept long in outside can cause reduced toxicity. Common symptoms are enlisted below:

Gastrointestinal - nausea, vomiting, heartburn most common

Cardiovascular - myocardial damage, myocarditis causes tachycardia, ventricular arrhythmia, conduction block, ST-T changes, uncommonly CCF
Respiratory - cough, dyspnoea, cyanosis, pulmonary edema, ARDS

Others - drowsiness if severe toxicity, profound metabolic acidosis, DIC etc. Its storage in liver may be responsible for extended toxicity in humans.

**Diagnosis:**

- History and Circumstantial evidence
- Decaying fish/Garlic like odour in mouth
- ECG-to look for ventricular arrhythmia
- ABG-metabolic acidosis
- Blood magnesium level
- Confirmatory - positive silver nitrate test with gastric fluid or breath
- Other routine parameters

In non-cooperative patients, unexplained shock or ventricular arrhythmia, acidosis of sudden onset clues to the diagnosis.

**Management:**

Early recognition is the key. The main goal of therapy is to sustain life till excretion of all of PH₃/Phosphine.

**Decontamination:**

It is advised for gastric lavage with potassium permanganate (KMnO₄) in 1:10000 to oxidise/inactivate the unabsorbed poison if presentation within 2-5 hours. Gastric lavage with water is not recommended. A few researchers from India has shown the role of liquid paraffin/coconut oil by reducing absorption creating a mucus layer and enhanced gut excretion. Slurry of activated charcoal (100gms) may be given to absorb PH₃.

**Reducing Tissue Toxicity:**

There is no antidote available for it. Magnesium sulphate acts as a membrane stabilising agent for arrhythmia. The dose is 1 gm intravenous, then 1 gm IV after 1 hour for 3 consecutive hours, then 1 gm IV infusion after 4-6 hours for 3-5 days. However this drug is not recommended by few authors due to relative lack of efficacy.

**Supportive therapy:**

BP control-excretion is through renal and respiratory tract, hence maintenance of renal perfusion is of paramount importance. Shock is managed initially by infusing 2-3 L of NS in first 3-6 hours under CVP and PCWP monitoring. Low dose dopamine (4-6ug/kg/min) can be used in shock with corticosteroids in prolonged hypotension. Metabolic acidosis-Sodium bicarbonate infusion may be given. Dialysis may be useful in ARF in hemodynamically stable cases.

**Take home message:**

Aluminium phosphide poisoning:

- Don’t give gastric lavage with plain water
- Use Magnesium sulphate in arrhythmia
- Use dopamine ± Steroid in hypotension
Organophosphorus poisoning

This accounts for a major chunk of suicidal cases. They are widely used as insecticides in agricultural, industrial and domestic poisoning. OPs include malathion, parathion, methylparathion, fenitrothion, diazinon, dichlorovas etc. Carbamates include carbaryl, propoxan and carbofuran. Absorption is possible by all routes- respiratory, eyes, skin and gastrointestinal tract.

- **Mechanism of toxicity:**

  OPs and carbamates inhibit the enzyme, acetylcholinesterase (AChE) which hydrolyses released acetylcholine at synaptic junction. OPs cause irreversible inhibition, whereas carbamates cause reversible inhibition. Inhibition of AChE thus facilitates cholinergic transmission resulting in initial stimulation of neurotransmission followed by paralysis. Increased Acetylcholine at nicotinic and muscarinic receptors causes characteristic effects. ‘Ageing’ of AChE determines therapeutic efficacy.

- **Clinical features:**

  Organophosphorus toxicity can be broadly divided into 3 clinical phases depending on temporal profile:

  **Acute intoxication:**

  1. **Muscarinic effects:**

     Cardiovascular-hypotension, bradycardia
     Respiratory-rhinorrhoea, bronchorrhoea, bronchospasm
     Gastrointestinal-abdominal pain, diarrhoea, hypersalivation
     Ocular-miosis, blurred vision
     Glands-diaphoresis, increased lacrimation
     Genitourinary-incontinence
2. **Nicotinic effects**:

   Weakness, Muscle fasciculation, cramps, hypertension, mydriasis and tachycardia

3. **CNS effects**:

   Restlessness, anxiety, ataxia, tremors, seizures and coma

4. **Other features**:

   Acute garlic odour from breath is characteristic. ECG may show ST-T changes and low voltage complexes. Uncommonly, arrhythmias like AV dissociation, multiple ventricular extrasystoles, polymorphic VT and Torsades de pointes can occur.

**Intermediate syndrome**:

- Develops 12-96 hours after exposure
- Reflects prolonged action of acetylcholine on nicotinic receptors
- Characterised by weakness of bulbar, ocular, neck, respiratory and proximal limb muscles.
- Sensory functions are normal, recovers in 4-18 days.

**Delayed polyneuropathy**:

- Common in compounds with weak anticholinesterase activity found to be neuropathic Mipafox, Merphos, Leptophos, Cyanophos, Trichloronat, DEF, EPN
- They start with paresthesia and calf pain
- Weakness appears in distal limb muscles causing foot drop, later on small muscles of hand and truncal muscles are also involved.
- Patient usually have gait ataxia with absent tendon jerks.
- Cranial nerves and autonomic system are uninvolved.

**Investigations**:

Diagnosis can be made from history of exposure, circumstantial evidence, clinical features and laboratory evaluation of Plasma Cholinesterase (PChE) and red cell acetylcholinesterase (Red cell AChE) activity inhibition. The depressed cholinesterase activity confirms diagnosis of OP poisoning and it remains to be depressed for 4-7 weeks. Though red cell AChE assay is more specific, PChE assay is easy and more widely available. ECG demonstrating previously mentioned features may be present.

**Treatment**:

Decontamination of the gut and skin are most important primary aspects of management. Clothes are to be removed to prevent further exposure from skin. Gastric lavage prevents additional absorption of OPs from gut if patient is brought in early phase. Sample should be preserved for medico-legal purpose. Activated charcoal is also effective. But for obtunded and hemodynamically unstable patients, resuscitation comes at top priority.

**Supportive measures**:

Airway, breathing and circulation should be maintained with maintenance of fluid balance and electrolytes. Intubation and mechanical ventilation may be necessary depending on clinical scenario. Diazepam is the standard treatment for seizures.
Antidote:

➢ Atropine:

Atropine antagonises the muscarinic effects with little effect on nicotinic receptors. Unlike glycopyrrolate, which can cross blood brain barrier, it counteracts CNS effects. Atropine can be given as bolus 2-5 mg IV in adults and 0.04-0.08 mg/kg in children. It can be repeated every 5-10 minutes till signs of atropinisation appear (Clear chest with absence of wheeze, Heart rate > 80 bpm, dry axilla, dilated pupil—not a good sign to monitor, BP > 80 mmHg). Once achieved, continuous infusion of atropine at rate of 0.02-0.1 mg/kg/hour can be given to maintain the atropinisation for 2-5 days with slow tapering.

➢ Pralidoxime:

Regeneration of AChE is done by Pralidoxime. It has little effect on those enzymes who already underwent ‘ageing’. It is given IV in doses of 1-2gm (30 mg/kg) over 15-20 minutes. Dosage may be repeated after 1 hour followed by continuous infusion in dose 500 mg/hour or 8-10 mg/kg/hour. It is not to be used in Carbaryl poisoning and has doubtful value in carbamate poisoning.

Take home message:

Organophosphorus poisoning:
- Urgent decontamination
- Use atropine in all cases
- Pralidoxime in early presentation

Organochlorine poisoning

Organochlorines are used as insecticides and pesticides and include DDT, methoxychlor, lindane, aldrin, dieldrin, endrin, heptachlor and endosulphan. All of them are absorbed by skin, orally and by inhalation-DDT being the least absorbed. They are lipid-soluble, partially metabolised by liver and excreted in feces, urine and milk. They interfere with nerve impulse stimulation-CNS stimulation followed by depression.

- Fatal Dose: DDT-30 gm, Lindane-15 gm etc.

- Clinical Features:

  Initial features are nausea, vomiting and epigastric distress. Most serious form is seizures where GI symptoms may be absent also. Other features are dizziness, myoclonus, opsoclonus, agitation and confusion. Organic solvents decrease the convulsive effects of DDT but increase CNS depression may also predispose to aspiration.

- Management:

  Ventilation and vitals are to be monitored as the patient can develop seizures, respiratory depression and aspiration due to hypoxia. Decontamination should be done by changing clothing and skin wash as well as gastric lavage if patient is brought in 3-4 hours. Cholestyramine is to be administered 16 gm/day in 4-6 divided doses in symptomatic patients as it interrupts the enterohepatic cycle of organochlorines. In children, Cholestyramine is to be given 240 mg/kg/24 hour in 3 divided doses. Seizure control is done with diazepam followed by Phenobarbital. Epinephrine is to be avoided; dopamine can be given in hypotension. Dialysis is not of much help.
**Take home message:**

**Organochlorine poisoning:**

- Urgent decontamination
- Administer cholestyramine to prevent enterohepatic cycling
- Seizure control with diazepam

**Paraquat poisoning:**

It is a bipyridylium compound, used as herbicide and weed-killer and major health problem among poisoning cases. Most cases are suicidal ingestion. The case fatality is very high (> 50%).

- **Mechanism:**
  Paraquat undergoes a NADPH dependent reduction to form the free radical which reacts with molecular oxygen to reform the cation and produces superoxide free radicals and hydroxyl radical (OH), which disrupt cellular function, structure. Concentrated solutions corrode GI mucosa.

- **Fatal dose:** 3-5 gm

- **Fatal period:** 2-7 days

- **Clinical features:**
  Ingestion of large amount (> 50-100 ml of 20% ion w/v) results in fulminant organ failure-death occurring from multi-organ failure within few hours to days. Moderate to severe poisoning usually targets kidneys and lungs. Renal failure develops rapidly. Acute alveolitis in 1-3 days is followed by secondary fibrosis leads to death from severe anoxia.

- **Treatment:**
  Resuscitation is top priority in unstable patients. There is no specific antidote for paraquat. Gastric lavage followed by a dose of activated charcoal is advised in literatures, but some authors are against it for caustic injury. Skin wash and changing clothes are recommended. Hemodialysis and hemoperfusion is standard treatment. Animal models have shown that HP was ineffective to reduce paraquat lung exposure unless started in 2 hr post ingestion. With no wide accepted treatment guidelines, many experimental therapies are adopted. Based on animal studies and limited human data, N-acetyl cysteine, vitamin-C, salicylate and dexamethasone appear to be promising but these agents need to be tested further to give opinion.

**Take home message:**

**Paraquat poisoning:**

- No specific antidote
- Urgent referral to higher centre for dialysis
Corrosive ingestion

These agents cause tissue destruction on coming to contact—they can be acids or alkali. Compared to Western world, acid ingestion is much more common in India where toilet-cleaning products are so much available.

<table>
<thead>
<tr>
<th>Strong Acids</th>
<th>Strong Alkalis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydrochloric acid</td>
<td>Lye</td>
</tr>
<tr>
<td>Sulphuric acid</td>
<td>Potassium hydroxide</td>
</tr>
<tr>
<td>Nitric acid</td>
<td>Sodium hydroxide</td>
</tr>
<tr>
<td>Phenol</td>
<td>Calcium hypochlorite (Bleach)</td>
</tr>
</tbody>
</table>

- **Mechanism:**
  
The extent of injury depends on nature of corrosive ingested, its physical state (solid corrosive tend to cause deep burns by adherence to pharyngeal, palatal and upper oesophageal mucosa), amount and concentration, timing of intake (full stomach after food intake reduces the damage). Alkalis cause liquefaction necrosis causing rapid penetration whereas acid ingestion causes coagulation necrosis with coagulum preventing further penetration—thereby it was considered that alkalis cause more injury to oesophagus and acids to stomach but according to some authors, concentrated acid or alkali both cause similar damage to stomach and duodenum. Solid corrosives in powder form cause esophageal burns while liquids damage the antrum & gastric mucosa.

- **Clinical features:**

  **Acute phase (1-10 days)**

  Intense pain in mouth, throat, hematemesis, dysphagia, odynophagia. Laryngeal oedema may cause hoarseness of voice and stridor. Full thickness necrosis may cause perforation, mediastinitis, peritonitis and septic shock. Chemical bronchitis can happen if aspirated. Burn, erythema can be seen at sites of local exposure e.g. angle of mouth etc.

  **Subacute phase (11-16 days)**

  Transient decrease in symptoms

  **Chronic phase (28 days-months)**

  Oesophageal stricture causing dysphagia and gastric injury causing gastric outlet obstruction.

- **Management:**

  Treatment consists of recognition and treatment of acute complications and its sequelae. Early treatment includes maintaining patient NPO, IV fluids, airway maintenance and supportive treatment. Gentle orotrachal intubation/fibre-optic intubation is advised as necessary in cases with use of IV ketamine if needed. Use of sedatives or NMBAs for intubation and blind nasotracheal intubation is contraindicated because of anatomic distortions and soft tissue perforation. Cricothyrotomy may be necessary in extreme tissue friability.
**Gastric emptying & decontamination:**

Emetics are contraindicated after corrosive ingestion as well as gastric lavage using NG tube. Activated charcoal is also ineffective because of poor absorption and endoscopic interference.

**Dilution:**

It may be beneficial for ingestion with solid/granular alkaline material if performed within 30 minutes of ingestion using small volume of water. However, because of the hazard of emesis and heat production, this is largely controversial and not done.

**Neutralization:**

Using weak acid or alkali is contraindicated because of exothermic reaction and chance of induced emesis.

**Upper GI Endoscopy:**

The optimum time for endoscopy is 12-24 hours post ingestion, to predict prognosis, further stricture formation and to guide next plan of management. Since inflammatory changes, vascular thromboses, sloughing and the healing by granulation tissue happen between 4th to 14th day, UGIE is contraindicated during this time frame. However endoscopy can be done after 3rd week, especially helpful for dilating strictures with repeated passage of bougie (less chance of perforation than balloon dilatation). Recently endoscopically guided nasojejunal tube placement is undergoing evaluation.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Kikendall’s classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Edema and erythema of the mucosa</td>
</tr>
<tr>
<td>IIa</td>
<td>Hemorrhage, erosion, blisters, superficial ulcers</td>
</tr>
<tr>
<td>IIb</td>
<td>Circumferential lesions</td>
</tr>
<tr>
<td>III</td>
<td>Deep grey or brownish black lesions</td>
</tr>
<tr>
<td>IV</td>
<td>Perforation</td>
</tr>
</tbody>
</table>

Some authors prefer Zargar classification.

**Steroids:**

Steroids were considered to prevent stricture formation in injury above grade IIb but most multicentric studies refuted this hypothesis with increased risk of perforation and peritonitis, mediastinitis. Steroid can be of value to reduce laryngeal oedema.

**Antibiotics:**

Use is under debate. Most authors do not recommend them on a routine basis.

**Nutrition:**

Debate exists regarding initiation of enteral feed. In patients with grade I/IIa injury liquid diet can be initiated after 24-48 hours until 10th day followed by liberalization of regimen. In patients with higher grade of injury concept of “Oesophageal rest” may extend to 10th to 15th day however, this is not supported in animal studies. In case of stricture formation, surgical procedure like feeding jejunostomy can be adopted.
Oesophageal dilation:
Can be initiated 6 weeks after injury to prevent perforation-then once every 2-3 months.

**Take home message:**

**Corrosive poisoning:**
- NG tube, emetics, activated charcoal-contraindicated
- Upper GI endoscopy-in 12-24 hrs or after 3 weeks; not in between
- Sequential institution of oral feed depending on degree of bum; later on, after evaluating the condition, surgical

---

**Mushroom Poisoning**

Mushroom is an important dietary constituent in certain ethnic populations in India, especially in South India and North-Eastern states. Often overlooked, this contributes significantly to the morbidity and mortality. There are almost 1200 species in India with 50-100 being known to be poisonous. North American Mycological Association (NAMA) maintains a case registry and tracks all cases of mushroom poisoning, hence it is important to report all cases. There are a total of 14 syndromes described worldwide-some of which abounds in India, some are rare.

**Gastrointestinal Irritants:**

Most frequent symptom of mushroom poisoning caused by wide variety. It can be early onset (<6 hours of ingestion) or delayed onset-in form of nausea, vomiting, diarrhoea. Early onset syndromes are less toxic, most commonly by *Chlorophyllum molybdites* species, however it does to exclude possibilities of lethal intake. If the gastrointestinal distress begins 6-24 hours after intake possibility of amatoxin can be there. Delayed onset gastroenteritis can be due to Allenic norleucine/Orellamine also. Treatment is largely supportive with maintenance of fluid balance & electrolytes and decontamination measures in case of early presentation.

**Amatoxins:**

Most serious form of mushroom poisoning. Causative agents are *Amanita phalloides*, *A. verna*, *Leperla helveola* etc.

Amanitins are a group of complex polypeptides that inhibit cellular metabolism by inhibiting RNA-polymerase mediated RNA synthesis. They are specially dangerous because of their delayed systemic manifestation 6-24 hours after ingestion by which time the patient may appear to recover after initial GI upset. Four stages of symptoms have been described in this regard:

1. Stage of latency-6-24 hours after ingestion with active damage to kidneys and liver but patient is asymptomatic
2. Stage of diarrhoea, vomiting-6-24 hours
3. Stage of clinical improvent-24-36 hours, victim appears to recover clinically
4. Stage of relapse- fulminant hepatic & renal failure

---

**Figure: Death Cap mushroom**
If amanitin toxicity is suspected then it is prudent to rush the patient to hospital without awaiting for symptoms. Activated charcoal is ineffective. Aggressive hydration with IV fluid is recommended to flush out the poison by excretion. LFT & blood clotting factors are to be closely monitored. In severe cases, experimental therapy by IV silibinin-5 mg/kg bolus followed by 20 mg/kg/day for 6 days or till patient recovers, may help to avoid liver damage. Use of Penicillin G in dose of 30000-100000 U/kg/day as continuous infusion is helpful as thought earlier but no longer considered effective by NAMA.

**Gyromitrin:**

*G. esculenta, G. inflata* etc. Product of hydrolysis-MMH (Monomethylhydrazine) is volatile, colourless, toxic and carcinogenic compound. So fatalities can happen during cooking of these brain-shaped mushrooms also. Symptoms include seizures, headache, delayed gastroenteritis and hepatotoxicity. Treatment is largely supportive with pyridoxine administration-70 mg/kg to 5 gm in seizure with anticonvulsant therapy. Methylene blue should be given in Methaemoglobinemia cases.

**Muscarinic effects:**

*Clitocybe delbata, Inocybe spp.*

Symptoms appear after 30 minutes of ingestion in form of bradycardia, salivation, lacrimation, bronchospasm, bronchorrhoea, diaphoresis-Atropine being the specific antidote. Repeated dosing may be necessary until drying of secretions.

**Isoxazole derivative (Muscimol, Ibotenic acid)**

*A.muscaria, A.gemmata* etc.

Muscimol is CNS depressant whereas ibotenic acid excites glutamate receptors in CNS. Symptoms include confusion, hallucination, delusion, seizure & comatose state. Treatment is largely supportive with reassurance. Atropine is not indicated.

**Psilocybin, Psilocin etc.:**

*P.cubensis* etc.

These are well-known hallucinogens. Effects are primarily psychological. There can be dangerous amatoxicity in hunt for magic mushroom.

**Coprine:**

*C.atramentaria*

Disulfiram like reaction can be triggered by consumption with alcohol.

**Renal toxicity:**

Delayed kidney toxicity may occur after Orellamine poisoning from *C.orellanus*. Renal failure is also documented after *Amanita smithiana* poisoning. Treatment is largely supportive.
Others:

Delayed rhabdomyolysis, erythromelalgia, encephalopathy, allergic reactions and immune haemolytic anaemia are also reported—treatment being mostly supportive.

**Take home message:**

**Mushroom poisoning:**

- Varied presentation—analyse the syndrome—CNS/GI-
- Hepatobiliary/Renal/Others
- Supportive therapy and specific drugs in some cases

**Kerosene poisoning**

Ingestion of hydrocarbons like gasoline, kerosene, mineral oil, lamp oil, paint thinners result in minimal systemic effects but severe aspiration pneumonitis. Toxic potential depends on viscosity. Liquids with low viscosity (SSU < 60) can spread over large surface area to cause aspiration pneumonitis rather than those with high viscosity (SSU > 60) like tar.

**Clinical Features:**

- Immediate burning sensation in throat
- Aspiration causing chemical pneumonia—cough, shortness of breath, tachypnoea, pulmonary oedema.
- CNS involvement—in large doses—lethargy, headache, seizure
- Sensitisation to catecholamines—fatal ventricular arrhythmia may ensue
- Chest X-ray and oximetry to be done after 6 hours of ingestion or earlier if symptoms are severe—ABG should be done if respiratory failure is suspected. Radiographic features consist of perihilar densities, atelectasis, pneumonitis and occasionally areas of consolidation. Pleural effusion may develop in some cases. Rarely, cysts or pneumatoceles may form.

**Management:**

- Contaminated clothing is removed and skin is washed.
- Gastric lavage is contraindicated for fear of aspiration pneumonia. Charcoal is not recommended.
- Patients who remain asymptomatic after 6-8 hours he can be discharged if chest X-ray is normal. Patients need to be admitted if present with respiratory distress and severe features in skiagram
- Ventilatory support may be needed.

**Take home message:**

**Kerosene poisoning:**

- No lavage
- Chest X-ray and pulse oximetry after 6 hrs or earlier
**Benzodiazepine poisoning**

CNS depression is most commonly seen in benzodiazepine poisoning due to exaggerated action at inhibitory GABA receptor. BZDs are considered safe and their overdose alone is seldom fatal. Overdose may cause reversible impairment of consciousness, excessive sleep, nystagmus, cognitive impairment, slurred speech, rarely deep coma and respiratory failure.

**Management:**

Only good supportive care is needed in most patients with occasional ventilator backup. Flumazenil is the specific BZD antagonist at GABA receptor and reverses symptoms rapidly—specially indicated in those cases with respiratory depression. It is given in dose of 0.1-0.2 mg IV over 30-60 seconds and is repeated every 1-2 minutes to a total of 1 mg at one time or 3 mg in one hour. Resedation can occur due to its short half-life and then it can be given as infusion. Flumazenil can precipitate seizure especially in co-poisoning with barbiturate/TCAs. Hence, toxicity screen should be done if suspected so.

**Dhatura poisoning**

Datura stramonium (Jimson seed, thorn apple) grows at high altitudes. All parts are poisonous but seeds and fruit are the most. Ingestion of 4-6 seeds even may prove fatal. Toxins are hyoscine, hyoscyamine, scopolamine and atropine.

![Dhatura fruit](image)

**Figure: Dhatura fruit**

- **Mechanism:**

  The toxins stimulate the higher centres of brain followed by depression and paralysis specially of medullary vital centres. Peripheral actions come from anticholinergic action.

- **Clinical features:**
  - Bitter taste, dry mouth and throat
  - Tachycardia, hypertension – later hypotension
  - Dry flushed skin, dry mucus membranes
  - Dilated pupil, red conjunctiva
  - Urinary retention, reduced bowel sounds
  - CNS effects include excitement, restlessness, seizure followed by depression, delirium (muttering delirium, picking at bed clothes, trying to pull imaginary threads) and coma.
Treatment:

Patient may improve in 24 hours, in some cases 2-3 days. If patient presents early after ingestion, gastric lavage can be performed with specimen preservation for medico-legal purpose. Activated charcoal can be helpful. Catheterisation can be done to help urinary retention. Hyperthermia should be treated with external cooling. Benzodiazepines are effective in treatment of agitation. Morphine is contraindicated in fear of depression of respiratory centre. IV fluid is necessary to combat tachycardia and acute renal failure.

The antidote for this toxin is physostigmine. It led to controversies despite its recent reports of safe use. Cardiac conduction defect is contraindication to physostigmine whereas some authors recommend it in severe cases of agitation/psychosis; intractable seizures/coma or tachycardia/dysrhythmia with hemodynamic compromise. Dose is 1 mg IV/IM followed by repeat dosing after an hour. In children, Inj. Physostigmine-0.001 – 0.03 mg/kg/dose, may be repeated at 15-20 mins interval as required (maximum 2 mg). Inj.pilocarpine nitrate 5 mg SC is useful but does not counteract dhatura action on brain. It can be repeated after 2 hours.

Take home message:

Dhatura poisoning:
- Gastric lavage; activated charcoal use
- Physostigmine in severe cases as well as pilocarpine

Rodenticide poisoning

Commonly used agents are Barium carbonate, Zinc phosphide and Anticoagulants.

Barium Carbonate:
It is marketed as white powder. Acute poisoning causes hypokalemia, neuromuscular blockade and respiratory failure. Treatment is largely supportive.

Zinc Phosphide:
It is available as dark grey powder & releases phosphine gas in stomach. Clinical features are similar to Aluminium phosphide poisoning but onset is little slower. Management is supportive and similar.

Superwarfarins (Anticoagulants):
They act as vitamin K epoxide reductase inhibitor, thus inhibiting synthesis of factor II, VII, IX, X. They act as 59 single-dose drugs because of long duration of action. Commonly used agents are Bromadiolone, Brodifacoum, Difenacoum and Diphacinone. Most patients do not develop significant abnormalities but a few develop bleeding tendencies. Prolonged Prothrombin time can be demonstrated after 36-48 hours and may persist for long. In cases with significant ingestion, vitamin K or Fresh Frozen Plasma may be required if patient develops features of bleeding. Recombinant factor VII has been found to be useful.
Copper sulphate poisoning

Copper sulphate (blue vitriol) is used as a fungicide, dye or in electroplating etc.

- **Fatal dose:** 30 gm
- **Clinical features:**
  - Metallic taste, hypersalivation, burning pain in stomach with colicky abdominal pain
  - Nausea, vomiting, diarrhoea (liquid-brown coloured)
  - Oliguria, hematuria, albuminuria, acidosis, uraemia
  - Severe cases: intravascular hemolysis, hemoglobinuria, methemoglobinemia, jaundice, pancreatitis, convulsions, coma
  - Death due to renal/hepatic failure

- **Treatment:**
  - Resuscitation in unstable patients
  - **Decreasing absorption:** Emesis is to be avoided. In prehospital setup, dilution with water/milk may be advisable. Stomach wash with 1% potassium ferrocyanide is recommended. A dose of activated charcoal while have unproven benefit is unlikely to be harmful.
  - **Managing corrosive burns:** UGI endoscopy
  - **Methemoglobinemia:** Symptomatic patients should be treated (20-30% of methemoglobin). Oxygen is to be administered while preparing for methylene blue. 1-2 mg/kg/dose methylene blue (0.1-0.2 mL/kg of 1% solution) IV over 5 minutes. The dose may be repeated if cyanosis does not disappear in one hour. Large doses itself can cause methemoglobinemia. It is contraindicated in G-6-PD deficient persons. Hyperbaric oxygen may be beneficial if methylene blue is ineffective.
  - Hypotension: fluid, dopamine, noradrenaline
  - Chelation therapy: d-Penicillamine-1000-1500 mg/day divided every 6-12 hourly before meals; avoided in penicillin allergy. In Children, D-Penicillamine-20 mg/kg/24 hours divided 6 –12 hourly, maximum 1 gm/ 24 hour
  - Dimercaprol/BAL: IM BAL is probably appropriate in patients with GI symptoms which precludes oral d-Penicillamine but less effective.3-5 mg/kg/dose deep IM every 4 hours for 2 days; 4-6 hourly for 2 days, then 4-12 hourly for 7 days. In children, Dimercaprol – 2.5 - 5.0 mg/ kg/ dose, 4 hourly - gradually tapering over 7–10 days.
  - Edetate calcium disodium-75 mg/kg deep IM or slow IV given in 3-6 divided doses for up to 5 days; may be repeated for a 2nd course after a minimum of 2 days; each course not to exceed 500 mg/kg.
  - Enhanced elimination- hemodialysis to remove copper is ineffective but it may be indicated in cases of renal failure.
Take home message:

Copper sulphate poisoning:
- Use Pot.ferrocyanide lavage
- Methemoglobinemia- Methylene blue
- Use d-penicillamine chelation therapy
- Hemodialysis in renal failure

Alcohol ingestion

Most commonly used is Ethanol. Most alcohol poisoning refers to ethanol poisoning.

- **Clinical features:**

  3 stages are noticed as it gradually depresses the cerebral cortex → vital centres in medulla and midbrain.

  1. Stage of excitement
  2. Stage of incoordination

<table>
<thead>
<tr>
<th>Blood Alcohol Level</th>
<th>Clinical Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 - 50 mg/dl</td>
<td>No Legal intoxication, some uncoordination potential changes in behaviour.</td>
</tr>
<tr>
<td>80 - 100 mg/dl</td>
<td>Legal intoxication, impaired ability to drive slurred speech, staggered gait, impaired sensory function</td>
</tr>
<tr>
<td>100 - 150 mg/dl</td>
<td>Markedly uncoordination, gross cognitive and judgment distortion</td>
</tr>
<tr>
<td>Above 200 mg/dl</td>
<td>Notable impaired sensory and motor function</td>
</tr>
<tr>
<td>Above 300 mg/dl</td>
<td>Potential for cardiovascular and respiratory collapse, coma, and death can occur.</td>
</tr>
</tbody>
</table>

- **Treatment:**

  - Removal of unabsorbed alcohol by gastric lavage
  - Adequate ventilation
  - Dextrose infusion to correct hypoglycaemia
  - Simultaneous thiamine administration (100 mg IV) to prevent Wernicke’s encephalopathy
  - Urinary alkalisation with sodium bicarbonate may be attempted.
  - Hemodialysis in severe cases
  - Co-poisoning or other injuries must be suspected in obtunded patients
Methanol poisoning

Common contaminants in unbranded alcoholic beverages, also a component of solvents, paints, methylated spirit.

- **Mechanism:**
  Primarily metabolised to liver to form formic acid responsible for toxicity-follows zero order kinetics.

- **Clinical features:**
  - Temporary CNS depression followed by latent period of 6-24 hours followed by metabolic acidosis and visual symptoms
  - Failing visual acuity, photophobia, ‘snow field vision’, finally blindness
  - Only 10 mL can cause blindness, 30 mL can be fatal.
  - Arterial pH < 7.2 in severe poisoning

- **Investigation:**
  - Routine along with LFT & RFT, serum glucose, osmolal gap
  - Serum methanol level

- **Treatment:**
  - Gastric lavage if present within 2 hours
  - Seizures to be treated with phenytoin as it has less CNS depressant action than diazepam
  - Folinic acid 1 mg/kg to a maximum of 50 mg should be administered-repeated every 4 hours
  - If not available, folic acid may be given in same dosage.
  - Specific agents-ethanol/fomepizole should be used to prevent formic acid formation.

<table>
<thead>
<tr>
<th>Indication for ethanol/fomepizole treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Documented plasma level &gt; 20 mg/dL</td>
</tr>
<tr>
<td>➢ Recent H/o ingestion and osmolal gap &gt; 10 mmol/kg</td>
</tr>
<tr>
<td>➢ Strong clinical suspicion and at least 2 of the following-pH &lt; 7.3; HCO₃ &lt; 20 mmol/L; osmolal gap &gt; 10mmol/kg; visual signs/symptoms</td>
</tr>
</tbody>
</table>

- Hemodialysis should be considered in critical situations

<table>
<thead>
<tr>
<th>Indication of Hemodialysis in methanol poisoning</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ pH &lt; 7.3</td>
</tr>
<tr>
<td>➢ Acute renal failure</td>
</tr>
<tr>
<td>➢ Visual signs/symptoms</td>
</tr>
<tr>
<td>➢ Deteriorating vital despite supportive care</td>
</tr>
<tr>
<td>➢ Documented plasma level &gt; 50 mg/dL</td>
</tr>
</tbody>
</table>
Paracetamol ingestion

It may be taken orally for intentional self-harm or for relief of pain-opioid combination is particularly harmful in this regard. Fatal fumarant liver disease is usually associated with single intake $>25$ gm. Blood level of acetaminophen usually correlates with hepatic injury (level $>300 \mu g/mL$ 4 hour after ingestion predicts severe damage, whereas $<150 \mu g/mL$ suggests unlikely hepatic injury).

- **Mechanism:**

  It is metabolised to N-acetyl-para-benzoquinoneimine (NAPQI) by P450 CYP2E1 which is detoxified by glutathione. Massive ingestion overwhelms this protective response leading to formation of protein adducts which are hepatotoxic.

- **Clinical features:**

  - Nausea, vomiting, abdominal pain occurs 4-12 hour after ingestion
  - 24-48 hours later, hepatic injury becomes apparent
  - Hepatic failure is evident after 3-5 days
  - Aminotransferases may exceed 10000 U/mL
  - Renal failure/myocardial injury may be present.

- **Management:**

  - Supportive measures
  - Gastric lavage-done before oral agents
  - Activated charcoal/cholestyramine in 30 minutes
  - N-acetyl cysteine-in patients with high acetaminophen blood levels ($>200 \mu g/mL$ at 4 hour or $>100 \mu g/mL$ at 8 hours), therapy should be initiated within 8 hour but at least partially effective when given as late as 24-36 hours. NAC is used orally but also as IV solution with loading dose of 140 mg/kg over 1 hour, followed by 70 mg/kg every 4 hour for 15-20 doses. Treatment can be stopped when plasma level indicate that liver damage chance is low.
  - Liver transplant-Liver failure despite NAC therapy

Inhalant fumes

It represents an important route for poisoning because of large surface area of the lungs and blood supply, accounting for absorption and distribution of toxic gases. Toxic gases and vapours can be classified based on their mode of action:

<table>
<thead>
<tr>
<th>Classes</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Irritants</td>
<td>Ammonia, chlorine, sulphur dioxide, ozone, phosgene, halogens, acrolein</td>
</tr>
<tr>
<td>Simple Asphyxiants</td>
<td>Nitrogen, hydrogen, methane, liquid petroleum gas, propane, carbon dioxide, dichlorotetrafluoroethane</td>
</tr>
<tr>
<td>Chemical Asphyxiants</td>
<td>Carbon monoxide, hydrogen, cyanide, nitriles, hydrogen sulphides</td>
</tr>
<tr>
<td>Central nervous system depressants</td>
<td>Aliphatic hydrocarbons, chlorinated hydrocarbons, acetone, ethylether, benzene</td>
</tr>
</tbody>
</table>
### Neurotoxic agents
- Carbon disulphide, mercury, acrylamide, n-hexane, methyl n-butyl ketone

### Hepatotoxic agents
- Carbon tetrachloride, chloroform, allyl alcohol, bromobenzene

### Nephrotoxic agents
- Carbon tetrachloride, chloroform, trichloroethylene

### Agents damaging blood
- Nitrobenzene, arsine, naphthalene

### Agents damaging bone marrow
- Benzene, trinitrotoluene

### Carcinogens
- Vinyl chloride, 2-naphthylamine, bis(chloromethyl)ether

---

#### Clinical features:

Soluble irritants cause burning & irritation to eyes, nose, trachea etc. Marked cough, hemoptysis, wheeze are common. Nonsoluble agents have less immediate symptoms but have cough/shortness of breath. ARDS patients have worsening dyspnoea and oxygen requirement.

Patients should have a chest x-ray and pulse oximetry. Chest x-ray findings of patchy or confluent alveolar consolidation usually indicate pulmonary edema. Spirometry may be done - obstructive anomalies are common but restrictive variety is seen after chlorine exposure. Late developing patients like bronchiolitis obliterans progressing to respiratory failure can be evaluated with CT chest.

#### Management:

Patients should be moved into fresh air and given supplemental oxygen. Rescuer should be armed with necessary protective gears before attempting to rescue the patient. Treatment is directed toward ensuring adequate oxygenation and alveolar ventilation.

Bronchodilators with oxygen therapy may suffice in less severe patients. Severe airflow obstruction can be managed with inhaled racemic epinephrine, endotracheal intubation/tracheostomy and mechanical ventilation. Because of the risk of ARDS, any patient with respiratory tract symptoms after toxic inhalation should be observed for 24 h. High-dose corticosteroids should not be routinely used for ARDS induced by inhalational injury; except few cases of zinc chloride fumes. After tiding over acute crisis, physicians must be aware of the reactive airways dysfunction syndrome, bronchiolitis obliterans ± organised pneumonia, pulmonary fibrosis and late onset ARDS. Therefore, necessary protective gear use is the most often preventive measure often forgotten leading to this calamity.

---

### Scorpion Sting

Indian red scorpion, *Mesobuthus tumulus* is the lethal species. Susceptible: laborers, farmers, villagers and urban slum dwellers. Features of scorpion sting:

- **Local symptoms-** pain. Adults commonly have local symptom only. Treated by ice compression, local anaesthetic agents, oral paracetamol

- **Systemic symptoms of envenomation:** common in children.
  - Parasympathetic effect (stimulation), most common, usually short lasting but may last longer called “Autonomic storm". The features are vomiting, excessive salivation, profuse sweating, bradycardia, cold extremities, hypotension, ventricular ectopics, priapism in males. Excessive bronchial secretion can cause respiratory failure.
• Sympathetic excitatory features: in severe envenomation and follows above. Signs of severe vasoconstriction, hypertension, tachycardia, cardiac arrhythmia, pulmonary oedema. Mortality is high when there is pulmonary oedema.

Investigations: CBC, serum electrolytes, BUN, Chest X-ray, ECG monitoring and Echocardiogram if facilities present.

Treatment:
• Features of early autonomic storm: Prazosin (post-synaptic alpha-1 receptor blocker) is the key drug. Dose 30 mcg/kg/dose. Can be repeated ½ to 1 hourly up to maximum 4-doses. Best result if administered within 4 hours. Only scored tablet (not sustained release tablet) to be used. Scorpion antivenom (SAV) + prazosin have revolutionized the outcome of scorpion sting by lowering mortality. But SAV may not be available.

• If Heart rate increases, poor perfusion present with BP- low: Child should be managed in PICU with continuous monitoring. IV fluid NS or RL - @ 10 ml/kg/aliquot in bolus up to 4 aliquots. If shock continues- Dobutamin infusion should be started. IV nitroglycerin and ventilator support and oxygen inhalation may be required.

• No steroid, no antihistamine, no calcium channel blocker: these may worsen the situation.

Take home message:
1. Children are prone to have systemic poisoning.
2. ‘Autonomic storm’ of parasympathetic stimulation- e.g., sweating, cold extremities, hypotension, priapism etc. are common features.
3. Pulmonary oedema is prognostically bad.
4. Prazosin is the key drug. (Don’t use Prazosin sustained release tablet, use scored tablet)

Iron Poisoning

Source:
Iron tablet (commonly supplied to expectant mothers or children from health centers), iron syrups. Tablets or capsules are radio-opaque, syrups not.

Toxic dose:
More than 60 mg/kg of elemental iron.

Toxic effects: locally on gastric mucosa and systemically by free iron in circulation.

Clinical features: In the following stages:-
(i) Stage of gastric irritation: within first 6 hours.
   - Recurrent vomiting and abdominal pain most common.
     If no such symptom within 6 hours significant toxicity is unlikely
(ii) Relatively stable stage: 6 hours post-ingestion up to 24 hours.
     Gastro-intestinal symptoms subside and the child is mostly asymptomatic at this stage.
(iii) Stage of shock: 24 hours post-ingestion up to 48 hours.
- Shock, gastro-intestinal bleeding and metabolic acidosis. Shock is due to hypovolaemia added by myocardial dysfunction.

(iv) Stage of hepato-toxicity: After 48 hours post ingestion.
- Deranged LFT with prolonged PT.

(v) Stage of scarring of stomach and upper intestine: within 2 – 6 weeks following ingestion.
- Scarring and strictures are formed.

Investigations:
- Straight X-ray abdomen: Iron capsule or tablets are visible. History of ingestion of iron tab/cap + presence of radio-opaque shadow on X-ray + presence of metabolic acidosis indicate significant toxicity.
- Serum iron level, if facilities permit. It predicts prognosis also. Less than 350 mcg/ dl = mild toxicity
  350 - 500 mcg/ dl = moderate toxicity
  More than 500 mcg/ dl = severe toxicity.
- Blood sugar usually moderately high
- CBC – usually leucocytosis
- Serum electrolytes and bi-carbonate
- LFT
- Blood Urea, creatinine.

TREATMENT:

1. Decontamination:
   - NG tube aspiration/ lavage : Limitation- tablet parts may not be taken out through the tube (size dependent)
   - Activated charcoal: no role as it does not absorb iron
   - Gastric lavage with iron binding agents – not recommended.
   - Whole bowel irrigation with Polyethylene glycol + electrolyte solution @ 30 ml/kg/hr up to a maximum of 500 ml/ hr, till rectal effluent is clear. This is best for removing iron from g-I tract. However serum electrolytes should be monitored.

2. Antidote: Desferrioxamine iv infusion:

   Indication: Serum iron level more than 500 mcg/ dl / OR less than that but presence of systemic toxic symptoms.

   Dose: 15 mg/kg/hr till there is reversal of metabolic acidosis and the child is clinically well.
Take home message:

1. Accidental ingestion in children can occur.
2. Presence of recurrent vomiting and abdominal pain within 6 hours of ingestion indicates toxicity.
3. Toxic dose is more than 60 mg/kg elemental iron.
4. Activated charcoal or gastric lavage with iron binding agent not recommended.
5. Bowel wash with polyethelene glycol + electrolytes can remove iron from gut.
6. Specific antidote is Desferrioxamine i.v. infusion.

Poisoning by Household insecticide / mosquito repellant liquids / solids

(Pyrethrin and synthetic pyrethruids)

Pyrethrins may cause allergic reactions. Synthetic pyrethruids usually do not cause allergy but can cause systemic toxicity if exposed to larger quantity.

Features of toxicity:

If ingested: Nausea, vomiting, drowsiness, altered mentation, convulsion, coma, pulmonary wheeze and oedema.

If dermal exposure: paraesthesia

If ocular exposure: lacrimation, pain, photophobia, chemical conjunctivitis.

Treatment:

1. Decontamination:
   - Activated charcoal- 1 gm/ kg
   - If hydrocarbon solvent is present- No stomach wash.

2. Systemic toxicity: Symptomatic and supportive management according to clinical features. No specific antidote.

Ingestion of disc/ button/battery

- Majority of children are asymptomatic and the battery is passed through stool by 2 days - 7 days.
- But in small children with bit larger battery ( more than 15 mm diameter) it may be lodged in the oesophagus. Location should be ascertained by X-ray. Such lodged battery is of serious concern as the caustic material of the battery may cause local burn injury and later on stricture formation. Such battery in oesophagus should be removed by flexible endoscopy.
- In all children below 6 years of age with a battery in stomach should have a repeat X-ray after 48 hours. If the battery has not yet passed pylorus, endoscopic removal should be done.
Take home message:

1. Majority passes through stool by 2 – 7 days
2. If stuck in the oesopagus or more than 48 hours in stomach endoscopic removal should be done, as caustic material from battery can cause local burn and subsequent stricture formation.

Naphthalene Poisoning

Source: toilet bowl – deodorant, mothball.

Mode of poisoning: by ingestion of balls, inhalation/ exposure to blankets or clothes stored with naphthalene. Consumption of more than one ball is always producing symptoms. The fatal dose is usually 2 gm and fatal period is 2–3 days.

Mechanism of poisoning: The toxic metabolite is alpha naphthol which causes haemolysis and hepatic necrosis. The haemoglobin liberated by acute haemolysis can block renal tubule and cause renal failure. The haemolysis is particularly common in subjects of G6PD deficiency.

Clinical features:

Haemolysis starts within 24 hours and positively presents within 3 days depending upon G6PD status. Acute haemolytic anaemia, haemoglobinuria and jaundice are the common features.

Other features are ingestion related gastric features like nausea, vomiting and pain abdomen. There can be optic neuritis and acute nephritis. In severe cases the fatal features are convulsion, coma and death.

Inhalation results in headache, nausea, vomiting, mental confusion or vision problem.

Laboratory feature: Peripheral blood showing anisocytosis and fragmented red cells and methaemoglobininaemia. Urine shows haemoglobin and albumin. Blood will have increased unconjugated bilirubin. The direct Coomb’s test will be negative.

Treatment:

- Gastric decontamination: Gastric lavage with warm water. Activated charcoal (though not of proven value).
- Whole bowel wash with polyethylene glycol - only with large ingestion.
- Milk and fatty food to be avoided for 2-3 hours as naphthalene is fat soluble.
- Urine to be made alkaline by IV fluid + sodium bi carbonate to prevent precipitation of crystals of acid haematin in renal tubule.
- Patient should be closely monitored for haemolysis for a week:
- If no evidence of haemolysis within 3 days = low risk patient
● If evidence of haemolysis is present treatment to be provided as
  o If severe anaemia – PRBC transfusion
  o Renal flush with IV fluid and diuretic
  o Hydrocortisone may limit haemolysis
  o Methaemoglobinemia to be treated with methylene blue

● Renal failure or hepatic failure to be treated as per standard guidelines.

**Take home message:**

1. Naphthalene can cause severe acute haemolysis in G6PD deficient subjects. No specific antidote
2. Symptomatic therapy to be done
3. Urine should be made alkaline.
When a patient presents to you with a failed suicide attempt, it is essential to determine the risk of future suicide attempts after the patient has been stabilized through adequate medical management of the poisoning.

The most important step in the assessment is “engagement”. Engagement means to what extent the clinician is able to build trust with the patient so that the later may confide private information and distress with him. Without proper engagement, the information collected during suicide assessment will be incomplete and unreliable. The best way to develop a good rapport is to be non-judgmental and show genuine interest and concern for the person’s situation. After engagement when the patient is calm and relaxed, the assessment proper begins.

First of all, the clinician should enquire about all the risk factors for suicide present in the patient. Risk factors include presence of psychiatric disorders, specially mood disorders, psychosis or substance abuse.

Family history of psychiatric disorders or suicide attempts should also be enquired. Main psychological symptoms, which pose a risk, include loss of pleasure in daily activities (anhedonia), hopelessness, anger, impulsivity, anxiety and insomnia.

Specific enquiry should be made about ongoing medical illness (chronic) and any recent stressful event including domestic violence, shame, humiliation, financial loss, social isolation and loss of relationships. Married women, especially with adjustment issues with husband and in-laws, form a high-risk group in our country.

Past suicide attempt is one of the strongest risk factors for an impending attempt and in such cases, definite enquiry should be made regarding access to lethal means including pesticides, other poisons or sharp objects.

While enquiring about risk factors, special note should be made of modifiable risk factors since these can be targeted during management plan (e.g. availability of pesticides within reach). After noting the risk factors, one should try to identify the protective factors that are present in the patient. This includes family and community support, access to medical and mental healthcare facilities, skills in problem solving and methods of coping with stress. Two strong protective factors are presence of young and dependent children at home and harboring religious beliefs, which discourage acts of self-harm. Management plan should always focus on attempts to enhance the protective factors.

After this stage, suicide enquiry is conducted. Suicide enquiry involves asking about suicidal thoughts and ideations. If the patient agrees to the presence of those thoughts, one must enquire how frequently do they occur and for how long they have been occurring. It should also be enquired whether the patient has any definite plans of committing the act and if so, how, when and through what means. It should be enquired how strong is the patient’s intent or desire to end his life and how dangerous or lethal is the means by which he intends to kill himself.
Finally, an attempt should be made to assess the patient’s ambivalence or hesitation to die by briefly asking about his reasons to live versus reasons to die. For completing the assessment, corroborative information should always be sought from family members and other sources as far as possible.

Finally, level of suicidal risk is determined based on clinical judgement after weighing the risk factors, protective factors and suicidal thoughts and/or plans.

- Those having severe psychiatric illness and/or acute precipitating event and a near-lethal suicide attempt or strong intent to carry out the act would fall in the high risk level.

- Those having multiple risk factors, few protective factors, definite suicidal idea or plan but no strong or definite intent would fall into moderate risk level.

- Those with modifiable risk factors, many protective factors, death wishes and/or suicidal ideas but no definite suicidal plans would fall in the low risk level.

In our case, when a patient of attempted poisoning has been brought to the emergency room for evaluation, just the fact that an attempt has been made does not place him in the high-risk level. One has to determine whether there was intent to die by committing the act or it was a sudden, impulsive attempt without any intent to die.

**Presence of a strong intent together with coexisting suicidal plan will be required to qualify for a high-risk level!**

- If the patient falls in the high-risk level, admission is generally indicated. Methods of crisis intervention should be started along with treatment of co-existing psychiatric illness, if any.

- In cases of moderate risk level, admission may be necessary depending upon risk factors and presence of support system. A crisis plan should be initiated in consultation with family members.

- In cases of low risk, outpatient management with regular follow-up should be advised. Contact numbers, which can be used in moments of crisis, should be given to all patients and their family members.

Reassessment of suicidal risk should be done across all levels- within 24 hours in case of high risk, 7 days in case of medium risk and 1 month in case of low risk. Since the first 28 days after discharge from hospital are especially risky, assessment of even a low-risk patient after discharge should be done within a week.
Approach for psychiatric assessment in case of attempted suicide cases

**Engagement:** Show genuine trust and concern, build rapport

**Assess risk factors:** Past attempts, major mental illness, substance abuse, stressful events, impulsivity, anhedonia, hopelessness, chronic medical illness

**Assess protective factors:** Strong family & community support, access to health care, young children at home, ability to cope with stress

**Detailed suicide enquiry:** Presence of suicidal ideas, definite plans, suicidal intent, lethality, ambivalence regarding carrying out plans

**Prioritize**

**HIGH Risk:** Definite plans, recent attempt:
Hospitalize

**Medium Risk:** Many risk factors, strong suicidal ideation: Consider hospitalization, mobilize family members

**Low Risk:** Regular Follow up
CASE STUDIES

CASE 1

A 4 year old boy has been taken to the ER with a history of scorpion sting about 3 hours back. Hardly any sting mark is visible. The mother has seen the scorpion which was red in colour. The child is very much irritable. There is excessive sweating all over the body, The periphery is cold and BP is 86/56.

(a) What should be your first line of management?
(b) Mention the specific therapy.
(c) How will you monitor the child for further course of management?

CASE 2

A 4 year old girl has accidentally ingested an estimated 20 tablets IFA tablet supplied by local health centre for the mother. The child has been taken to hospital about 3 hours after ingestion of the tablet due to pain abdomen and recurrent vomiting.

(a) How will you assess the child?
(b) What should be the immediate management?
(c) Name the specific antidote. How will you administer the antidote?
(d) Briefly outline the systemic toxicity of the drug.
(e) Will you suggest any investigation?

CASE 3

A 35 year old male farmer was brought to the emergency room by his relatives. He was found in a semiconscious state in his room and on examination, his GCS was 10/15. There was strong smell of kerosene from his mouth, his pupils were constricted and non-reacting and there was excessive salivation and secretion. You have provisionally diagnosed the case as of Organophosphate poisoning.

What will you do as an attending Medical Officer?

1) Will you book this case as a Medico Legal Case’?
2) The relatives have said that the person has consumed OP on his own i.e, a case of attempted suicide and requests you not to inform the Police – Will you comply?
3) What are the samples you will preserve during treatment and what will you do with them?
4) If the investigating officer seeks any information regarding this case, what will be your duty as an attending physician?
5) If the condition of the patient deteriorates and you suspect that the death may be imminent – what will be your duty as attending doctor?
6) In case of Death of the patient what will be your duty as attending doctor?
Case 4

A 50 year old farmer was brought in ER in semiconscious state-he was found to be lying in his field and discovered by his son. According to his son, he was working very hard because of loss in previous year. On examination-the attending doctor finds that he had BP-70/50 mmHg, pulse-50 bpm, chest full of crepts, pupil-constricted, extremities moist and cool.

- Can you suspect any poisoning?
- The attending doctor told you that the patient responded after 10 vials of atropine given by nurse but has been drowsy again. What can be the possible reason?
- What should be the antidote?
- The attending doctor told you that his pupils have dilated and periphery became dry after 20 vials of atropine. Is it adequate?

Case 5

A 24 year old girl had taken a suicide attempt by ingesting some bathroom cleaner. His brothers bring the patient to you in 1 hour. They have told that they have tried to neutralise it by milk but the girl was not able to swallow and urges you to do a “wash” as they have seen in TV.

- Are you going for NG tube wash?
- What precautionary measure to take if the relatives pressurise you?
- What intervention is ideal in this early presentation?