Handbook of Poisoning Management

Primary Care to Tertiary Care

NATIONAL HEALTH MISSION
TAMIL NADU

Handbook of Poisoning Management Primary Care to Tertiary Care

Tamil Nadu Accident & Emergency Care Initiative
TAEI - Toxicology

Thiru. MA. Subramanian
Minister of Health and
Family Welfare

Secretariat
Chennai - 09.

Foreword

Over the past decade, poisoning has become an increasing cause of concern in India and the world at large. In Tamil Nadu nearly 1400 people die of suicides in a month of which Ingestion of poison (34%) is the predominant cause of concern next to Hanging (51.2%).

On account of this alarming rise in accidental and intentional /suicidal poisoning, the Government of Tamil Nadu is now taking scientific innovative strategies to curtail the access to the poisonous agents in collaboration with various stake-holding departments like Agriculture, Pesticide control, National and international experts.

The recent Announcement (No.114 dated April 29th 2022) made by me in the floor of the legislative assembly stating "Steps will be taken to Ban the sale of Rat killer Poison across the State" is a milestone in this regard.

Parallelly, National Health Mission, through the Tamil Nadu Accident & emergency Care initiative (TAEI) has provided necessary infrastructure, equipment and consumables in the Emergency Department/ Room in all 86 TAEI centers to ensure assured emergency care services to the general public. Training for the doctors, nurses and paramedic is being provided to ensure timely treatment to reduce the deaths due to poisoning and suicides. However, it was considered important to update and upgrade the Poison Management Protocols of the State which was prepared a decade ago.

In this regard, I appreciate Tmt. Shilpa Prabhakar Sathish IAS., and team for taking efforts to update the Poison Management Protocols in the State.

I also congratulate the team of experts from Govt and private hospitals from across the State and country who have come up with this easy-to-follow ready reckoner & Flow Charts for management of commonly encountered Poisoning in our State.

I hope this will benefit the health care community at large in providing state of art services in our Government hospitals. Best wishes,

Thiru. M. Subramanian

Dr. P. Senthil Kumar, IAS.,
Principal Secretary to
Government,
Health & Family Welfare Dept.,
Govt. of Tamil Nadu.

Secreatirat
Chennai - 600 009
Phone: 91-44-2567 1875
Fax: 91-44-2567 1855
E-mail: hfwsec@gmail.com

Foreword

Tamil Nadu a probably the first state in India to have identified poisoning as a serious public health issue requiring focused attention. It is in this light that the Govt. of Tamil Nadu and the National Health Mission has taken up this initiative to reduce deaths due to poisoning by conducting awareness to general public and ensure assured Emergency Care Services at the Govt. hospitals.

I congratulate the efforts taken up by our Expert committee for coming up with this clear concise and comprehensive text for management of poisoning in the form of simplified flow chart which can be applied at all levels of practice.

This booklet summarizes the essential elements of care of commonly encountered poisoning in the emergency departments of our state. It is more focused on pre-hospital and primary care management as they are the first encounters of poisoning in majority of cases and also on the minimum care to be ensured at the hospitals before referral to higher facility in case of complications.

I hope it will be very useful for the readers to put in practice these standard operating protocols for management of poisoning at all levels of practice. Best Wishes

Dr. P. Senthil Kumar, IAS.,

Tmt. Shilpa Prabhakar Sathish IAS.,
Mission Director &
Commissioner of Trauma Care
National Health Mission

National Health Mission
5th Floor, DMS Annex Building,
Chennai - 600 006
Tel. Off : 044 - 2433 6747
Fax : 044 - 2432 0563

Message

Today, one of the most critical problems that challenges the health care system in the State is "Poisoning and Self harm". Everyday there are 300 survivors of self-harm wheeled into our Emergency Departments in the State for whom prompt emergency medical and psychological health services are being provided at our TAEI centers (Source: TAEI app data).

There are 86 TAEI centers across the State which are equipped with adequate infrastructure for resuscitation, equipment and consumables to manage emergencies. With specific reference to management of Poisoning, all the TAEI centers have been provided with decontamination room with shower, adequate antidotes, equipment and crash cart with consumables. For the year 2022-2023, to tackle the rising deaths due to Rodenticide Poisoning, Government of Tamil Nadu has taken up steps to create 'Plasmapheresis facility' in all Medical College Hospitals such that every district has one. The good news is that, Government of Tamil Nadu is simultaneously drafting on a bill to regulate the sale of pesticides and efforts are underway to ban the sale of rat killer paste poison in the State.

Though Poisoning is a very complex issue to be managed, "Training" is considered as the key component to gear the entire health care system towards tackling this issue right from first responders, emergency medical technicians of 108 ambulances, Doctors, nurses and paramedical staff for resuscitation and emergency care management at the hospital and treatment at higher center in case of referral due to complications. However, this requires for Standardization of Protocols and Guidelines for Poison Management to be uniformly followed across the State for effective outcome.

This task of bringing out the Standard Operating Protocols and Guidelines has been meticulously carried out by team of experts from Government and private health sector who have taken their time to compile this book with easy-to-follow flow charts and ready reckoner. I believe it will be beneficial to each and every reader to upgrade their knowledge to the latest protocols in Management of Poisoning for the ultimate benefit of the patients.

I encourage each and every doctor, nurse and support staff at the field level to read this booklet as the success of the program completely rests on effective implementation of these protocols in the field. I wish you all the best!

Tmt. Shilpa Prabhakar Sathish IAS.,

THE CHANGING SCENARIO

The practice of Medicine is constantly changing, and the same applies to poisoning management also.

There is a decreasing incidence of certain poisoning like organochloride compounds while on the other hand there is increase in rat killer, Hair dye, Paraquat, Artificial cowdung poisoning etc. Tamil Nadu is being the hub for agriculture, still these poisonings continue to increase in addition to insecticide poisoning, bites and stings. Several changes and challenges have taken place in the field of poisoning management following better understanding of pathophysiology and pharmacology of poisonous substances. It is imperative to update and create awareness of poisoning management at all levels especially at primary care levels where more poisoning cases are being received and managed. We have taken the effort to standardize the protocol for the management of poisoning which will ensure uniformity in management across the state. This book provides the general principles in management of poisoning, what are the do's and dont's in toxicological management, flow charts with more focus on prehospital care, management at primary levels and referral criteria for all types of poison. It also provides clear guidelines in certain areas of controversies in management with updated references based on several RCTs and Meta analysis then arrived to a common consensus by the experts in the field of toxicology. We thank the contributors for their inputs and guidance in preparing this manual.

I hope the readers will find this book very useful for the effective management of poisoning in their day to day practice.

With regards

Dr. S. Ragunathanan
Professor of medicine,
Madras Medical College and RGGGH,
Chennai - 3.
Co chairman – Expert Committee

Dr. P. Paranthaman
Professor of medicine,
Govt. Kilpauk Medical college,
Chennai - 10.
Chairman – Expert Committee

TAMIL NADU SHOWING THE WAY

It has been a privilege to contribute to the updating and revision of Tamil Nadu protocols for poisoning management (2022).

Tamil Nadu has identified poisonings as an important public health problem. It is in this light that the Government of Tamil Nadu government and the National Health Mission has taken forward the initiative to reduce poisoning deaths through public health measures and improvements in emergency management. Tamil Nadu is probably the first state in India to identify poisonings as a serious public health issue, requiring focused attention.

The Tamil Nadu initiative has focused on strengthening poisoning management at the level of primary care. A strong primary health care system supported by tertiary and secondary care, can provide effective poisoning management. This requires protocols, training, and infrastructure for poisoning management.

Doctors looking after poisonings should have knowledge about the general principles and specific management of common poisonings. This requires an understanding of the mechanisms of absorption, distribution, metabolism, elimination, and toxicity. Doctors also need to know about emergency management, decontamination, supportive measures, antidotes and elimination. Since the spectrum of poisonings is changing, doctors need to be constantly updated with relevant new information. The medical curricula in MBBS and MD has insufficient focus on poisoning care. Therefore updating of medical curricula and continuing medical education are necessary for improving poisoning management.

The Tamil Nadu poisoning protocols have several new elements such as the focus on primary care, the use of flow charts and tailoring evidence-based management appropriate to local context. The protocols includes sections on when to refer, when to contact a poison helpline, new poisonings and pediatric management.

These protocols are not comprehensive and do not cover all the poisonings, for which more detailed resources can be accessed. These guidelines have been developed by senior toxicologists in the state collating their knowledge and experience, through several review meetings and discussions.

These protocols should be used for training of doctors at primary and secondary level. They require periodical review on feedback from practicing doctors and new published information. There is also need to setup a Tamil Nadu poisoning registry to gather real time data on the causes of poisonings and deaths in the state. Tamil Nadu needs monitoring and evaluation to assess the impact of the current public health interventions towards reducing poisonings deaths.

With regards,

Anand Zachariah
Professor of Medicine
Christian Medical College,
Vellore - 632 004.

PROJECT COORDINATORS

Expert Committee for Poison Management Protocols

Dr. P. Paranthaman, Chairman, Dept. of Internal Medicine, Kilpauk Medical College Hospital.

Dr. S. Ragunanthanan, Co-Chairman, Dept. of General Medicine- Madras medical college and RGGGH.

Dr. Anandh Zachariah, Professor and HOD - Dept. of General Medicine, CMC, Vellore.

Dr. C. Rajendran, Senior Medical consultant - Billroth Hospital - Shenoy Nagar.

Dr. Thirumalai kolundu, Professor & HOD of General Medicine-SRM, Trichy.

Prof. IndhumathiSanthanam, Retired Professor & HOD –Pediatrics, ICH, MMC.

Dr. S. Chandrasekar, Professor & HOD Dept. of Internal Medicine, Stanley MCH, Chennai.

Dr. Namitha Narayanan, Associate Professor, Dept. of Internal Medicine, Stanley MCH, Chennai.

Dr. S. Senthilkumaran HOD Emergency Medicine, Manian Medical Center, Erode.

Dr. Narendranath Jena, HOD Emergency Medicine, MMHC Madurai.

Expert committee for Rodenticide Poison Management Protocols

Dr. Narayanasamy, Chairperson, Professor & HOD Dept. of Hepatology - RGGGH.

Dr. S. Ragunanthanan, Co-Chairman, Dept. of General Medicine- Madras Medical College and RGGGH.

Dr. V. Jayanthi- Professor, Sri Ramachandra Medical College Hospital, Porur.

Dr. C.E. Eapen, Professor & HOD - Hepatology, CMC Vellore.

Dr. Mangayarkarasi , Professor & HOD, Transfusion Medicine, Coimbatore Medical College Hospital.

Dr. Nirmala, Professor & HOD, Paediatric Gastroenterology, ICH, Egmore.

Dr. Jaswanth, Professor & HOD, Liver Transplant, Stanley Medical College Hospital, Chennai.

Dr. M. Edwin Fernando, Professor & HOD, Nephrology, Stanley Medical College Hospital, Chennai.

Dr. Venkatakrishanna, Professor & HOD, Ganstroenterology, Coimbatore Medical College Hospital.

Dr. M. S. Revathy, Professor, HOD Medical Gastroenterology Stanley Medical College Hospital.

Dr. Kandhasamy, Professor & HOD , Gastroenterology, Tirunelveli Medical College Hospital.

Dr.Umarani.R, Professor & HOD General Medicine, Govt. Raja Muthaiah Medical College Hospital, Cuddalore.

Dr. S. Chandrasekar, Professor HOD, General Medicine, Stanley Medical College Hospital, Chennai.

Dr. Ramani, Associate Professor, Gastroenterology, Govt. Rajaji Hospital, Madurai.

Dr. Ram kumar, Associate Professor, Gastroenterology, Thanjavur Medical College Hospital.

Dr. Sumathi, Associate Professor, Gastroenterology, Stanley Medical College Hospital, Chennai.

Dr. Namitha Narayanan, Associate Professor, General Medicine, Stanley Medical College Hospital, Chennai.

Dr. Senthil Kumar, Associate Professor, General Medicine, Govt. Trichy Medical College Hospital.

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We are very much grateful to The Principle Secretary of Health and Family Welfare Dr.P. Senthil Kumar IAS, for his encouragement , support and guidance to form the guidelines and protocol for the management of Poisoning.

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Our heartfelt thanks to Dr. Darez Ahamed IAS. The commissioner of Rural Department and Panchayat Raj, Panagal Park Chennai was instrumental in initiation and forming this experts committee. We also sincerely thank for the constant support and encouragement given to design this updated protocol for the uniformity in management of poisoning across the state and his vision is to make our state a model state for poisoning management in the country .

Our sincere thanks to The Director of Medical Education Prof. Dr. R. Shanthimalar for the constant support ,sharing wisdom and Valuable contributions in editing this book

Our sincere thanks to Prof. Dr. R. Narayana Babu – The Retired Director of Medical Education for the generous support, help and the guidance in framing this protocol.

Our heartfelt thanks to all our Expert committee members for having shared their knowledge, wisdom and expertise to make this book to be useful for all levels of practice from primary care to the tertiary care.

We are very much grateful to Prof. Dr. P. Balaji, Dean, Govt. Stanly Medical College, Prof. Dr. E. Therani Rajan, Dean, Madras Medical college and RGGH Chennai for their valuable contributions in sharing knowledge ,support and guidance in editing this book

Our sincere thanks to Dr. V.Rajendran Associate Professor of Medicine RGGH chennai, Dr.D.Ramesh Associate professor of Medicine, Prof. Dr.S.Geetha Professor of Medicine. Dr. C.S. Gauthaman, Associate professor of Medicine, Govt. Prof. Dr. K.E Govindarajulu Professor of Medicine Government Kilpauk Medical college Chennai for their contribution in sharing knowledge and wisdom in editing this book.

Dr. S. Ragunathanan
Co Chairman

Dr. P. Paranthaman
chairman

CONTENTS

1. INTRODUCTION

Introduction

According to the World Health Organization suicide is the fourth leading cause of death among those between 15- 29 years. In India suicide has become the number one cause of death among those aged 15- 29 years. In the past 3 years the suicide rate in India is increased from 9.9 to 10.4 per 1000,000 population. The most common method of suicide in INDIA (2019) was estimated to be Hanging 54% followed by poisoning 26%. In the practice of medical toxicology an immediate attention to the assessment of Airway, Breathing, Circulation and decontamination plays an important role in addition to assessing and monitoring the overall status of the patients. The vital signs are the essential part of initial evaluation of every patient and frequent monitoring of vital signs are necessary throughout the subsequent management which is key to the successful management in critically ill patients .

The supportive measures should supersede all other considerations in management of the poisoning patients. All poisoning patients will benefit from an organized and rapid clinical management plan. Once airway, breathing and circulations are addressed the focus may switch to confirmation of toxic ingestion and specific management issues based on the Toxidromes

The treatment goal of poisoning patients are supportive measures as mentioned earlier, prevention of further poison absorption by stomach wash and activated charcoal, administration of antidotes, the prevention of reabsorption by entero-hepatic circulation of poisons by gut dialysis(multiple doses of activated charcoal),skin contamination and enhancement of elimination of poisons by cathartics, whole bowel irrigation, dilution, forced alkaline diuresis and extra corporeal removal of poisons.

Often the history from accompanying persons such as family members,friends and prescribing physicians gives clue to diagnosis. The physical examination should also include evaluation of head trauma, focal neurological deficits ,needle tract marks, unusual odour from the patient and auscultation for lung signs. By and large, an overall comprehensive team approach is required to bring down the morbidity and mortality in Toxicological Emergencies.

2. GENERAL PRINCIPLES OF TOXICOLOGY

APPROACH TO POISONING MANAGEMENT IN PRIMARY CARE

Patient presenting with history of poisoning or suspicious poisoning

Assess and stabilise airway breathing circulation

Collect available evidence about time of consumption, Quantity, compound, its form, colour, use and symptoms (tablets, agrochemical, household chemical, plant)

Perform focused examination on clues and to identify toxidrome, appropriate lab tests (atropine challenge, cholinesterase*, blood or urine drug assays, electrolytes, ABG, ECG, chest X-ray etc.,)

Contact poison centre for if brand name available, toxidrome can be identified and specific guidance on management, If needed.

If specific compound is identified initiate specific management

- ABC
- Decontamination
- Antidote
- Elimination

If compound is unknown

Perform gastric lavage preferably within 6 hours* of ingestion and administer activated charcoal if there is no contraindication

Continue to monitor for early symptoms and signs of intoxication and provide supportive management

Continue to search for compound

Refer: After stabilization & Informing the referring centre

1. Hypotension or QT prolongation
2. Hypoxia, respiratory distress
3. Coma or agitation
4. Recurring seizures
5. Signs of severe poisoning
6. Lethal poisoning: rodenticide, paraquat, iron, Lithium

Discharge after clinical stabilisation and psychiatric assessment & Counselling

POISONING MANAGEMENT- DECONTAMINATION

Skin should be washed thoroughly with soap and water including genital area and clothes changed, if skin contamination is present

For Eyes exposure - cleaned using syringe with clear saline or water for injection for 10 -15 mins..

Gastric lavage

Indications:

1. Life-threatening poisoning and unconscious presentation within 1 hour with precautions to prevent aspiration
2. For Pesticide poisoning – gastric lavage is useful within 6 hours and can be extended even after 6 hours if clinical condition warrants.
3. Tablet poisoning with anticholinergic effects presentation within 4 hours.
4. Sustained release preparation, salicylates, heavy metals within 12 hours
5. Iron or lithium poisoning

Contraindications:

1. Corrosive
2. Comatose patients (secure airway before gastric lavage

Procedure

1. Left lateral position.
2. Use Ryle's tube
3. First aspirate the stomach contents and store the first content for medico legal purpose
4. Use normal saline or clean tap water, infuse 200 ml at time and aspirate. Repeat process till the aspirate is clear.

Complications:

1. Aspiration
2. Trauma

Activated charcoal 1 g/kg as a single dose for poisoning with significant toxicity, if ingestion less than 1-2 hours.

Multiple dose activated charcoal 1 g/kg q4h

Indication: oleander poisoning, carbamazepine, dapsone, phenobarbital, quinine and theophylline

Both can be given orally or through Ryle's tube diluted in 150ml of water

Contradindications: Ileus, vomiting, corrosive poisoning, kerosene poisoning

Polyethylene glycol (PEG)

Indications: poisonings where activated charcoal alone is not satisfactory.

1.Iron and lithium

2.Sustained release preparations (e.g. theophylline and verapamil)

3.Toxins that form pharmacobezoars (e.g. salicylates)

Procedure:

PEG Given either orally or through nasogastric tube at 2 litres per hour for 2-6 hours.

TABLET TOXIDROME IDENTIFICATION

Anti-cholinergic Toxidrome

Tachycardia, Hyperthermia, Mydriasis, Dry skin and mucous membranes, Decreased gastric motility, urinary retention

Atropine, Datura, antihistamines, TCAs, anti-psyhcotics, Carbamezapines Add

Cholinergic Toxidrome

Bradycardia, Hyper / Hypotension, miosis, Increased Secretions - salivation, lacrimation, urination, diarrhoea, bronchorrhea; fasciculations, paralysis

Organophosphates, Carbamates

Sympathomimetics

Tachycardia, Hypertension, Tachypnoes, Hyperthermia, Agitation / hallucinations, mydriasis, tremors, diaphoresis, seizures

Atropine, Datura, antihistamines, TCAs, anti-psyhcotics, Carbamezapines Add

Cholinergic Toxidrome

Bradycardia, hypotension, depressed mental status, hyporeflexia, ataxia

Benzodiazepines Tricyclincantidepressants (TCAs) Neuroleptics Anticonvulsants Alcohol and toxic alcohols opioids

AGROCHEMICAL TOXIDROME IDENTIFICATION

Organophosphates and carbamate

Increased secretions, pupillary constriction, fasciculations, Bradycardia (or tachycardia), CNS depression

Oral and pharyngeal Erosions, vomiting, gastrointestinal bleeding, Acute respiratory distress syndrome, pulmonary fibrosis, acute tubular necrosis,hepatocellular injury, and dermatitis

Pyrethroids

Paresthesia, Blurredvision, nausea, vomiting, diarrhea, abdominal pain, anaphylaxis, wheezing, dermatitis, palpitations, dizziness, convulsions, coma, pulmonary edema

Dizziness, hypertension, tachycardia, nausea, vomiting, agitation, fasciculations, seizures, ventricular fibrillation, myocardial ischemia, and acuter renalfailure

Glyphosate

Mucous membrane irritation, Vomiting, abdominal pain, dehydration, vasodilatory shock, pneumonitis

Nausea, vomiting, CNS depression, Methemoglobinemia - the clinical features of Methemoglobinemia include low Oxygen saturation, headache, lightheadedness, fatigue, irritability, respiratory depression, coma, Seizures

Phenoxyaceticacids 2.4-D

Mucous membrane irritation, abdominal pain, vomiting, diarrhea, coma, flaccid quadriparesis or hypertonia, hypermeflexia, clonus, extensor plantar responses, fasciculation, Myotonia, Extensive rhabdomyolysis, Hyperpnea, Hyperthermia, Mixed respiratory alkalosis and metabolic acidosis, renal injury and disseminated intravscular coagulation

Nausea, vomiting, salivation and diarrhea, Early onset coma with hypoventilation, Mydriasis, partial ptosis, hypermetria, tremors, ataxia, seizures, coma, Hypotension, Rhabdomyalysis.

ELIMINATION

Alkaline diuresis

- Mechanism: Alkalinisation of the urine increases urinary excretion of weak acids (e.g. salicylates, phenobarbitone).
- Indications: Salicylates, chlorpropamide, phenobarbitone and possibly the chlorphenoxy herbicides.
- Method : Each cycle consists of 500 ml of 0.9 % NS over 1 hour followed by 400 ml of 5% dextrose with 100 ml Soda bicarbonate over 1 hour and then followed by 500 ml 0.9% NS with 10 mEq of kcl over 1 hour.
- If urine output less than 100ml/hr- then inj. Frusemide IV stat to he given, if urine output not increasing give another dose inj. frusemide IV stat is give and even after that if it is less than 100 ml/hr stop FAD and plan hemoldialysis
- If urine out put more than 100 ml/hr then continue FAD (Patient may be catheritized for monitoring urine output)
- Look for Lungs signs to rule out Pulmonary edema during FAD cycle

Indication:
Copper sulphate
Phenobarbitone
Salicylates
Phenoxyaceate
Fluoride
Methotrexate

Contraindication:
Congestive heart failure
Renal failure
Cerebral edema

Haemodialysis

- Hemodialysis can be used for poisons which are water soluble, low molecular weight and have low volume of distribution.
- Indications: Ethanol, toxic alcohols (Methanol, Ethylene glycol, Isopropyl alcohol), Lithium, Salicylates, theophylline and phenobarbitone

Charcoal hemoperfusion

- Mechanism: Blood is pumped through a charcoal cartridge. Charcoal adsorbs the poison compound. Compounds that are removed must have affinity for charcoal.
- Indications: Carbamazepine, Theophylline and Paraquat (first 2-3 hours only)
- Adverse effects: Thrombocytopenia, consumptive coagulopathy and hypotension

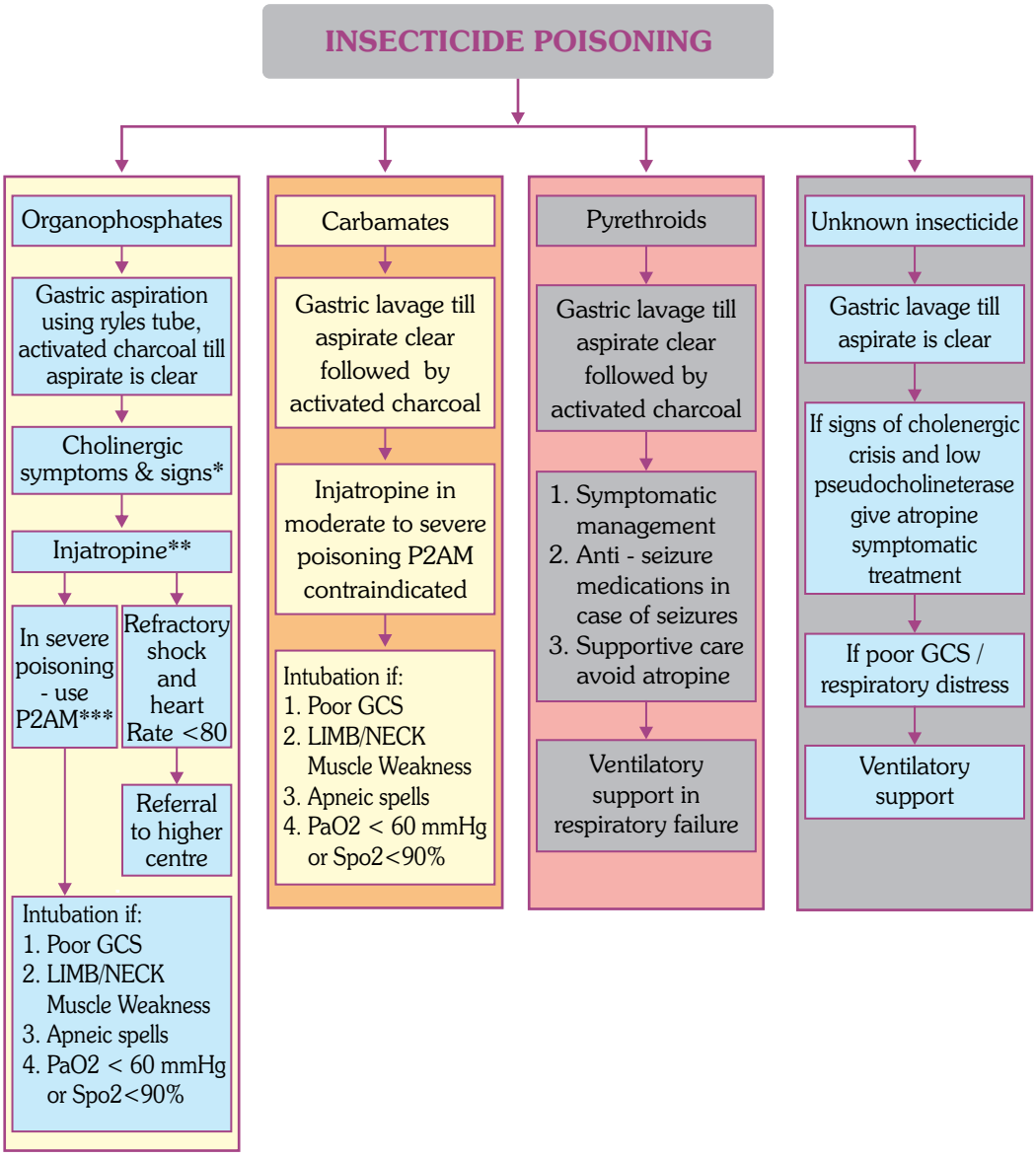
INDICATIONS FOR EXTRA-CORPOREAL REMOVAL THERAPY IN TOXICOLOGY

HEMODIALYSIS	HEMOPERFUSION
Acetaminophen (serum con >1000ug/ml)	Aminophylline
Alcohols Ethanol, lsopropanol, Acetone, Methanol, Ethylene glycol	Barbiturates Amobarbital, Pentobarbital, Phenobarbital, Primidone, Thiopental
Beta-blockers Atenolol, Nadolol, Sotalol, Metoprolol	Chloroquine, Dapsone
Metformin	Colchicine
Lithium	Digoxin, Diltiazem, Procainamide, Quindine
Salicylates	Diaquat, Paraquat, Parathion, Starfruit, Tetramine
Theophylline	Carbon tetrachloride, Dimethoate, Demetonsulphoxide
Valproate (serum con. > 1300mg/L}, Phenytoin {Moderate}, Carbamazepine	Amantin (Amanta Mushrooms), Organophosphorus
lsoniazid, Pyrazinamide, Ethambutol	Chlorpromazine, Promethazine
Dabigatran	Chloral Hydrate

ECRT not indicated: Tricyclic antidepressants, Diazepam, Midazolam, Methotrexate, Copper sulphate, Cisplatin, Rifampicin

* ECRT is indicated as primary therapy only if clinically warranted and/or if primary management falls.

3. INSECTICIDE POISONING


INSECTICIDE POISONING
Organophosphates
Gastric aspiration using ryles tube, activated charcoal till aspirate is clear
Cholinergic symptoms & signs*
Injatropine**
In severe poisoning - use P2AM***
Refractory shock and heart Rate <80
Referral to higher centre
Intubation if:
1. Poor GCS
2. LIMB/NECK Muscle Weakness
3. Apneic spells
4. PaO2 < 60 mmHg or Spo2<90%
Carbamates
Gastric lavage till aspirate clear followed by activated charcoal
Injatropine in moderate to severe poisoning P2AM contraindicated
Intubation if:
1. Poor GCS
2. LIMB/NECK Muscle Weakness
3. Apneic spells
4. PaO2 < 60 mmHg or Spo2<90%
Pyrethroids
Gastric lavage till aspirate clear followed by activated charcoal
1. Symptomatic management
2. Anti - seizure medications in case of seizures
3. Supportive care avoid atropine
Ventilatory support in respiratory failure
Unknown insecticide
Gastric lavage till aspirate is clear
If signs of cholenergic crisis and low pseudocholineterase give atropine symptomatic treatment
If poor GCS / respiratory distress
Ventilatory support

** Atropine:

- Atropine is given in cholinergic crisis.
- **Dose:** 1 to 3 mg every 5 minutes till atropinisation target is achieved (dose can be doubled to achieve atropinisation)
- **Targets:**
  a. SBP > 90 mm Hg
  b. HR > 90/min
  c. Clear chest
  d. Dry axilla
- After atropinisation maintenance infusion: 10-20 percent of the dose required for atropinisation – to be given for 48 hours in organophosphorus compound poisoning and for 24 hours in organocarbamate poisoning.
- The total duration of atropine therapy is 3 to 4 days for moderate to severe OPC poisoning and then it is tapered.
- Atropine toxicity: Stop Atropine infusion for 60 minutes and restart with 80 percent of the dose if following symptoms are observed - delirium , confusion, fever, absence bowel sounds, dry skin, urinary retention, hallucination)

Alternate drugs for atropine:

Glycopyrrolate. Patients with agrochemical poisoning which is not due to OP or carbamate poisoning should not be administered atropine as it may be harmful.

3.1 ORGANOPHOSPHATE POISONING

Prehospital Care:

1. Rush to nearby hospital
2. Try to get the label of poison
3. Rapid skin decontamination – remove the cloths contaminated with poison, tap water irrigation to clear poison over the skin, clear water irrigation of *eyes* if *eye* exposure is present .

At Primary Health Center:

Follow – A, B, C, D of poisoning management Assess for Airway, Breathing, circulation and maintain vitals Look for cholinergic, symptoms and sign (Urination, diahorrea, sweating , pin point pupil, bradycardia, bronchoconstriction, Emesis, Lacrimation, miosis)

Decontamination – skin and gastric decontamination And start antidote if indicated, focus on timely intervention – (TIME IS LIFE) when time is lost. Life is lost. Get toxicologist consult.

Diagnosis of OP poisoning is based on

a. Cholinergic crises and

b. Identification of the compound from the bottle label and or low cholinesterase value.

* In the absence of readily available cholinesterase estimation, a bedside Atropine challenge test may be performed. Atropine at dose of 1mg in adults or 0.01 to 0.02mg/kg in children is administered intravenously and patient heart rate is monitored. If the heart rate is increased by more than 25% of its baseline or more than 30 beats per minute, the test is considered positive and the patient has likely cholinergic toxicity.

When to refer	Where to refer	Things to be done in PHC
➢ Moderate / Severe poisoning (Peradeniya scoring) ➢ Impending respiratory Failure	➢ Availability of ICU Care	➢ Decontamination ➢ Securing airway ➢ Atropine administered

Peradeniya Scoring

PARAMETER	CRITERIA	SCORE
Pupil size	>= 2mm	0
	<2 mm	1
	Pinpoint	2
Respiratory rate	<20	0
	>= 20	1
	>= 20 with central cyanosis	2
Heart rate	>60	0
	41 to 60	1
	<40	2
Fasciculations	None	0
	Present, Generalized/ Continuous	1
	Both generalized and continuous	2
Level of consciousness	Conscious and rational	0
	Impaired response to verbal command	1
	No response to verbal command	2
Seizures	Absent	0
	Present	1

* Clinical response to atropine is not diagnostic of OP poisoning.

* Not all patients who present after pesticide poisoning with altered sensorium, vomiting, sweating, salivation and breathing difficulty have Organophosphorus poisoning.

Gastric Lavage And Activated Charcoal:

- Little benefit after 1 hour and no benefit of gastric lavage after 6 hours
- Gastric lavage is contraindicated in comatose patients
- The initial aspiration is probably of maximum benefit.
- Lavage can be done using Ryle's tube as an alternative to large orogastric tube.

- Activated charcoal is given as 1g /kg within 1 hour. Repeat the dose every 4 to 6 hourly for 24 hours until it appears in stool

Airway, Breathing And Circulation:

- Early mechanical ventilation in case of impending respiratory distress
- Assess the take of the world clinical severity for intubation:

1. RR> 30/min
2. PaO2 <60 mm/hg
3. Apneic spells
4. Poor GCS
5. Type I respiratory failure refractory to oxygen therapy,
6. Respiratory muscle paralysis (Type II respiratory failure- neck muscle weakness, reduced single breath count)

(1)* Pralidoxime:**

- The role of pralidoxime treatment is unclear.
- Benefit may be in patients with severe acute OP poisoning who present early (<6 hours) and with diethyl OP compound poisoning.
- Contraindicated in carbamates.
- Systemic reviews suggest that pralidoxime was associated with either null effect or harm.
- Side effects: Laryngospasm, Tachycardia, Muscle rigidity and allergic reactions.

Dose:

Pralidoxime: 30 mg/kg loading dose over 30 min followed by 8 mg/kg/h for 48hrs .

Note:

Knowing about the class of an OP compound is imperative because dimethyl and diethyl group of compounds differ completely in their ageing and reactivation kinetics. Dimethyl inhibited AChE enzyme reactivates and ages quickly, while these processes are slow for diethyl compounds. In patients presenting more than 6 hours of exposure to a poison, they would be benefitted only if the ingested compound was diethyl OP . Dimethyl OP would already have aged and it would be impossible for pralidoxime to reactivate it.

Pralidoxime may work when administered at the right time and appropriate dose. Probably, the treatment needs to be individualized taking into account the severity of poisoning symptoms, type and amount of OP compound ingested and the time elapsed since the poisoning.

The benefits of atropine in the treatment of acute organophosphate (OP) poisoning have been well established, while that of oximes is still uncertain. Pralidoxime is the most often used oxime worldwide. In vitro experiments have consistently shown that oximes are effective reactivators of human acetylcholinesterase enzyme, inhibited by OP compounds. The beneficial effect of oximes had been proven in animals and the optimum dose been identified, which was later extrapolated in humans. However, similar efficacy has not been demonstrated clinically.

Adrenaline:

- Start adrenaline infusion if Heart rate < 80/ min or refractory shock despite high doses of atropine (atropine dose of 100 mg within 6 hours of admission or an infusion of 30 mg/hr or more for at least 3 hours).
- Dose : 2-4 µg/min.

Ventilation:

The average respiratory rate of these patients increased from 22 to 38 breaths/min, which is an important sign of respiratory distress. Early recognition of respiratory failure resulting in intubation and mechanical ventilation is a life-saving intervention for patients with OP poisoning. Respiratory failure is the most troublesome complication, which was observed in OPC poisoning patients. Patients with OP poisoning may have respiratory failure for many reasons, including aspiration of the gastric content, excessive secretions, pneumonia and septicemia complicating acute respiratory distress syndrome, respiratory muscle paralysis.

Weaning: Follow weaning protocols for mechanically ventilated patients.

- Assess respiratory muscle performance before weaning off patient from mechanical ventilator

PARAMETER	WEANING THRESHOLD
PaCO2	<50 mmHg
Minute Ventilation (Spontaneous)	<10-15 L/min
Tidal Volume	>5 mL/kg
Maximum Voluntary Ventilation	>20 L/min
Respiratory Frequency	<35 breaths /min or >6 breaths /min

Seizures / Sedation:

Agitation and seizures: diazepam 10 mg slow IV push, repeated as necessary. Up to 30-40 mg diazepam per 24 hours can be given. Use diazepam infusion for status epilepticus.

Give general anesthetic agents (propofol, midazolam) if seizures are not controlled by diazepam.

Do not use Phenytoin, Haloperidol or Atracurium.

Monitoring:

Monitor every 15 minutes by OPC chart

Once atropinized, follow up hourly for 6 hours.

Then follow up every 2 hours once infusion is stopped .

Look for muscle paralysis every day – with neck holding time, limb muscle power. Intermediate syndrome should be diagnosed in patients with skeletal muscle paralysis between 24 to 96 hours after poisoning. Such patients can develop respiratory arrest and may require early intubation and mechanical ventilation

Other Treatments:

- If Atropine is unavailable, diphenhydramine and glycopyrrolate can be used in trials.

Prognosis:

- Death can occur within 24 hrs if untreated and within 10 days in treatment failure cases.
- Recovery occurs within 10 days but residual sequelae can occur.

Discharge Criteria:

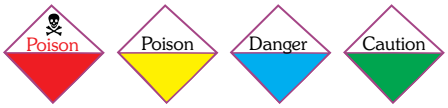
- Patient with clinically stable vitals and respiratory effort with single breath count more than 25 without neck muscle weakness, patient can be discharged with psychiatric counselling.

Referral Criteria From Primary Care:

1. Patients who require intubation and ventilation
2. Intermediate syndrome
3. High atropine dose requirement
4. Patients with severe poisoning (coma, hypotension, seizures, respiratory distress, paralysis)

Poison : Identification

WHO colour code on container



Red lable	Extremely toxic	Monocrolophos. zinc phosphide. ethyl mercury acetat
Yello lable	Highly toxie	Endosulfan, carbaryl. quinalphos and others
Blue lable	Moderately toxic	Malathion. thiram. glyphosate. and others.
Green lable	Slightly toxic	Mancozeb. oxyfluorfen. mosqui to repellant oils and hiquides, and most other household insecticides.

Sources of OPC:

- Garden sheds – In particular insecticidal preparations, but also other products that are marketed as fertilizers but contain some OP Pesticides, available as solid or liquid formulations
- Surface and Room Sprays
- Baits for Cockroaches and other insects eg.chlorpyrifos
- Shampoos against head lice eg.Malathion
- Pet preparations eg. Pet washes

- Industrial/Occupational- Crop protection, Live stock dipping, Fumigation
- Terrorism Or Warfare – Nerve Agents eg. Tabun and sarin

Classification:

The term organophosphorus (OP) compounds refers to any group of organic chemicals that contain phosphorus. The most predominant valances of phosphorus are 3 and 5. The majority of OP compounds (OPs) with environmental and industrial applications are of the pentavalent types. Organophosphates are a significant group of OPs which are essentially esters of phosphoric acids, in which the nature of the substituents attached to phosphorus plays a key role in determining the toxicity of the agents.

Phosphorylcholines	Flourophosphates	Cyanophosphates & Halophosphates	Multiple Constituents
Echothiopate	Dimefox sarin mipafox	Tabun	Dimethoxy, Diethoxy, Dialkoxy Diamino, Trithioalkyl, Triphenyl, Chlorinated, Mixed substituent

Classification Of OPC According to Their Group

Nerve Agents Include:

- GA (Tabun), GB (Sarin), GD (Soman), GF, and VX.
- These are potent organophosphorous agents cause inhibition of acetylcholinesterase and subsequent excessive muscarinic and nicotinic stimulation
- Organophosphorous can be conveniently classified according to their toxicity and use:

1. Highly toxic organophosphates: (e.g. tetra-ethyl pyrophosphates, parathion). These are mainly used as agricultural insecticides.
2. Intermediately toxic organophosphates: (e.g. coumaphos, chlorpyrifos, trichlorfon). These are used as animal insecticides.
3. Low toxicity: (e.g. diazinon, malathion, dichlorvos). These are used for household application and as field sprays

Low toxicity	Moderate toxicity	High toxicity
Bromophos	Acephate	Azinphos-Methyl
Eltrimfos	Bensulide	Bomyl
Lodofenphos	Chlorpyrifos	Carbophenthion
Malathion	Crotoxyphos	Chlorfenvinphos
Phoxim	Cyanophos	Chlormephos
Primiphos-Methyl	Cythioate	Cyanofenpos
Propylthiaopyrophosphate	DEF	Demeton
Temephos	Demaeton-S-Methyl	Dialifor
Tetrachlorvinphos	Diazinon	Dicrotophos
	Dichlofenthion	Disulfoton
	Dimethoate	EPN, Famphur
	Dichlorphos(DDVP)	Fenamiphos,
Fenophosphon	Edifenphos	Fonofos,
Isofenphos	EPBP	Isofluorphate,
Mephosfolan	Ethion	Methaidophos,
Methiadathion	Ethoprop	Mevinphos,
	Fenthio Fenitrothion	Parathion, Phorate
Formothion,	Heptenophos	Phosflan,
Phosphamidon	Heptenofos, IBP (Kitacin)	Prothoate
Schradan	Isoxathion, Leptophos	Sulfotep,
		Terbufos,
		Methyltrithion
Tetraethylpyrophosphate	Naled,	Oxydenprofos
Triorthocresylphoosphate	Oxydemeton—Methyl	
	Phenthoate, Pyridaphenthion	
	Phosalone Profenofos	
	Pyrazophos,Quinalphos	
	Sulprofos, Thiometon	
	Triazophos, Tribufos	

OPC Chart:

Name : **I.P No :**

Age & Sex : **Compound :**

Date & time	Heart Rate	BP	Pupil Size	Dry Axilla	Clear Lung	Bowel sound	Mental State	Fever	Spo2	Atropine dose	Other drugs	Remarks

Reference:

1. *Eddleston, M, Buckley, NA, Eyer, P & Dawson, AH 2008, 'Management of acute organophosphorus pesticide poisoning', The Lancet, vol. 371, no. 9612, pp. 597-607.*

2. IJCCM 2003:7(2) 85-87.

3.2 ORGANOCARBAMATES POISONING

Organocarbamate Compounds and Trade Name

Propoxur (Baygon)	Aldicarb (Temik)	Bendiocarb (Ficam)
Bufencarb	Carbaryl (Sevin)	Carbofuran(Furadan)
Formetanate (Carzol)	Methiocarb (Mesurol)	Methomyl (Lannate, Nudrin)
Oxamyl (Vydate)	Pinmicarb (Pirimor)	

Organochlorine Poisoning

Classified based on Toxicity

Low	Intermediate	High
Ethylan	Chlordane	Aldrin
Hcb	Ddt	Dieldrin
Methoxychlor	Hepatochlor	Endrin
	Kepone	Endosulfan
	Lindane	
	Mirex	
	Toxaphane	

Management:

Treated as per OPC Poisoning protocol except - pralidoxime - whish is contraindicated.

3.3 CRANE KILLER POISONING

Introduction

- Marketed as furadon and curater
- Odourless crystalline solids and soluble in water
- Carbofuran is one of the most toxic carbamate pesticides
- Group of salts or esters of N substituted carbamic acid
- Used as agricultural and household insecticides for the control of soil dwelling insects in maize, oilseed, sorghum, sugar beet, sunflower and some vegetables.
- Although OPC and carbamates are structurally distinct, they have similar clinical manifestations and generally require the same management.

Pathophysiology

- OPCs and carbamates bind to an active site of acetylcholinesterase and inhibit the enzyme causing excess of Ach in synapses and neuromuscular junctions resulting in muscarinic and nicotinic features
- The main difference in mechanism of action between OPCs and carbamates is that carbamates spontaneously hydrolyze from AchE site within 24 hours . And carbamates don't undergo aging, unlike Opcs.
- WHO classifies these poisonings as class 1 (extremely toxic) to class 3 (slightly hazardous) .

History

- Patients with carbamate toxicity usually have a history of exposure, which may be self injurious, occupational, environmental or exploratory in nature.

- Pesticides can be rapidly absorbed through skin, lungs, gastrointestinal tract and mucous membranes

- Carbofuran is most lethal when ingested.

- Patients present with evidence of cholinergic toxic syndrome

1. Nicotinic effects at NMJ and sympathetic ganglia _mnemonic:days of the week - MTWHF

- Mydriasis, tachycardia, weakness, hypertension and fasciculations

2. Muscarinic effects- SLUDGE/BBB

- Salivation, lacrimation, urination, defecation, GI symptoms, emesis, bronchorrhea, bronchospasm, bradycardia

3. CNS abnormalities -in severe carbofuran poisonings like convulsion and coma

- Death can occur due to respiratory failure

- Chronic toxicity:- C/f same as acute exposure. Chronic exposure can affect reproductive system and nervous systems

Diagnosis

- Known or suspected history of carbofuran use.

- Presence of cholinergic symptoms and signs.

- Because cholinesterase inhibition by carbofuran is rapidly reversible, cholinesterase testing may be unreliable in diagnosing carbofuran poisoning.

- High NLR ratio at hospital admission is reported to be an independent risk factor for mortality.

- Imaging-CXR in patients with respiratory distress due to bronchorrhea may show hazziness and pulmonary edema. Seriel cxr, with pulse oximetry and auscultation may guide therapy.

- ECG-Prolonged QTc interval;sinus tachycardia occurs just as commonly as sinus bradycardia; heart block can also be seen.

4. PLANT POISONING

4. GENERAL APPROACH TO PLANT POISONING

At the Level of Primary and Secondary Care

Introduction:

Plants are easily accessible and account for a significant number of poisonings presenting to the Emergency Department. The common plants used in South India for deliberate self-harm are Yellow Oleander, Oduvanthalai, Datura and Strychnos nux-vomica. Plants contain numerous classes of compounds. Major groups of classes of organic molecules that result in toxicity include alkaloids, glycosides, terpenes and resins; proteins, peptides and lectins; phenols and phenylpropanoids.

This approach is focused on common plant poisonings, **Oleander seed Poisoning (Yellow), Oduvnathalai poisoning, Datura poisoning and Abrus Precatareous Poisoning.**

History (what to ask)

1. Plant and plant part consumed. For oduvanthalai - whether consumed crushed or as a decoction. Quantity consumed and time since consumption.

   Positive identification of the plant species should be attempted whenever possible, especially when the patient becomes symptomatic. Expert identification should be sought from a Poison Control Centre to help identify unknown plant species. Laboratory analysis is generally not timely.

2. System specific symptoms.

Examination (what to examine)

1. Skin and oral cavity
2. Vital signs
3. Systemic examination

Investigations (to be requested)

1. Complete blood count
2. Blood glucose
3. Serum electrolytes
4. Creatinine/ Urea
5. LFT
6. Creatinine phosphokinase
7. ABG
8. ECG
9. Chest X ray

4.1 OLEANDER SEED POISONING (YELLOW)

It is a common plant consumed in rural south India for deliberate self-harm.

Photograph copyright: Poison Control Centre, CMC, Vellore

The toxic constituents are thevetin A thevetin B and neriifolin which are cardiac glycosides and found in all parts of the plant.

The plant:

The seeds are commonly ingested crushed, resulting in a clinical picture similar to digoxin overdose. Toxicity can also occur following cutaneous absorption and inhalation of smoke from burnt wood.

Clinical presentation:

Patients may be asymptomatic. In those who are symptomatic, the symptoms include:

Gastrointestinal: Nausea, vomiting, abdominal pain and diarrhea. Persistent vomiting may be seen in severe poisoning.

Cardiovascular: Chest pain, dyspnea, tachycardia or bradycardia, hypotension and cyanosis.

Respiratory: Tachypnea or bradypnea

Neurological: Weakness, dizziness, confusion and coma.

Dermatological: skin contact with the sap may produce dermatitis.

Laboratory abnormalities include hyperkalemia, hypomagnesemia elevated creatinine.

ECG changes include marked bradycardia ST segment changes and conduction blocks. In cases of severe toxicity ventricular tachycardia and ventricular fibrillation may occur.

Investigations to be done:

Complete blood count, capillary blood glucose, serum electrolytes, magnesium, creatinine and urea, LFT, ABG, Chest x ray, ECG.

Management

1. ABC – At presentation, the airway, breathing and circulation should be assessed and stabilised. Careful attention should be given to the level of consciousness, hemodynamic stability and the ability to protect the airway.

2. Continuous cardiac monitoring for a minimum of 72 hours in all symptomatic patients. Delayed cardiac effects may be seen up to 48 to 72 hours.

3. Decontamination

a) Gastric lavage: May not be useful.

b) Activated Charcoal: Activated charcoal (1gm/Kg) should be considered in patients who present within an hour of ingestion.

4. Antidote – Digoxin-specific antibody fragments.

5. Basic management and supportive care

1. Serum potassium should be monitored every 6 hours. Hyperkalemia should not be corrected. Hypomagnesemia should be corrected.

2. Shock should be aggressively managed with crystalloids and inotropes.

3. Renal function should be monitored by close charting of the intake and output and serum creatinine levels.

4. Temporary cadiac pacing: Bradyarrhythmias (HR < 40/min), sick sinus syndrome or heart block.

5. Ventricular tachyarrhythmias should be treated with Lidocaine.

6. Enhanced elimination – Multiple doses of Activated Charcoal have been used and may be considered.

When to refer: (Referred to Tertiary care centre as early as possible after stabilization)

1. Cardiogenic shock requiring inotropic support.

2. Respiratory failure requiring invasive mechanical ventilation.

3. Bradyarrhythmias requiring temporary cardiac pacing.

When to contact a Poisons Control Centre:

1. To help with plant identification
2. In patients with severe toxicity

References:

1. *Toxbase.org*
2. *Hypertox. Htm*
3. *Senaka Rakapakse. Management of Yellow oleander poisoning. Clinical Toxicology. 2009. 47:3, 206-212*

4.2 ODUVANTHALAI POISONING

It is the second most common suicidal plant poison in rural south India after oleander poisoning. Easy availability has resulted in it being preferred by young rural women.

Photograph copyright: Poison Control Centre, CMC, Vellore

The plant:

All parts of the plant are toxic and the leaves are commonly used. Parts of the plant may be crushed, leaves chewed or consumed as a paste or juice or boiled and the decoction consumed. The toxic constituents are Cleistantanthoside A and Cleistanthin A which are cardiac glycosides.

Clinical presentation:

Patients may be asymptomatic. In those who are symptomatic, the symptoms include:

Gastrointestinal:

Nausea, vomiting and abdominal pain. Less common symptoms are diarrhea, constipation, dysphagia, salivation, abdominal distension and decreased bowel sounds.

Cardiovascular:

Chest pain, dyspnea, tachycardia or bradycardia, hypotension and cyanosis.

Respiratory:

Tachypnea or bradypnea

Neurological:

Mydriasis, muscle cramps, weakness, altered sensorium, giddiness, headache, tremors and ptosis.

Laboratory abnormalities include hypokalemia, normal anion gap metabolic acidosis, leucocytosis, elevated hepatic transaminases, elevated creatinine phosphokinase, elevated cardiac enzymes, hyponatremia, hyperbilirubinemia and coagulopathy.

Investigations to be done:

Complete blood count, capillary blood glucose, serum electrolytes, Creatinine and urea, LFT, ABG, Chest x ray, ECG.

ECG changes include sinus tachycardia, sinus bradycardia, Ventricular premature complexes, ventricular fibrillation, flat "P" waves, Prolonged QT and QTc intervals, ST segment depression and inverted "T" waves.

Management

1. ABC – At presentation, the airway, breathing and circulation should be assessed and stabilised.

2. Continuous cardiac monitoring for up to 5 days in all symptomatic patients.

3. Decontamination

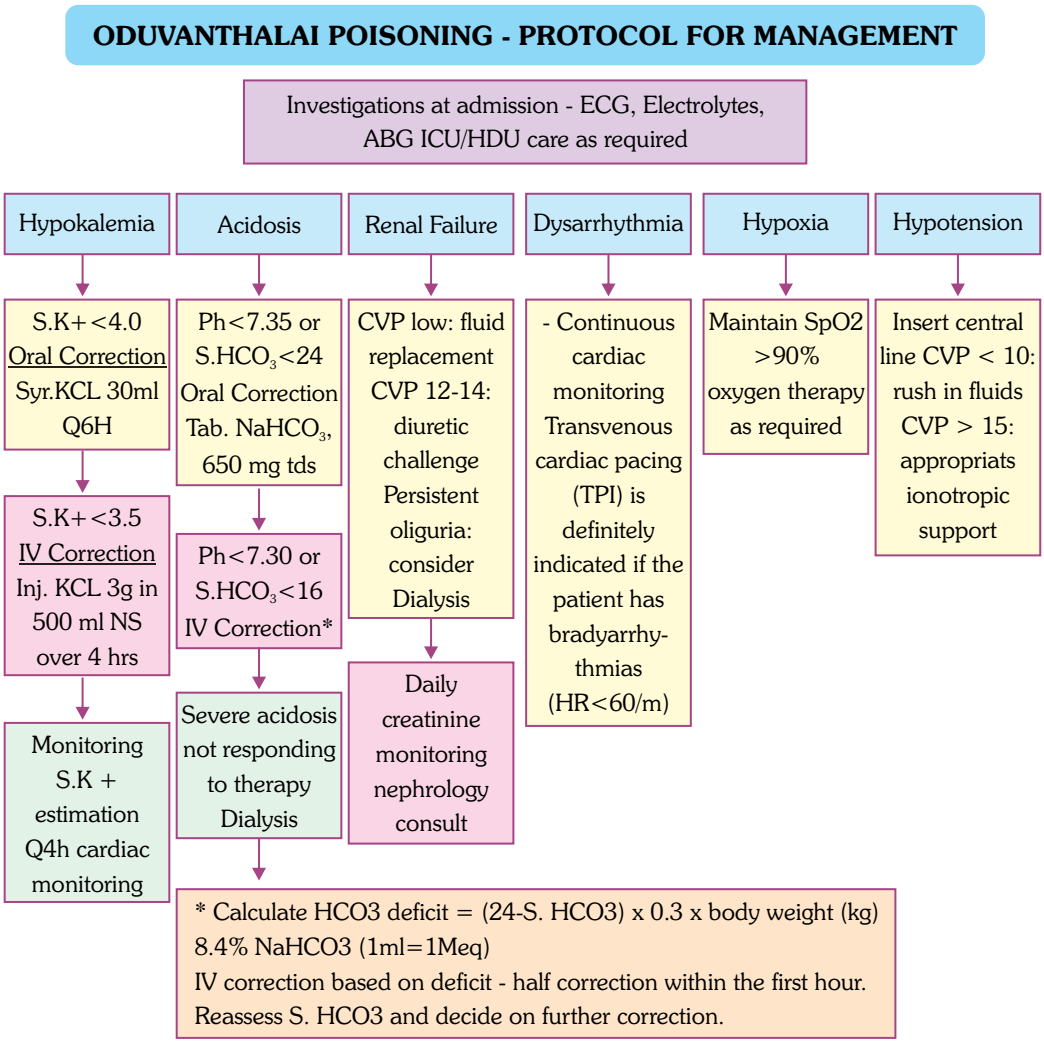
a) Gastric lavage: Can be done within an hour of consumption. It is unlikely to be beneficial 1-2 hours after ingestion in those who have consumed a decoction.

b) Activated Charcoal: A single dose of activated charcoal (1gm/Kg) may be considered.

4. Antidote – There is no known antidote.

5. Basic management and supportive care

1. The two important aspects of management is close monitoring and correction of hypokalemia and metablic acidosis.
2. Shock should be aggressively management with crystalloids and inotropes.
3. Renal function should be monitored by close charting of the intake-output and serum creatinine levels.
4. Temporary cardiac pacing: Bradyarrhythmias (HR < 60/min).



6. Enhanced elimination – No role in the management.

When to refer:

1. Refractory metabolic acidosis or persistent oliguria requiring dialysis.
2. Shock requiring increasing doses of inotropic support.
3. Respiratory failure requiring invasive mechanical ventilation.
4. Bradyarrhythmias requiring temporary cardiac pacing.

When to contact a Poisons Control Centre:

1. To help with plant identification
2. In patients with severe toxicity

Reference:

1. *Toxbase.org*
2. *Anugrah Chrispal. Cleistanthus collinus poisoning. Journal of Emergencies, Trauma and shock. 2012 Apr-Jun; 5(2): 160–166*

4.3 DATURA POISONING

Datura stramonium

The plant:

All parts of the plant are toxic. The toxic constituents are alkaloids with antimuscarinic properties and include atropine, scopolamine, and hyoscyamine. The flowers, stem, fruit/seeds, leaves and roots contain the alkaloids in decreasing amounts.

Mechanism of action

Anticholinergics block the neurotransmitter acetylcholine at muscarinic receptors causing toxicity in the peripheral and/or the central nervous systems

Clinical presentation:

Onset of symptoms: 1 to 4 hours post ingestion. Duration of effect – dose dependent (few hours to days).

Patients may be asymptomatic. Symptomatic patients classically present with an **anticholinergic** toxidrome - '*red as a beet, dry as a bone, blind as a bat, hot as a hare, mad as a hatter'*

Dermatological:

The skin and mucous membranes appear dry. The skin may appear flushed and warm. Hot and dry skin.

Gastrointestinal:

Dry mouth and tongue, nausea, vomiting, decreased bowel sounds.

Cardiovascular:

Transient bradycardia, tachycardia, hypertension

Respiratory:

Tachypnea

Neurological:

Blurred vision due to mydriasis, agitation, disorientation, hallucinations,

ataxia, speech disorders, seizures, myoclonus, hypertonia. In severe cases CNS excitation may lead to CNS depression with circulatory and respiratory failure and coma.

Other symptoms:

Hyperthermia, urinary retention, Skin contact may cause dermatitis and blistering and systemic features may arise.

Eye contact causes marked dilatation of the pupils and systemic features.

Laboratory abnormalities include hyperglycemia, abnormal renal and liver function tests and rhabdomyolysis.

ECG changes:

Sinus tachycardia. conduction abnormalities and arrhythmias may occur rarely.

Investigations to be done:

Complete blood count, capillary blood glucose, serum electrolytes, Creatinine and urea, LFT, creatinine phosphokinase, ABG, Chest x ray, ECG.

ECG changes:

Sinus tachycardia. conduction abnormalities and arrhythmias may occur rarely.

Management

1. ABC – At presentation, the airway, breathing and circulation should be assessed and stabilised. Careful attention should be given to the level of consciousness, hemodynamic stability and the ability to protect the airway.

2. Decontamination

b) Activated Charcoal: Activated charcoal (1gm/Kg) should be considered if the patient presents within an hour of ingestion.

3. **Antidote** – Physostigmine (may be lifesaving in patients with seizures or an agitated delirium. The dose is 0.5mg to 2mg (0.02mg/kg IV up to a maximum of 0.5mg per dose in paediatric patients), given as a slow push over 5 minutes. The dose may be repeated every 10 to 30 minutes.

4. Basic management and supportive care

a. Management is mainly supportive.

b. Hyperthermia (core temperatures more than > 40^{0}C should me managed

   a. aggressively with external cooling measures and include evaporative cooling with mist and fan and application of ice packs to the groin and axillae.

c. Shock should be aggressively management with crystalloids and inotropes.

d. The patient should undergo urinary catheterisation and the intake-output monitored.

1. Enhanced elimination: No role

2. When to refer:

a. Cardiogenic shock requiring inotropic support.

b. Respiratory failure requiring invasive mechanical ventilation.

When to contact a Poisons Control Centre:

1. To help with plant identification

2. In patients with severe toxicity for whom Physostigmine is being considered.

Reference:

1. *Toxbase.org*

4.4 ABRUS PRECATAREOUS POISONING

Introduction:

Abrus is a slender, twinning, ornamental, tropical plant belonging to the family Leguminosae and is commonly found all over India. It has tough slender branches with 5 to 10 cm long compound leaves bearing 10 to 20 pairs of leaflets. Leaves are alternate, opposite, pinnately divided (feather like) with small oblong leaflets. Stem is green when young and grey black when mature. Flowers are pink, purple or white and borne in clusters. Seeds are a very distinctive part of the plant, which is oval, 5mm in diameter and has an attractive glossy outer shell that is usually scarlet red with a black centre. Seeds are present inside fruit pods with each pod containing 3 to 5 seeds.

Toxic part: Seeds, Root, Leaves

Other Names:

Jequiry bean, Indian bead, Buddhist rosary bead, Rosary pea Seminole bead, Prayer bead, Jungle bead, Crabs *eye*, Weather plant, Love bean. It is called Kundumani in Tamil.

Toxic Principles:

Abrin – a toxalbumin, similar to viper snake venom

Abrine – N METHYL TRYPTOPHAN -An amino acid haemagglutinin in the cotyledons.
Abralin, a glucoside - A lipolytic enzyme
Glycyrhyzzin

ABRIN is the main active principle:

It's a lectin composed of two polypeptide chains A and B connected by di-sulphide bridges. Basic structure of this toxin is similar to that of Botulinum, Tetanus, Cholera, Diphtheria, Insulin

Mechanism of action:

ABRIN is a powerful gastro-intestinal toxin. Its polypeptide chain B binds to the intestinal membrane.

chain A enters cytoplasm

↓

acts on 60s ribosomal subunit

↓

Prevents binding of elongation factor 2

↓

Inhibiting protein synthesis and leading to cell demise

Toxin causes: Tissue edema, vascular leakage or capillary leak syndrome.

Fatal Dose:

1 to 3 seeds - 0.1mg / kg to 1 mg/kg is sufficient enough to cause death.

Fatal Period:

3 to 5 days

Clinical Manifestations:

Mainly gastro-intestinal with nausea, vomiting, abdominal pain, diarrhea and gastrointestinal bleeding. Diarrhea can occur even after 3-4 days and can be profuse. Other uncommon presentations include arrhythmias, encephalopathy, renal failure due to volume loss.

Systemic manifestations:

Hypotension. Acute renal failure, Hepatotoxicity, Intravascular hemolysis, Altered sensorium, Cerebral edema and Seizures. Delayed cytotoxic effects occurs in CNS, liver, kidneys and adrenal glands 2 to 5 days after exposure.

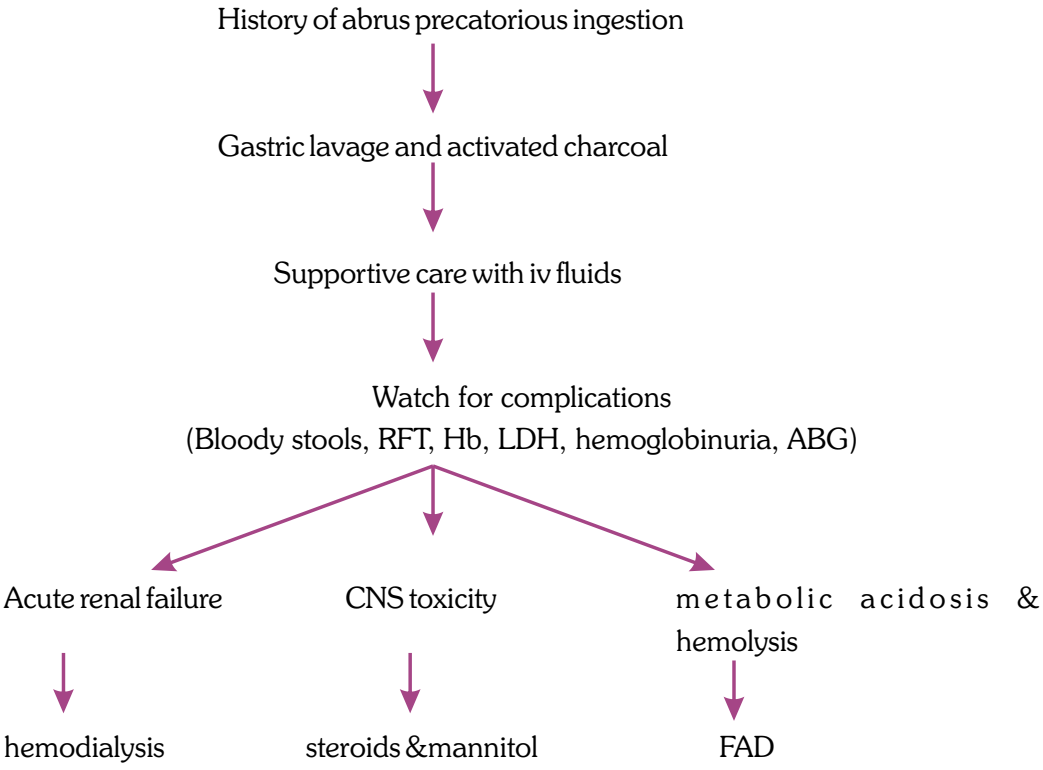
Treatment:

Gastric lavage, purgative and whole bowel irrigation, activated charcoal and supportive care.

Injection of anti-abrin.

IV methyl prednisolone and mannitol for CNS toxicity and hemodialysis for acute renal failure, diazepam for seizures, alkalinization of urine for preventing agglutination of red cells and blockage of renal tubules

Management Protocol:



Clinical Pearls:

1. Abrus is a slender, twinning, ornamental, tropical plant belonging to the family Leguminosae and is commonly found all over India. Toxic part: Seeds, Root, Leaves. It is called Kundumani in Tamil.

2. Toxic principles: Abrin – a toxalbumin, similar to viper snake venom.

3. Fatal dose: 1 to 3 seeds - 0.1mg / kg to 1 mg/kg is sufficient enough to cause death. FATAL PERIOD: 3 to 5 days

4. Clinical features: Gastro-intestinal with nausea, vomiting, abdominal pain, diarrhea and gastrointestinal bleeding.

5. Treatment: Gastric lavage, Injection of anti-abrin, hemodialysis for acute renal failure, diazepam for seizures, alkalinization of urine for preventing agglutination of red cells and blockage of renal tubules

References:

1. *Drug Design, Development and Therapy 2012:6*

2. *Arrowsmith JB, Kennedy DL, Kuritsky JN, Faich GA. National pat-terns of aspirin use and Reye syndrome reporting, United States, 1980 to 1985. Pediatrics. 1987;79(6):858–863*

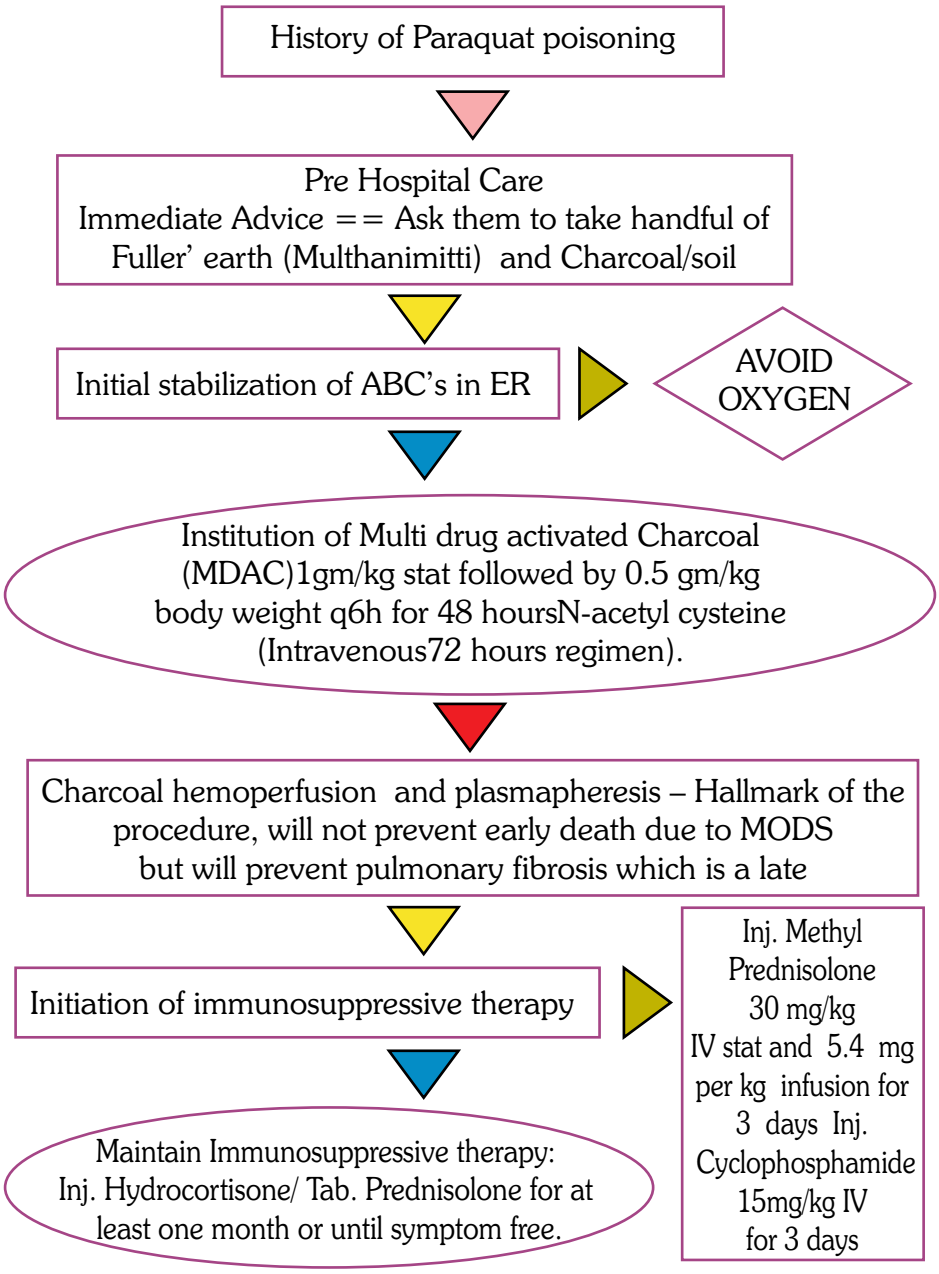
3. *Ismail M. The treatment of the scorpion envenoming syndrome: the Saudi experience with serotherapy. Toxicon. 1994;32(9): 1019–1026.*

4. *Nascimento EB Jr, Costa KA, Bertollo CM, et al. Pharmacological investigation of the nociceptive response and edema induced by venom of the scorpion Tityusserrulatus. Toxicon. 2005;45(5):585–593.*

5. *Bawaskar HS, Bawaskar PH. Severe envenoming by the Indian red scorpion Mesobuthustamulus: the use of prazosin therapy. QJM. 1996;89(9):701–704.*

5. WEED KILLER POISONING

5. WEED KILLER PARAQUAT POISONING



5.1 PARAQUAT POISONING

Paraquat Poisoning

Paraquat (1,1'-dimethyl-4-4'-bipyridinium dichloride) is a widely used herbicide that was introduced to agriculture in 1962. It is a non-selective herbicide and is commonly used in developing countries. Most cases of paraquat poisoning are caused by suicidal attempts rather than by accidental exposure.

Mechanism of Toxicity

Paraquat concentrates in (pulmonary) alveolar type I and II cells via an energy dependant transport system .High concentration of paraquat once accumulated into lung or renal cells results in redox cycling and generation of toxic reactive oxygen species. This can overwhelm cellular defence mechanisms and lead to lung damage (acute and subchronic) and renal tubular necrosis. The end result is high mortality rate due to

- Acute renal failure
- Acute lung injury and later fibrosis
- Multi organ failure and hemodynamic changes.

Low dose (< 20 mg paraquat ion per kg)-patients are often asymptomatic, or may develop gastrointestinal symptoms, but usually recover completely;

Moderate dose (20 to 40 mg paraquat ion per Kg)- Mucosal damage of mouth GIT, renal failure, Respiratory failure after few days but eventually die within few weeks due to massive fibrosis

High dose (> 40 mg paraquat ion per kg) Toxicity is severe and death occurs within hours from multiple organ failure.

Lab analysis

Urine spot test (alkali and sodium dithionite) as soon as – a negative urine test should be repeated at 6 hours post-ingestion and if this is still negative then serious sequelae.

Quantitative measurement in plasma gives a measure of severity and prognosis.

Urine Dithionite Test

1. A qualitative urine test for Paraquat, which detects concentrations of 1 mg/mL or above (1 ppm), can be made by adding 2 mL of a 1 percent solution of sodium dithionite in1N sodium hydroxide to 10 mL of urine

2. A blue colour indicates the presence of Paraquat

Management

- If patient presents within one hour activated charcoal can be given at 1 gm/kg. Stomach wash is unlikely of any benefit.
- Early insertion of nasogastric tube need to be considered for feeding purposes
- Early referral to higher centres.
- Symptomatic and Supportive management, no specific antidote. Nasal oxygen should be avoided until the patient is severely hypoxic as it can accelerate the lung damage.

- Haemoperfusion has been postulated as a treatment (enhanced elimination) for a number of years but its efficacy remains controversial.
- Hemodialysis is required in patients with Acute renal failure.

Inj Dexamethasone 8 mg IV q8h can be considered in patients with acute poisoning. If patient develops severe toxicity, this can be continued up to five weeks.

Tab Vitamin C 1 gm twice daily

6. CORROSIVE POISONING

APPROACH TO CORROSIVE POISONING- PRIMARY CARE

Eye splash
Irrigate *eye* with Tap water, Ringer's lactate, Normal saline
Using several litres for minimum of 15 minutes
Ophthalmic examination

Patient presenting with corrosive poisoning- establish identity of compounds

Triage with highest priority, stabilise ABC, establish IV line, give fluids to stabilise BP, NPO
Look for laryngeal oedema (stridor), intubate if required

Contraindicated : Gastric lavage, charcoal, neutralization, emesis

IV pantoprazole -80mg in 40 ml NS 0.9% at 5ml/hr
Parenteral Analgesics

Refer to secondary or tertiary level if there are oral burns, drooling of saliva, odynophagia, abdominal pain, breathing difficulty, hemodynamic disturbance, acidosis

Tests: CBC, electrolytes, creatinine, ABG, chest x-ray and ECG

If there are no oral burns, local or systemic symptoms oral feeding can be started in 24 hours

Aymptomatic adults with non-intentional poisoning - can be discharged if they can ingest fluids, normal speech after 48 hrs of Observation

SECONDARY CARE

ENT evaluation and early upper GI endoscopy (6-24 hours) if possible General surgeon/ surgical gastroenterologist opinion to be obtained

Steroids are generally avoided*
Parenteral antibiotics in case of infection or perforation

Grading on endoscopy

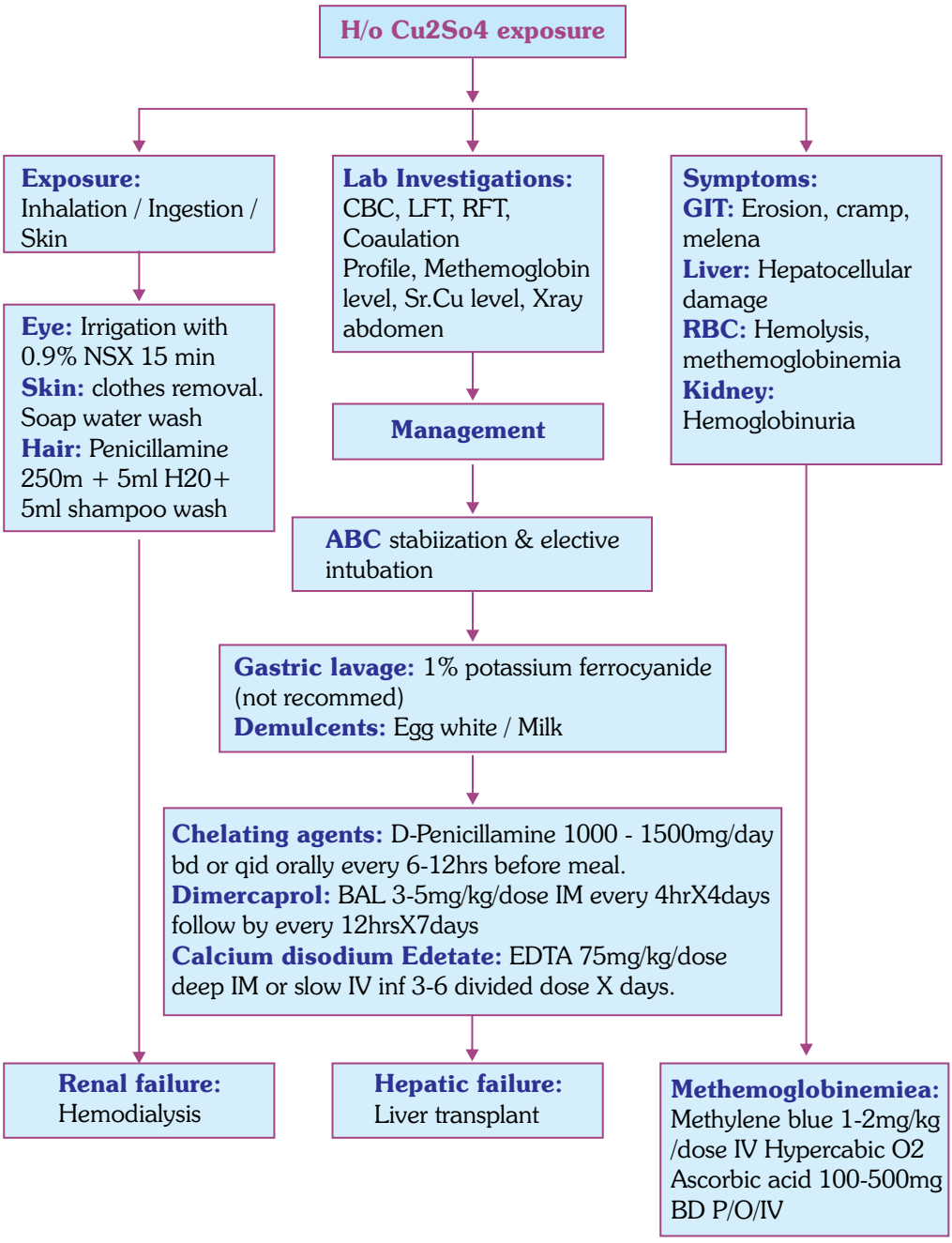
- Grade 1- Erythema
- Grade 2- Ulceration, submucosal lesions, haemorrage
- Grade 2 a –Not circumferential
- Grade 2 b – Near circumferential*
- Grade 3- Deep ulcers and necrosis into peri-esophageal tissue*

When to feed

- Grade 1- Start feeding on Day 1
- Grade 2a - Liquid food after 48-72 hours or ryle's tube passed under direct visualisation
- Grade 2 b or 3
  Nil oralNaso-gastric tube
  Feeding jejunostomy
  TPN

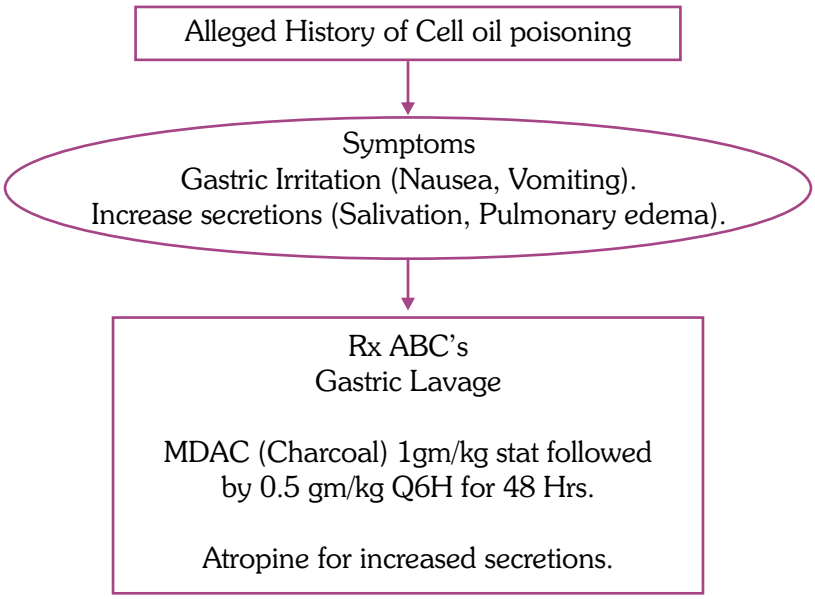
Plan barium swallow after 3-4 weeks

7. COPPER SULPHATE POISONING


H/o Cu2So4 exposure
Exposure: Inhalation / Ingestion / Skin
Eye: Irrigation with 0.9% NSX 15 min
Skin: clothes removal. Soap water wash
Hair: Penicillamine 250m + 5ml H20+ 5ml shampoo wash
Lab Investigations: CBC, LFT, RFT, Coaulation Profile, Methemoglobin level, Sr.Cu level, Xray abdomen
Management
ABC stabiization & elective intubation
Gastric lavage: 1% potassium ferrocyanide (not recommed)
Demulcents: Egg white / Milk
Chelating agents: D-Penicillamine 1000 - 1500mg/day bd or qid orally every 6-12hrs before meal.
Dimercaprol: BAL 3-5mg/kg/dose IM every 4hrX4days follow by every 12hrsX7days
Calcium disodium Edetate: EDTA 75mg/kg/dose deep IM or slow IV inf 3-6 divided dose X days.
Symptoms:
GIT: Erosion, cramp, melena
Liver: Hepatocellular damage
RBC: Hemolysis, methemoglobinemia
Kidney: Hemoglobinuria
Renal failure: Hemodialysis
Hepatic failure: Liver transplant
Methemoglobinemiea: Methylene blue 1-2mg/kg /dose IV Hypercabic O2 Ascorbic acid 100-500mg BD P/O/IV

8. CELL OIL POISONING

CELL OIL POISONING



CELL OIL POISONING

It is used in carpentry. It is applied over the wood surfaces (door, window) as antitermite. Exact nature of the compound is not known.

Clinical Features:

- Gastric irritation (nausea, vomiting),
- Increased secretions (salivation, pulmonary edema)
- Methemoglobinemia causing central cyanosis, altered sensorium and coma

Investigations:

- Routine investigations like CBC, RFT.
- X ray chest
- Arterial blood gas analysis (Normal PaO2, low oxygen saturation, metabolic acidosis)
- Methemoglobin level if possible

Management:

- Airway, breathing and circulation
- Gastric lavage and multidose activated charcoal.
- Treatment of methemoglobinemia S4 Methylene blue 1 - 2 mg/kg, upto Hax 5 mg/kg
- Supportive management like mechanical ventilation and atropine for increased secretion.

9. ARTIFICAL COW DUNG POWDER POISONING

Management of Cow Dung - Sani Powder (Yellow)

Gastric Lavage
MDAC (Charcoal) 1gm/Kg stat followed by 0.5 mg/kg Q6H for 48 hours.

↓

If Seizures Present:

- Inj. Lorazepam 4 mg IV stat and followed by 4mg thrice daily
- Inj. Phenobarbitone 20 mg/kg IV stat and 10 mg IV TDS for 3 days if refractory or persistent low GCS, intubate and ventilate

↓

Watch for

- Electrolyte abnormality.
- Respiratory Distress.

COWDUNG POWDER

Introduction:

The yellow and green cow dung powders are frequently consumed for suicidal poisonings in the region of central Tamil Nadu. It is locally known as "saani powder". These are traditionally inorganic dyes which are used for colouring the courtyards of houses. It is a modern substitute for the fresh cowdung solution (green) and cow dung and turmeric (yellow) solution, that is used for religious, sanitary and aesthetic purposes

Toxins:

Green powder (Malachite green – Triphenyl methane dye)

Yellow powder (AURAMINE O – Diaryl methane dye)

Mechanism of toxicity:

Auramine:

If causes DNA damage (Chronic exposure): It is carcinogenic on chronic exposure and has been linked to bladder cancer. Inhalational exposure can result in pneumoconiosis.

Malachite green:

Intercalates with DNA. Is cytotoxic and induces cell transformation and lipid peroxidation. It causes delayed toxicity. Its role as a carcinogen is equivocal.

Toxic dose: Not known

Clinical Presentation:

Gastrointestinal:

It is an irritant – nausea, vomiting, hematemesis, melena, epigastric pain, tenesmus, cramps, diarrhea

Hepatobiliary:

Auramine causes centrilobular necrosis of the liver;

Central nervous system:

Confusion, irritability, muscle cramps, decreased mental status, convulsions, coma.

Skin:

Discoloration of the body – especially hands, face and tongue. Auramine – Yellow; Malachite green – green

Other:

Tachycardia, metabolic acidosis, and hyperglycemia

Eye contact:

Auramine can produce conjunctival edema, hyperemia, purulent discharge and total opacification of the cornea.

Diagnosis:

Based on history and evidence of use of the specific compound.

Decontamination:

Stomach wash – If the patient presents within 2 hours of consumption.

Activated charcoal – Multiple doses (1gm/Kg every 6 hours) can be given for 24 hours.

Eye Exposure:

Remove contact lenses and irrigate exposed eyes with copious amounts of room temperature 0.9% saline or water for at least 15 minutes. If irritation, pain, swelling, lacrimation or photophobia persists after 15 minutes of irrigation, the patient should be seen by an Ophthalmologist.

Dermal Exposure:

Wash with copious amount of water. Remove contaminated clothing, wash exposed skin thoroughly with soap and water, and irrigate exposed eyes

Emergency and supportive measures:

Supportive therapy is the mainstay of treatment.

Monitor vital signs and mental status.

Treat hypotension with intravenous fluids, add vasopressors if hypotension persists.

Arrange for early (ideally within 24 hours) endoscopy for patients with concern for caustic GI injury. Treat bronchospasm with inhaled beta agonists and corticosteroids.

Seizures to be treated with benzodiazepines.

Endotracheal intubation and mechanical ventilation may be needed in patients with significant CNS depression or respiratory distress

Daily Monitoring:

liver function tests, PT/INR (may develop toxic hepatitis 2 to 3 days after consumption). Refer to a higher centre if the patient develops fulminant liver failure.

Antidote:

There is no specific antidote available.

Enhanced elimination:

Urinary alkalinization has been suggested by Senthilkumaran et al as a means of promoting solubility of the dye (Malachite green is a weak acidic) and subsequent urinary elimination.

10. HAIR DYE POISONING

HAIR DYE POISONING - PROTOCOL FOR MANAGEMENT

Toxic Components: Paraphenylene diamine, Resorcinol, Sodium ethylene diamine tetra acetic acid and Propylene glycol

Target Damage:

1. Cervico facial edema → Respiratory compromise
2. Rhabdomyolysis → ATN+ ARF

± Hypocalcemia

ICU / HDU

CERVICO FACIAL EDEMA

Tracheostomy:
Stridor
Severe oedema with impending respiratory compromise

* Steroids may be administered to reduce the inflammatory oedema

RHABDOMYOLYSIS

Central venous line catheter to help monitor the intravascular volume

Forced Saline Diuresis:
0.9% NaCl - 1 L over 1 hr rush
Followed by 1 L over 2 hrs
Diuretic - Frusemide infusion @ 1mg/hr
Monitor I/O and titrate volume & diuretics

Maintain urine output 100 - 120 ml /hr

If acidosis + Bicarbonate Infusion (8.4%) @10 ml/hr

± HYPOCALCEMIA

Calcium Gluconate boluses and Infusion if necessary

RENAL FAILURE

If conservative measures do not improve urine output to consider dialysis

Monitor Na^+/K^+HCO^{3-} Q6H x72hrs
Creatinine, Hb, Calcium - OD

HAIR-DYE POISONING

Introduction:

It contains active ingredient – paraphenylene diamine. It is widely used in many countries along with "Henna" for dyeing hair. In India it is marketed as

"super vasmol-33".

It is known to have several toxic effects like Asthma and Dermatitis on topical application. However, on ingestion it is highly toxic which is dose dependant. Large doses are known to cause death due to angioneurotic edema and cardiotoxicity (within 6 - 24 hours). Small doses cause only angioneurotic edema and recovery is quick. Moderate doses cause Acute Renal Failure in the first week.

Presentation:

It presents with cervico-fascial oedema and angioneurotic oedema. Abdominal pain and vomiting occurs in most cases. Conjunctival discoloration, dermatitis, paraparesis, flaccid paralysis, respiratory distress, respiratory failure, oliguria, anuria, jaundice, hepatosplenomegaly, exophtholmos, optic neuritis, blindness, muscle pain, muscle tenderness, rigidity, rhabdomyolysis, intravascular haemolysis, metabolic acidosis, electrolyte disturbance often resulting in MODS.

Laboratory Findings:

- Anisocytosis, Poikilocytosis in peripheral smear.
- Increased serum osmolality, ALT, CPK, LDH, and Blood Urea and Serum Creatinine
- Hyperkalemia, methaemoglobinaemia, haemoglobinaemia, metabolic acidosis.
- Urine – increased osmolality, proteinuria, hematuria, hemoglobinuria, myoglobinuria, and albuminuria.

Treatment:

- Early recognition.
- No specific antidote
- Decontamination: Unlikely of any benefit
- Activated charcoal administration: Unlikely of any benefit

- Alkalinisation of urine – FAD
- Asphyxia – may require ventilatory support, steroids, antihistamines and vasopressors.
- Renal support – Dialysis.
- Antibiotics, antiulcer drugs and blood products.

11. RODENTICIDE POISONING

HEALTH & FAMILY WELFARE DEPARTMENT

Ready Reckoner : Management of patient who has ingested rodenticide

FIRST AID

1. Assess airway, breathing, circulation and provide support, if needed.
2. Try obtain details of poison ingested, if possible.
3. In patients who present within 1hour of poison ingestion, perform gastric lavage.
4. In patients who present within 4 hours of poison ingestion, start oral activated charcoal.
5. All patients need to be admitted at secondary or tertiary level hospital.
6. Avoid sedative medicines.

	Care of patient with no organ dysfunction	Care of patient with liver / other vital organ dysfunction
Admit	To monitor for complications	in a closely monitored area like **HDU** or **ICU** to manage complications
N-Acetyl cysteine	✓	✓
Proton pump inhibitor	✓	✓
Vitamin K (if anti-coagulan containing poison ingested and PT/INR is prolonged)	✓	✓
Sedative Medicines	Avoid	Avoid if possible. Use smaller dose, for restricted indications only, if needed If cerebral edema : Keep patient in semi-reclined posture with head end up by 20 - 30°; IV Mannitol bolus, as needed
Specialised treatments		Plasma exchange: indications 1. Serial worsening INR 2. INR ≥4 3. encephalopathy Counsel about urgent liver transplant
Discharge	If stable, 7 days after poison ingestion	If stable, 7 - 10 days after poison ingestion

For more details :

(Please see TAEI - NHM Guidelines on Management of Rodenticide Poison)

Tamil Nadu Accident and Emergency Care Initiative (TAEI) – National Health Mission (NHM) Guidelines: Management of Rodenticide Poisoning

Preamble

Poisoning is major health problem in India. It can be suicidal, accidental, and occupational. It is deliberate self-poisoning that cause the great majority of deaths and the immense strain that pesticides put on hospital services. Rodenticides are commonest poisoning in Asia countries like India. However comprehensive data regarding the spectrum, outcome of rodenticide poisoning is scant.

Rodenticide is easily available, accessible and cheaper than other pesticide in the market. Almost every system is affected in rodenticide poison and there is no antidote and no definitive treatment guidelines available. Most of them are young adults, Gender are varied, Rural and Urban prevalence are not clear. Lethal dose and late presentations, Major public health problem, Early symptomatic care is the key in reducing the mortality. This TAEI NHM program aims to reduce deaths from poisoning by rodenticide ingestion across Tamil Nadu. In a study conducted across 6 districts of Tamil Nadu by the Tamil Nadu chapter of Indian Society of Gastroenterology (TN-ISG) between January to June 2019, of 451 patients with rodenticidal hepatotoxicity,35% of patients diedor were discharged in a moribund state and only one patient underwent urgent liver transplantation.1In response to this study findings, the TN-ISG brought out guidelines on management of rodenticide poisoning focusing on non – transplant treatments to improve survival.2

These TAEI NHM guidelines have been adapted from the TN-ISG guidelines on management of rodenticide poisoning.This program will establish a "hub and spoke" model in each district for delivery of care for these patients. Primary Health Centres need to provide first aid and then refer all patients with rodenticide ingestion to nearest secondary / tertiary hospital. At secondary level hospital, all patients with rodenticide ingestion need to be admitted, mainly for providing supportive care and monitoring for complications. Patients who develop liver / other organ damage need to be transferred to tertiary hospital or select secondary level hospitals with facilities for specialised treatment (dedicated team of doctors trained to manage these patients as per a specific protocol, with a designated area to admit these patients for monitoring andproviding specialisedtreatmentincluding plasma exchange, if needed). Institution of appropriate treatment early in the illness is likely to reduce death rates.

*Please see Page 7 for information regarding dedicated facilities

Management of rodenticide ingestion patient : "Hub and spoke model" within a district

Location	Treatment
Primary Health Centre	First aid
Secondary level hospital (Taluk hospital)	Manage patient with no organ dysfunction (Supportive care)
Tertiary level hospital (Government Medical College), select secondary level hospital with dedicated facilities*	Manage patient with dysfunction of liver / other vital organ (Specialised treatment including plasma exchange)

A) Management of rodenticide ingestion patient : at Primary Health Centre

1. ABCD of rodenticide poisoning primary management.

   Assess (ABCD) - airway, breathing, circulation and decontamination (skin and gastric). These need to be assessed and appropriate treatment given, if these are abnormal.

2. Identifying the poison(s) consumed by the patient

   Details of poison ingested are ascertained by interviewing the patient or family member; by obtaining the poison package / cover / pamphlet and also, by asking to identify the poison from a pictorial display of various rodenticides available in the locality. (Picture enclosed). It is important to check if the product has anti-coagulant as one of its components.

3. Gastric lavage to treat patient who presents early after rodenticide ingestion.

- It can be done using Ryle's tube as an alternative to large orogastric tube.
- It is not indicated in condition where repeated vomiting after ingestions rodenticide.
- It should be avoided after 3 days of ingestion.

Gastric lavage indication	Benefit
Within 1 hour	More
After 1 hour	Less
After 6 hour	Nil

4. Oral activated charcoal to help remove the ingested poison

- Benefit of Oral activated charcoal is not clear.
- Patients presenting within FOUR hours of ingestion of rodenticide should be given activated charcoal (25–100 g, mixed with 150 to 200 ml water), orally.
- Activated charcoal is given as 1g/kg within 1 hour.
- Multiple dose every four to six hours for 24 hours may be given.
- Oral activated charcoal should be avoided after 3 days of ingestion.

5. Avoid Sedative Medicines

As almost all sedative medicines are metabolized in the liver, use of these may result in (unintentional) overdose in patients with liver dysfunction. Sedative overdose can make the patient drowsy, increase the risk of respiratory depression and culminate in death. IT IS IMPORTANT TO AVOID SEDATIVE MEDICINES IN THESE PATIENTS.

- **Investigations may be done at primary level centers.**
  - Complete Blood Count,
  - Blood Sugar.
  - Liver function test
  - Renal function test
  - Electrolytes
  - ECG if available

6. Milk or fatty foods may promote phosphorus absorption therefore high carbohydrate, high protein, low/no fat diet, with supplementary intravenous glucose, vitamins is preferred

7. Referral Criteria from Primary Care Center to nearest Secondary Level Hospitals:

- Derangement of Liver Function Test (LFT) (Ex: Liver damage often occurs 3 to 5 days after poison ingestion)
- For monitoring for complications
- All patients need to be admitted in the secondary level hospitals

B) Management of rodenticide ingestion patient with no organ dysfunction : at secondary level hospital

Please follow Guidelines A.1) to A.5) as mentioned above.

7. Admitting the patient who has ingested rodenticide for monitoring

Patient should be admitted in hospital for SEVEN days to monitor for any complications. Daily assessment of clinical parameters (including sensorium) and laboratory tests (hemogram, liver function tests [LFT], prothrombin time/international normalized ratio (PT/INR), sodium, potassium, creatinine, ABG, Blood sugar, serum calcium, X-ray abdomen) is needed.

Patients who develop any organ dysfunction or failure (e.g. deranged LFT, prolonged PT / INR, acute pancreatitis, raised serum creatinine) need immediate transfer to a tertiary level hospital or (secondary level hospital with these facilities) for specialised treatment like plasma exchange.

8. N-acetyl cysteine to prevent rodenticidal hepatotoxicity

The patient needs to be given IV N-Acetyl Cysteine (150 mg/kg body weight in 250 ml 5% dextrose over 1 hours, followed by 50 mg/kg in 500 ml 5% dextrose over 4 hours, then 100 mg/kg dose in 1000 ml 5% dextrose over 16 hours). Assess fluid status while giving N-Acetyl Cysteine as volume over load is common in giving dilution, if volume over load, dilution of N-Acetyl Cysteine is to be changed.

9. Ameliorating gastric inflammation in patient who has consumed rodenticide

The patient needs to be given as Intravenous proton pump inhibitor for 1 week.

10. Management of patient who has ingested anti-coagulant containing rodenticide.

If anti-coagulant containing poison is ingested and if PT/ INR is prolonged, the patient needs to be given vitamin K.

11. Discharge Criteria

- Psychiatric evaluation, counseling and support by trained personal should be offered to patient with suicidal poisoning and their families before discharge.
- The decision to discharge is to be collectively decided by the clinical team taking care of the patient.
- Patients who are stable SEVEN DAYS after poison ingestion can be discharged.
- He/she should report immediately to the nearest hospital in their locality, if new symptoms appear after discharge.

Type of Anti coagulation	Dose	Route	Duration
First Generation	3mg, in infants, 5mg in 1-5 years, 10mg if more than 5 years	Subcutaneous	2 weeks
Second Generation	3mg, in infants, 5mg in 1-5 years, 10mg if more than 5 years	Subcutaneous	4 weeks

12. Follow Up Home Visit

Trained social workers need to make follow up home visit within the next ONE WEEK to assess needs and provide appropriate support to patient and family and counseling, especially about the stressor event which triggered the poison ingestion.

C) Management of rodenticide ingestion patient with damage to liver or other vital organs :

at tertiary level hospital

13. Admit in closely monitored area e.g. high dependency unit (HDU) / intensive care unit (ICU)

- Patient with liver damage (i.e. abnormal LFT / prolonged INR) or other organ dysfunction / failure (like acute pancreatitis, acute kidney injury) need to be

managed in closely monitored area like HDU / ICU with frequent clinical and daily laboratory (hemogram, LFTs, PT/ INR, sodium, potassium, creatinine) assessment.

14. Intravenous N Acetyl Cysteine

- The patient needs to be given IV N Acetyl Cysteine (150 mg/kg body weight in 250 ml 5% dextrose over 1 hours, followed by 50 mg/kg in 500 ml 5% dextrose over 4 hours, then 100 mg/kg dose in 1000 ml 5% dextrose over 16 hours).

15. Ameliorating gastric inflammation:

- The patient needs to be given as intravenous proton pump inhibitor for 1 week.

16. Management of patient who has ingested anti-coagulant containing rodenticide:

- If the ingested poison contains anti-coagulant and if PT/INR is prolonged, the patient needs to be given vitamin K.

17. Treatment of cerebral edema in patients with rodenticide induced acute liver failure

Patient with encephalopathy needs anti-cerebral edema measures (nurse in semi-reclined posture with head end up by 20°–30°. Intravenous mannitol (0.5–1 g/kg body weight) bolus, as required.

For cerebral edema, in children 3% NaCl is used commonly all over world. The dose is 5ml/kg bolus followed by 0.1 to 1 ml/kg/hr infusion 3% NaCl is stopped if serum sodium more than 160 or serum osmolality more than 360.

18. Caution on use of sedative drugs in patients with rodenticidal hepatotoxicity

It is preferable to avoid sedation in these patients in the absence of encephalopathy. If sedative drug is needed (e.g. for endotracheal intubation), then reduced dose (ex. 1/4th the regular dose) of sedative drug maybe used. 2nd dose of sedative is given, only if needed, after clinical assessment of its response. Short acting like midazolam may be given if indicated. Avoid Midazolam in intubated children. If sedation required, Fentanyl at 1 to 2 mcg/kg/hr.

Type of Anti coagulation	Dose	Route	Duration
First Generation	3mg, in infants, 5mg in 1-5 years, 10mg if more than 5 years	Subcutaneous	2 weeks
Second Generation	3mg, in infants, 5mg in 1-5 years, 10mg if more than 5 years	Subcutaneous	4 weeks

19. Urgent liver transplantation to treat rodenticidal hepatotoxicity

In patients with severe liver damage (encephalopathy or worsening INR), the patient and family members need to be counselled by the admitting unit regarding urgent liver transplantation. The option of specialised non-transplant treatments like plasma exchange (PLEX) also needs to be discussed.

20. Plasma exchange (PLEX) to treat patients with rodenticidal hepatotoxicity, PLEX to be decided on case basis in children

20.1 Indications for PLEX in these patients are presence of deranged LFT AND any of following criteria:

i) INR $\geq$ 4

ii) serial worsening of INR

iii) depressed consciousness/altered behavior.

20.2 Contra-indications for PLEX are presence of either of thefollowing criteria:

i) hemodynamic instability

ii) active sepsis.

20.3 Details of PLEX procedure

20.3.1. Obtain signed consent from patient / next of kin after counseling about all treatment options.

20.3.2. It is preferable to insert venous access line for PLEX and to perform PLEX at the bedside in monitored setting (HDU / ICU).It is preferable not to transfer these critically ill patients for various tests or for treatment to other places (e.g. dialysis ward etc) within the same hospital.

20.3.3. Femoral vein is the preferred access for PLEX port insertion under ultrasound guidance. This access is exclusively for PLEX and should not be used for any other purpose (e.g. taking blood samples, administering medicinesetc). Avoid prophylactic platelet or fresh frozen plasma transfusion for line insertion, even if platelet is low or INR is prolonged.

20.3.4. Start prophylactic intravenous antibiotic(chosen as per local hospital antibiotic policy)after sending the blood culture.

20.3.5. Start Tablet Prednisolone 10 mg or equivalent as soon as decision to PLEX is made, continue for further 1 to 4 weeks after stopping PLEX, based on clinical assessment.Prednisolone reduces the cytokine storm.

20.3.6. Low volume plasma exchange (50% of plasma volume exchanged per PLEX session) is recommended.

20.3.7. The preferred replacement fluid is fresh frozen plasma at 1:1 volume.

20.3.8. Centrifugaltype PLEX ispreferred to membranetype PLEX.

20.3.9 PLEX is done daily for 3days. Decision todo PLEX is reviewed each day; total number of PLEX sessions is decided based on tolerability/patient's clinical condition. During PLEX, maintain strict asepsis/avoid hemodynamic instability/give calcium supplementation (Dose/Route). Remove venous access as soon as PLEX is over.

20.3.10. It is preferable to do PLEX during daytime working hours, when experienced personnel are likely to be available. However, forencephalopathic patients (i.e. acute liver failure), PLEX can be undertaken at anytime.

20.3.11. If feasible, blood culture should be done on days of PLEX. In case of bacteremia/clinical signs of sepsis while on PLEX, it is necessary to withhold/discontinue PLEX until sepsis is controlled and escalate antibiotics, awaiting culture / sensitivity report.

20.3.12. Organ-specific standard of care for critically ill patient should be continued.

21. Monitoring for complications other than hepatotoxicity: Patients are monitored for cardiac toxicity/ other complications like pancreatitis and treated appropriately.

22. Discharge criteria

- Patients who are stable SEVEN TO TEN DAYS AFTER INGESTION OF POISON, with improving clinical and laboratory parameters, can be discharged from hospital after assessment by the treating clinical team. In case of any new symptoms after discharge from hospital, patient is advised to report to the nearest hospital in their locality.

23. Follow-up Care

23.1) Psychiatric evaluation, counseling and support by trained personal should be offered to patient with suicidal poisoning and their families before discharge.

23.2) Hospital outpatient visit, ONE WEEK later, for clinical assessment and laboratory tests (LFT, INR, creatinine):

- In addition, patients and their families need support from Psychiatrists / trained social workers to help
- address the underlying trigger which precipitated the suicide attempt.

23.3) Home visits: by trained social workers, ONE WEEK LATER AND ONE MONTH LATER, to provide support and counseling, especially to address the stressor which led to poison ingestion.

Key Points to be Noted:

✦ Transfer of patient to another hospital

Patient transfer from Primary Health Centre HC to secondary level hospital OR from secondary level to tertiary level hospital is best done in liaison with the medical team at the receiving centre

✦ Dedicated facilities for specialised treatment in tertiary level / select secondary level hospitals

Long distance transfer may worsen the condition of critically ill patients. Hence, it is advisable to provide specialised treatments within each district. These hospitals should have the following provisions:

A) Dedicated team (medical and nursing faculty, emergency technician, social worker) to manage these patients as per this management protocol. The medical team should comprise of consultants from Emergency Medicine, Clinical Toxicology / Medicine / Pediatrics / Hepatology / Gastroenterology/ Nephrology/ Transfusion Medicine and Intensive Care. It is important for each hospital to identify the lead / admitting unit who will co-ordinate care for these patients.

B) Dedicated area for monitoring (with adequate staffing, restricted access to patient attendants)

C) Facility for plasma exchange (dialysis or apheresis machines can be used for PLEX, uninterrupted power supply needed for the PLEX machine; adequate blood bank facilities [4 – 6 units of fresh frozen plasma maybe needed per PLEX session for a patient]).

✧ Hotline for guiding protocol based management needs to be provided

A) Helping doctors at Primary Health Centres and secondary level hospitals to manage patients

B) Helping doctors at hospitals providing specialisedtreatment(queries regarding urgent liver transplantation, trouble-shooting PLEX)

Recommendations

1. Prevention of toxicity by restricting access to this toxin to public by banning its sale, storage or promotion of alternative least toxic rodenticide.
2. Safer packaging which is difficult open for kids and prominent labeling of toxicity.
3. Awareness of its severe toxicity and high lethality in vary small quantities among public, people handling the toxin and clinicians
4. Rodenticide toxicity is more common in the Indian continent, multi center study could be able to better define the optimal management – Plasmapheresis and develop criteria for liver transplant
5. Legislation restricting availability, labeling, packing, storage of the rodenticide may help reduce the poisoning.
6. Creation of rodenticide poison registry.

7. Training of health care personnel.
8. Toll free number for public to contact for informations

Ideal rodenticide should have following features:

- It must be effective in small enough amounts that adding it to food or water supply will not cause bait refusal.
- The manner in which it kills the rodent cannot arouse suspicious in the surviving animals
- The substance should be specific to rodents in toxicity and less toxic to humans and domestic animals.
- The anticoagulant rodenticides best meet these criteria

References:

1. *Rodenticide ingestion is an important cause of acute hepatotoxicity in Tamil Nadu, southern India. Indian J Gastroenterol. 2021 Aug; 40 (4) :373-9.*
2. *Management of rodenticide poisoning: Tamil Nadu chapter of Indian Society of Gastroenterology guidelines. Gastroenterol Hepatol Endosc Pract 2022;2:1-6*

மருத்துவம் மற்றும் மக்கள் நல்வாழ்வுத் துறை

எலிக் கொல்லி விற்பனை தடை செய்யப்பட்டுள்ளது

எலிப் பசை தடை

தேசிய நலவாழ்வு குழுமம்

தமிழ்நாடு

12. TABLET OVERDOSE

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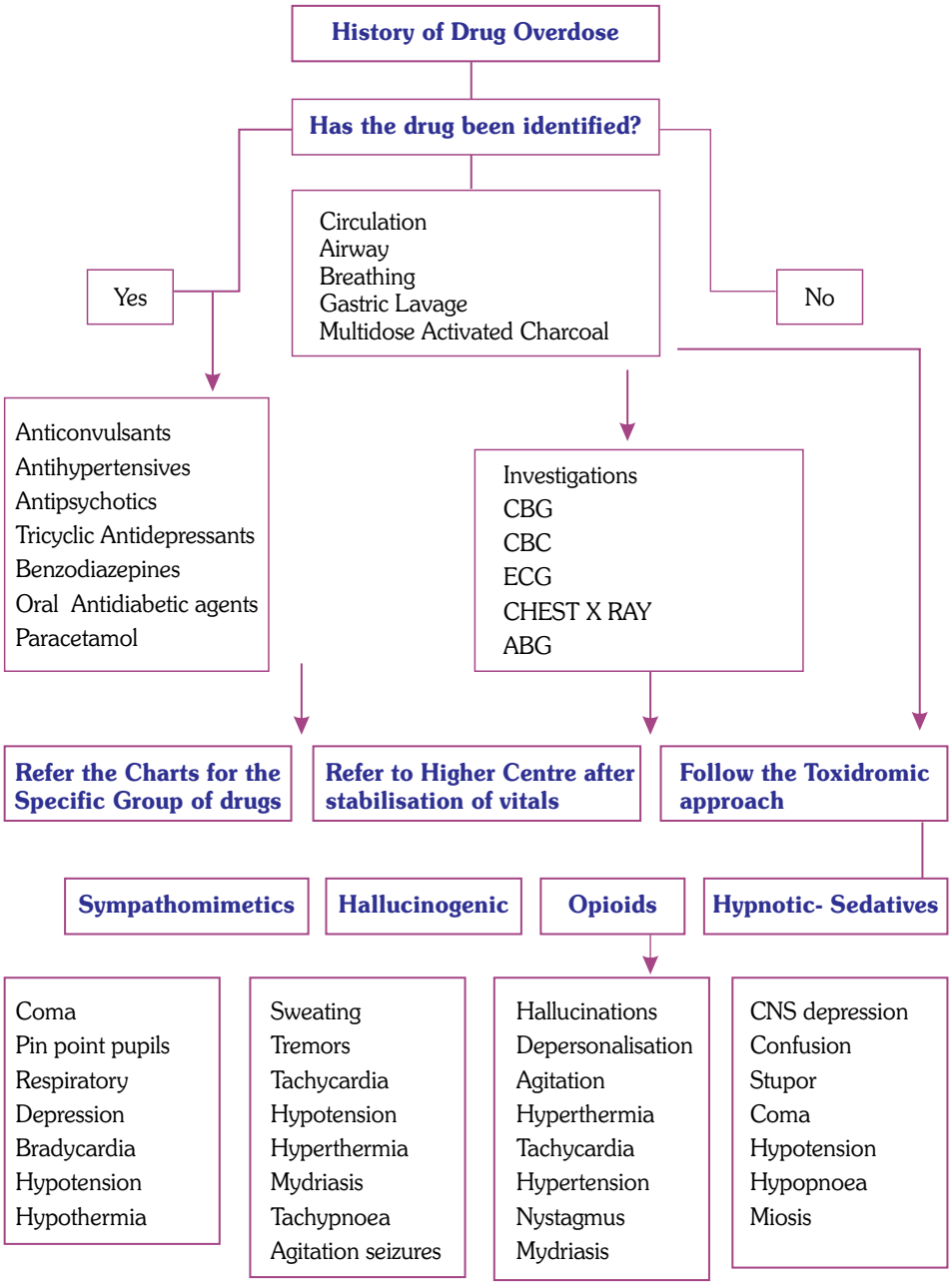


Treatment Pearls

1. Benzodiazepines - MDAC, Flumazenil 0.1 mg - 0.2 mg iv bolus repeated up to 2 mg, Forced alkaline diuresis for long acting preparations and Hemoperfusion for refractory cases

2. Antipsychotics - Lidocaine for ventricular arrhythmias, Magnesium sulphate for Torsade pointes, Diphenhydramine hydrochloride 25- 50mg, Benztropine 1-4 mg, for dystonia.

   NMS- Dantrolene 1- 10 mg/kg/day, and Bromocriptine 2.5 - 10 mg IV tid.

   Filgrastine if Agranulocytosis.

3. Oral Hypoglycaemic Agents - IV dextrose, Hydrocortisone100mg with IV Dextrose if recovery does not occur in 10 mins .Inj. Glucagon1mg if IM or SC if IV access not obtained.

   In Sulphonyl urea overdose, recurrent hypoglycaemia occurs due to insulin release (triggered by transient hyperglycaemia). Inj. Octreotide (4-5mcg/kg/day) is used to treat.

4. Carbamazepine - Activated charcoal, charcoal hemoperfusion, IV Lipid Emulsion if indicated.

5. Barbiturates - MDAC, FAD for long acting preparations, Haemodialysis / Hemoperfusion

6. Phenytoin toxicity – FAD

7. Sodium Valproate - Activated charcoal, Hyperammonaemic encephalopathy treated with L – carnitine, CNS depression treated with Naloxone , Haemodialysis / Hemoperfusion

8. TCA - Gastric lavage up to 12 hours due to delayed gastric emptying, IV Sodium Bicarbonate 1 - 2mg/kg bolus for wide complex tachycardia. If tachycardia is persistent - IV lidocaine / Defibrillation, Magnesium Sulphate 25 - 50 mg/kg IV over 2 minutes bolus followed by continuous infusion 5 - 10 mg/ min. for Torsade pointes. Haemodialysis, Hemoperfusion, ECMO. FAB fragment specific for TCA.

9. Paracetamol overdose - N acetyl cysteine, for worsening liver function with MODS refer for Liver Transplantation.

12.1 TRICYCLIC ANTIDEPRESSANTS OVERDOSE

Toxic Mechanisms:

Central and peripheral anticholinergic effects Peripheral alpha adrenergic blockade

Quinidine like cardiac membrane stabilizing action blockade Inhibition of synaptic neurotransmitters reuptake in CNS presynaptic neurons.

Pathophysiology:

- Hypotension
- Arrythmias - The most common is sinus tachycardia
- Agitation, delirium and depressed sensorium
- Acute lung injury

Clinical Features:

1) Anticholinergic effects:

Sedation, delirium, coma, dilated pupils, dry skin and mucous membranes

Diminished sweating, tachycardia

Urinary retention, Myoclonic jerks

2) Cardiovascular Toxicity:

Conduction Delays

- PR interval, QTc interval, QRS complex prolongation
- Rightward axis rotation
- Atrioventricular block

Dysrhythmias:

- Sinus tachycardia
- Supraventricular tachycardia
- Wide complex tachycardia
- Torsades de pointes
- Ventricular fibrillation
- Asystole

3) Cns Toxicity: Altered mental status, Delirium, Lethargy, Myoclonus

4) Pulmonary Toxicity: Acute lung injury

Diagnosis: ECG

QRS duration > 100 msec
Axis between 120 and 270
An abnormal rightward axis
Amplitude of terminal R wave and R/S wave ratio in lead aVR

Lab Tests:

TCA concentrations > 1000 ng/ML : significant toxicity occurs
DDetected by urine toxicolgy screening and thin layer chromatography / HPLC
CPK, urine analysis for myoglobin, ABG, RFT, CXR

Treatment

Continuous monitoring of the temperature, other vital signs and ECG in asymptomatic patients for minimum of 6 hours, and admit patients to ICU for atleast 24 hours if there are any signs of toxicity

Emergency and Supportive Measures:

Decontamination:

Administer activated charcoal Gastric lavage - upto 12 hours after ingestion yields unabsorbed drug Anticholinergic actions of some TCAs decrease spontaneous gastric emptying.

Seizure : Benzodiazipines(Diazepam or Lorazepam)

Dysrhythmias

Supraventricular tachycardia with hemodynamically unstable status require synchronized cardioversion.

Wide complex tachycardia- Sodium bicarbonate 1-2mEq/kg IV boluses, if persistent requires IV lidocaine and defibrillation.

Magnesium sulfate 25-50 mg/kg IV over 2 min for TDP followed by continuous infusion 5-10 mg/min.

Bradyarrhythmias - Atropine is contraindicated due to anticholinergic activity Isoprotenelol 0.1mcg/kg/min used with caution.

Torsades de pointes
Magnesium Sulphate 2gms IV
IV 3%Saline 1-3ml/kg over 10 minutes
Overdrive pacing
Do Not Give Class I antiarrhythmics, Beta Blockers, Calcium Channel Blockers, Class III anti arrhythmic

Hypotension

- Isotonic saline boluses upto 30mg/kg
- Correct hypoxia, acidosis
- Sodium bicarbonate 1-2mEq/kg IV boluses/ infusion
- Norepinephrine (0.05 -0.1 mcg/min infusion is preferred over dopamine due to its predominant alpha adrenergic effect

Other Modalities:

HD, PD, Hemoperfusion not effective due to high protein binding and large volume of distribution ECMO

Newer Therapy:

The bradycardiac agent UL-FS 49 effectively impedes marked sinus tachycardia and frequency dependent ventricular conduction delay FAB fragments specific for TCA - investigational treatment

Admission and Recovery:

The following patients should be admitted in ICU for 12-24 hours

- ECG abnormalities
- Altered mental status
- Seizures
- Respiratory depression
- Hypotension

12.2 ANTIPSYCHOTIC DRUGS OVERDOSE

Antipsychotics exerts their effects largely by binding to Dopamine receptors in CNS, Also affects cardiovascular & endocrine systems.

Antipsychotics with their usual dose	Toxic Effects
Risperidone 4 - 16 mg	E,H,Q
Chlorpromazine 200 - 2000 mg	A,E,H,Q
Prochlorperazine 5- 40 mg	E
Promethazine 25- 200 mg	A,E
Aripiprazole 10- 30 mg	A,E,H,Q
Cloazapine 100 – 900 mg	A,H
Haloperidol 1-100 mg	E,Q
Quetiapine 150 – 750 mg	A,E,H,Q
Olanzepine 5 – 20 mg	A,E,H
Trifluperazine 1 – 40 mg	E
Ziprasidone 60- 160 mg	A,E,H,Q

Note: A - Anticholinergic
E - Extrapyramidal Effects
H - Hypotension
Q - QT prolongation

Clinical Features:

Mild – sedation, agitation, dry mouth, tachycardia, fasciculations, clozapine causes hypersalivation

Severe – Coma, Seizure, and Respiratory arrest, QT prolongation, ventricular arrhythmias and torsade's de pointes.

Extrapyramidal symptoms – dystonia, torticollis, Trismus, oculogyric crisis, rigidity, bradykinesia, tremor

Clozapine has agranulocytosis

Neuroleptic Malignant syndrome (NMS) – hyperthermia, altered mental status, muscular rigidity, autonomic dysfunction, with elevated creatinine kinase with myoglobinuria, hyperkalaemia, acidosis, renal failure

Diagnosis:

History of ingestion
Blood levels – not helpful
Lab investigations – CBC, Electrolytes, BUN, Creatinine, CPK, ABG, ECG

Treatment:

No specific antidote

Emergency And Supportive Measures-

- Gastric Lavage, Activated Charcoal
- Maintain Airway and Breathing
- Monitor vital signs and cardiac monitoring
- Severe hypotension corrected by Bolus of NS 20 ml/kg and vasopressors if no response

Ventricular arrhythmias: Lidocaine

Torsades de pointes: I.V magnesium 2Gms

Seizures: Benzodiazepines

Dystonia: Diphenyhydramine 25-50 mg, Benztropine 1-4 mg

Hyperthermia: cooling

NMS:

Dantrolene 1-10 mg/kg/day for 48 to 96 hrs
Bromocriptine 2.5-10 mg i.v tid until patient improves
Benzodiazepines Consider **Filgrastim** for agranulocytosis

Psychiatric

Clearance needed before discharge

References :

1. *Washington manual of Medical therapeutics McGraw Hill Access Medicine Poisioning and drug overdose.*

12.3 ANTICONVULSANTS OVERDOSE

Barbiturates are used as sedatives, anaesthetic agents and anticonvulsants They are GABA agonists.

Toxic Dose:

Short acting barbiturates- 6mg/kg Long acting barbiturates - 10mg/kg

Clinical Features:

Lethargy, slurred speech, nystagmus and ataxia Hypoglycemia, hypothermia Hypotension, coma and respiratory arrest.

Diagnosis:

Clinical pointers are any epileptic patient with stupor or coma, skin bullae Routine urine toxicologic screening.

Treatment:

No specific antidote

Emergency And Supportive Measures:

- Vitals monitoring
- Check for blood glucose if comatose
- Fluid for dehydration and hypotension
- Severe hypotension corrected by vasopressor

Decontamination:

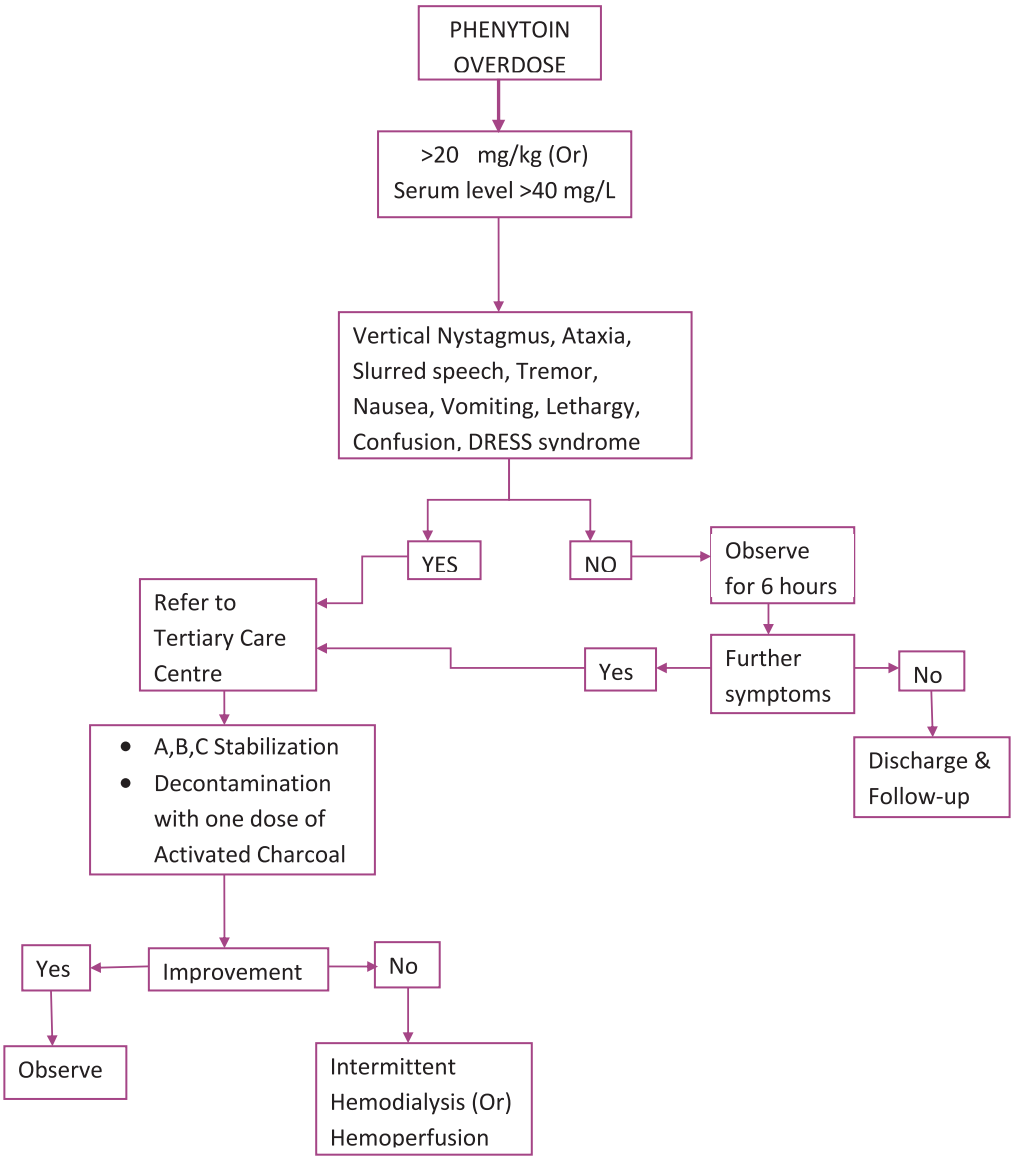
Gastric lavage for massive ingestion Protect airway in comatose patients before gastric lavage Administer activated charcoal 1gm/kg then followed by MDAC for every 2-4 hours.

Further Clearance:

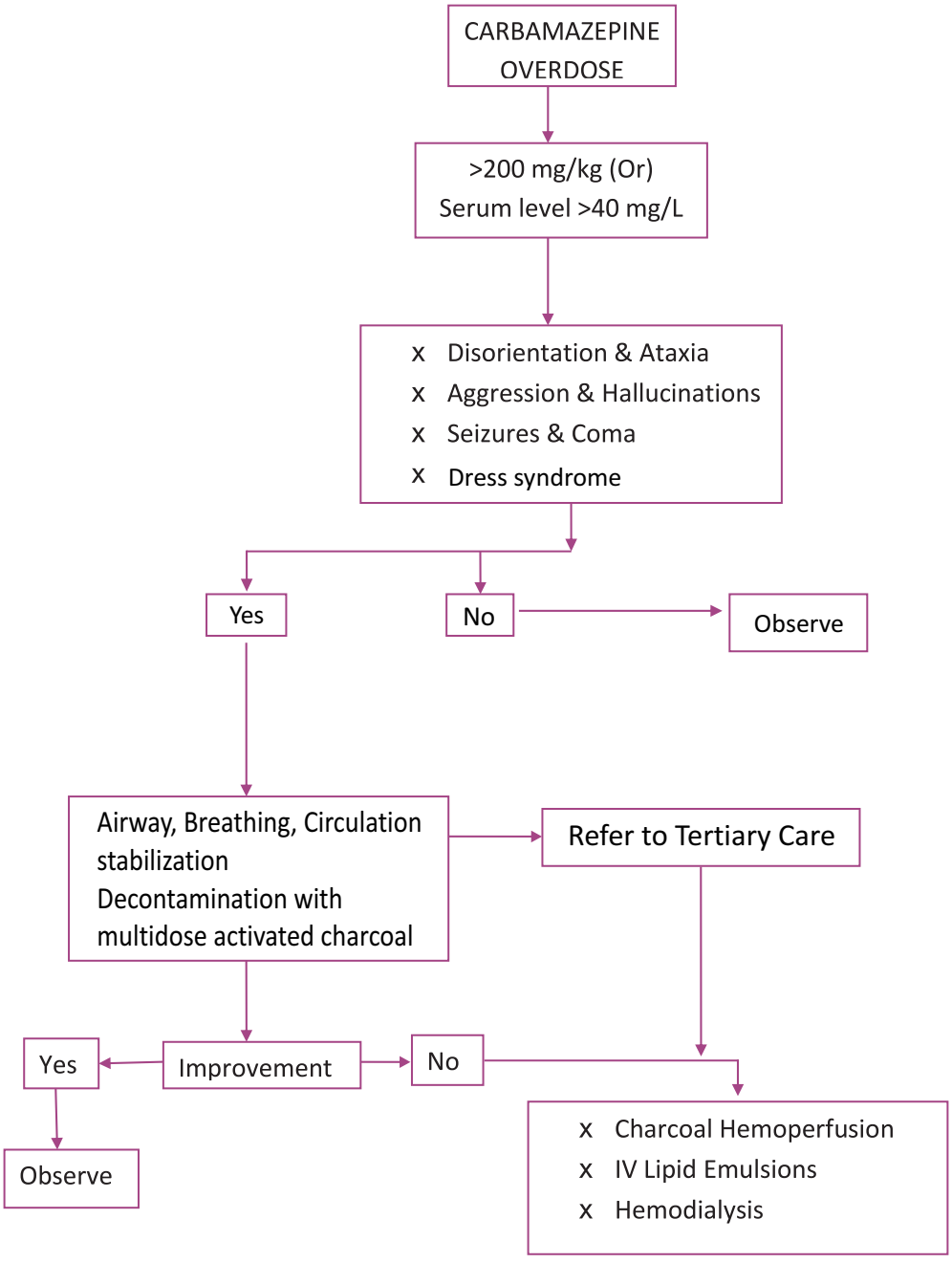
Alkalization of urine for long acting barbiturates Hemodialysis and hemoperfusion for patients not responding to supportive care and plasma concentration > 150mcg/ml..

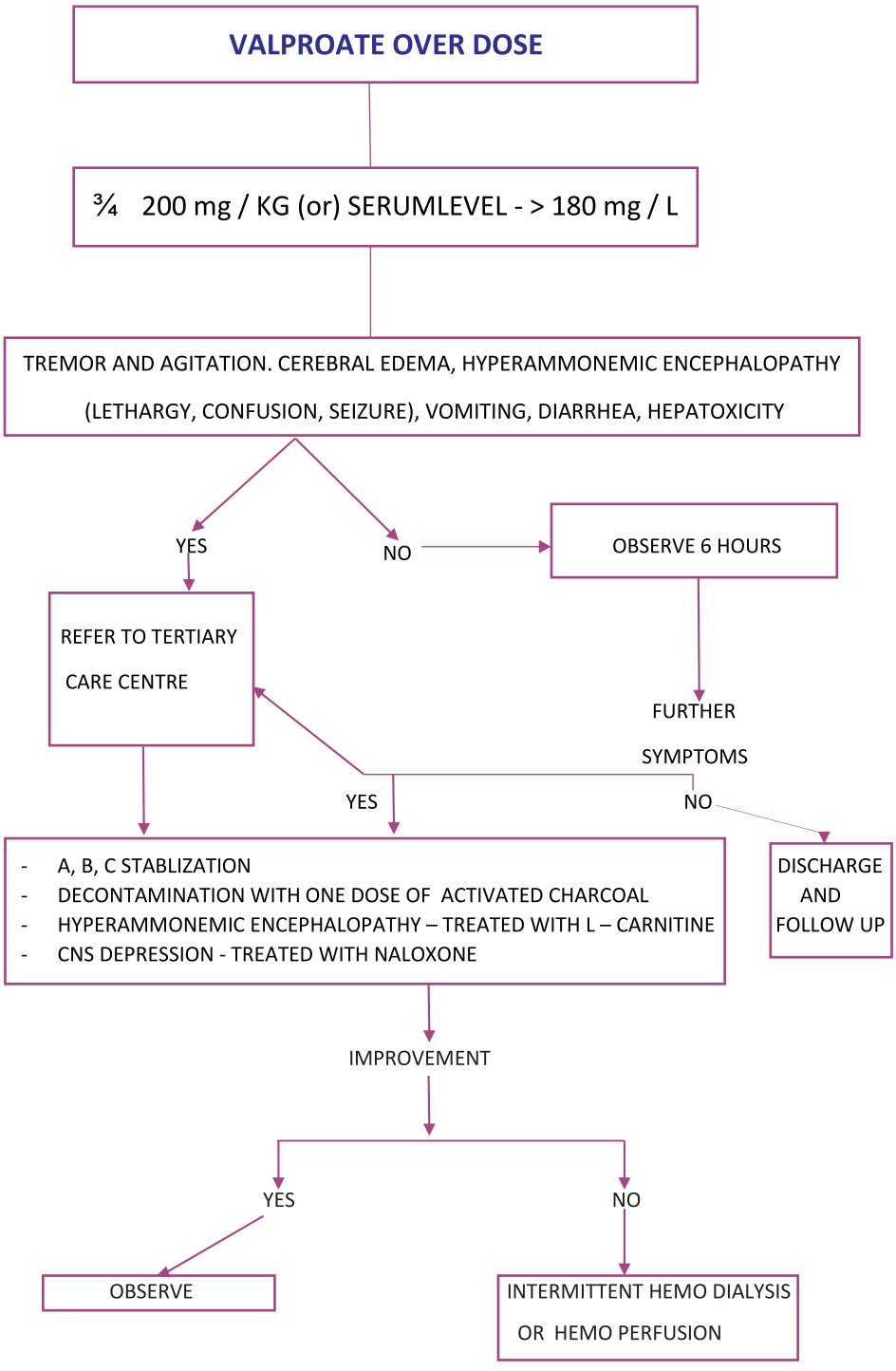
Psychiatric Clearance needed before discharge

PHENYTOIN OVERDOSE



CARBAMAZEPINE OVERDOSE




VALPROATE OVER DOSE
¾ 200 mg / KG (or) SERUMLEVEL - > 180 mg / L
TREMOR AND AGITATION. CEREBRAL EDEMA, HYPERAMMONEMIC ENCEPHALOPATHY
(LETHARGY, CONFUSION, SEIZURE), VOMITING, DIARRHEA, HEPATOXICITY
YES
NO
OBSERVE 6 HOURS
REFER TO TERTIARY
CARE CENTRE
FURTHER
SYMPTOMS
YES
NO
- A, B, C STABLIZATION
- DECONTAMINATION WITH ONE DOSE OF ACTIVATED CHARCOAL
- HYPERAMMONEMIC ENCEPHALOPATHY – TREATED WITH L – CARNITINE
- CNS DEPRESSION - TREATED WITH NALOXONE
DISCHARGE
AND
FOLLOW UP
IMPROVEMENT
YES
NO
OBSERVE
INTERMITTENT HEMO DIALYSIS
OR HEMO PERFUSION

12.4 BENZODIAZEPINES OVERDOSE

They enhance the action of inhibitory neurotransmitter GABA causing generalised depression of spinal reflexes and reticular activating system

Clinical Presentation:

Lethargy,slurred speech, ataxia, coma and respiratory arrest Hypothermia, hypotension and bradycardia may occur Mid or small pupils.

Diagnosis:

History suggestive of ingestion Urine toxicological screening

Treatment:

Flumazenil is the antidote of choice

Supportive and symptomatic measures

Maintain airway

Hypotension - IV fluids and supine posture Mechanical ventilation in case of respiratory depression.

Flumazenil:

It is a competitive benzodiazepine receptor antagonist Dosage- 0.1mg - 0.2 mg as bolus, repeated upto maximum of 3 mg It reverses CNS depression Adverse effects-induce acute withdrawal, including seizures and autonomic instability. Resedation is common.

Management Protocol:

Decreased mentation, slurred speech, loss of reflexes, hypotensiom and slow respiration.

Maintain airway, breathing and circulation Gastric lavage and activated charcoal 1 gm/kg.

For long acting ones, give forced alkaline diuresis and multidose activated charcoal Hypotension- Crystalloids, dopamine and noradrenaline.

Antidote: Flumazenil 0.2 mg over 30 seconds followed by 0.3mg at 1 min interval upto total dose 3mg, if there is response give additional dose of 0.5 mg increments to total dose 5 mg.

Hemoperfusion:

Can be useful

12.5 AIRWAY, BREATHING, CIRCULATION STABILIZATION

Clinical Features:

Cardiovascular – Hypotension and Bradycardia, shock, Bradyarrhthymias (Heart block of any degree), QRS/QT prolongation.

Respiratory – Bronchospasm, Respiratory depression

CNS - Confusion, seizures and coma (in lipophilic Beta blockers like Propranolol)

Metabolic – Hypoglycemia(BB), hyperglycemia(CCB), hyperkalemia

Stabilise Airway, Breathing and Circulation (ABC)

Endotracheal intubation to be considered in case of Respiratory depression, CNS depression or severe hypotension.

Attach Cardiac monitors, obtain IV access, check CBG, draw blood samples for Serum electrolytes, calcium, magnesium and take a standard 12 lead ECG.

Decontamination:

- *Multi Dose Activated Charcoal (MDAC)*: Ideal if given within 1-2 hours of ingestion but can be given upto 6 hours after ingestion.

- *Whole Bowel Irrigation with Poly Ethylene Glycol (PEG)*: It is useful in delayed presentation, in cases of large ingestion of sustained-release formulations and in long acting drugs like Amlodipine. It should be administered orally or via a nasogastric tube at a rate of 1-2L/hr and should be continued until the rectal effluent is clear. If the patient received a dose of charcoal, passage of charcoal per rectum may also be a sign of adequate evacuation.

IV fluids - Administer IV bolus crystalloid(Normal Saline) 500-1000 ml for hypotension

Atropine - In case of bradycardia. The dose is 0.5 to 1mg iv every 3 to 5 mins upto a maximum cumulative dose of 3mg.

Calcium - In patients with refractory shock, 30 ml of 10% calcium gluconate (0.6 ml/kg) over 5 to 10 minutes can be given followed by 0.6-1.5 ml/kg/hour.

Glucagon *(preferred for BB toxicity; can be tried for CCB overdose also)*- exerts positive inotropic and chronotropic effects by increasing intracellular cAMP levels. It is given as a bolus dose of 1-5 mg IV over 5 minutes upto 15 mg total. Bolus therapy can be repeated if ineffective in 3 to 5 minutes. If the bolus dose causes hemodynamic improvement, this can be followed by a continuous infusion at a rate of 3-10 mg/hr.

Vasopressors - In patients with shock not responsive to fluid resuscitation, Noradrenaline 2 microgram/minute IV; titrate rapidly to a SBP of 100mmHg

Hyperinsulinaemia-Euglycaemia (HIE) therapy – HIE therapy should be started early in patients with cardiogenic shock since it takes at least 15-60 minutes to work. It is given as 1 unit/kg IV bolus along with 25% dextrose(1oo ml), followed by 1 unit/kg/hour infusion along with 10% dextrose solution. If the response is unsatisfactory, insulin may be up-titrated every 10 to 15 minutes upto 10 units/kg/hour. CBG and Serum electrolyte levels to be regularly monitored.

Intravenous Lipid Emulsion(ILE) therapy - Intralipid 20% at a bolus dose of 1.5 ml/kg over 2 to 3 minutes followed by 15 ml/kg over 60 mins.

Additional Therapies:

Sodium Bicarbonate - for beta blocker induced arrhythmias. It can be given to patients with QRS prolongation and severe acidosis at a dose of 1-3 meq/kg of 8.4% solution.

Magnesium - for Qtc prolongation and Ventricular arrhythmias.

Cardiac pacing : Transvenous pacing can be implemented if there is no response to pharmacologic therapies and the patient remains bradycardic and hypotensive.

Intra Aortic Balloon Pump (IABP) - in severe hemodynamic instability.

Hemodialysis - It has a minimal role in Beta blocker overdose and is effective only in hydrophilic, minimally protein-bound beta blockers such as Atenolol, nadolol, sotalol and acebutolol.

ECMO – It is reserved for severe ingestions not responding to standard therapy.

12.6 ORAL ANTIDIABETIC DRUG OVERDOSE

Oral hypoglycemic agent poisoning

Hypoglycemia (RBS<70mg/dl)

I.V access possible

100ml of25% dextrose I.V
Over minutes.

Recovery anticipated in 10 minutes.

If not

Repeat 100ml of 25% dextrose
+
Inj.hydrocortisone 100mg

recovered
Maintain 5% dextrose infusion
to maintain sugar level (75-100ml/hr)

no I.V access possible

inj. Glucagon 1mg I.m or s.c

recovery delayed >5hrs

consider cerebral edema

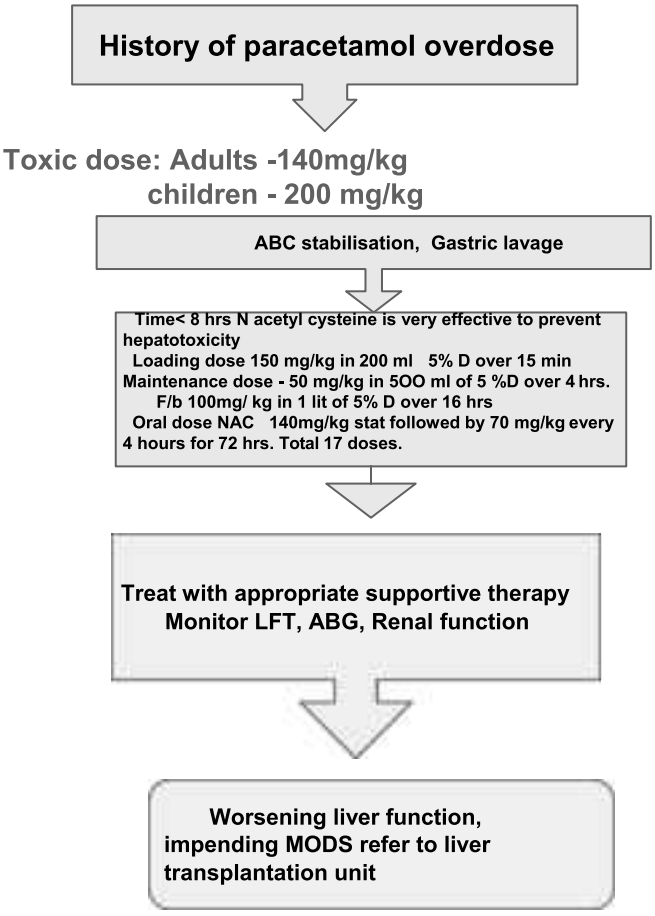
Treat with I.V mannitol
(1.5 - 2g/kg I.V over 30mins)

✦ For sulfonylurea overdose, I.V dextrose is not enough, as transient hyperglycemia triggers insulin release, leading to recurrent hypoglycemia.

Inj. Octreotide 50mcg (4-5mcg/kg/day)
By I.m or s.c route every 6hrs for 24 hrs.

12.7 PARACETAMOL POISONING

Management protocol



King college criteria for liver transplant

Acetaminophen induced ALF

Arterial PH< 7.3(regardless of HE) or all of the following

- INR >6.5
- Creatinine > 30Omicromol/ L
- HE grade 3-4

Non Acetaminophen induced ALF

- INR >6.5 (regardless of HE)

OR 3 of 5 of the following(regardless of HE)

- Age <10 or > 40
- Etiology - indeterminate , drug induced
- Time interval from icterus to encephalopathy > 7days
- INR> 3.5
- Bilirubin> 300 micromol/L

Annexure - 1

Compound/class	Antidote
Opioid	Naloxone: 0.04-0.05 mg iv, titrate up every few minutes till RR>12/min (Max: 5-10 mg) If apneic: 0.2-1 mg iv stat; If cardiac arrest is present: at least 2 mg iv stat
Benzodiazepine	Flumazenil: 0.1-mg doses (or 0.01 mg/kg) slow push over 1-2 minutes, repeat to a maximum of 1 mg, or until an effect is achieved. Recurrent sedation may occur.
Serotoninergic syndrome	Cyproheptadine: 12mg stat followed by 2 mg every 2 hours; Agitation: Lorazepam:2-4 mg iv or diazepam 5-10 mg iv
Paracetamol	N acetyl cysteine: 150 mg/kg in 200 ml 5% dextrose over 15-60 min then 50 mg /kg in 500 ml of 5% dextrose over 4 hours then 50 mg /kg in 500 ml of 5% dextrose over 8 hours then 50 mg /kg in 500 ml of 5% dextrose over 4 hours Total dose: 300 mg/kg in 20-21 hrs Late presentation/persistent hepatotoxicity: 50 mg/kg in 250 ml 5% dextrose over 8 hours to continue
Beta blockers/calcium channel blockers	For bradycardia: Atropine 1 mg IV; may repeat up to 3 total doses For severe poisoning: Calcium gluconate: 30 to 60 mL of 10% solution Insulin and dextrose: Regular insulin – 1 Unit/kg IV Dextrose – 25 to 50 grams IV; repeat for hypoglycemia; hold if serum glucose >300 mg/dL [16.7 mmol/L]; give potassium for hypokalemia (hypomagnesemia often associated with hypokalemia) Glucagon : 1-5 mg IV push, may repeat up to 15 mg total
Methhaemoglobinemia	Methylene Blue is identified if there is evidence of Methemoglobinemia of >30% or if there are symptoms of methemoglobinemia with a Saturation gap of more than 10. Inj. Methylene blue 1 to 2 mg/kg intravenously, given over five minutes. May be repeated in one hour if the methemoglobin level remains high (eg, >20 percent) and/or is increasing. However, administration of more than 2 to 3 doses (>7 mg/kg) is generally avoided due to the possibility of causing hemolysis.

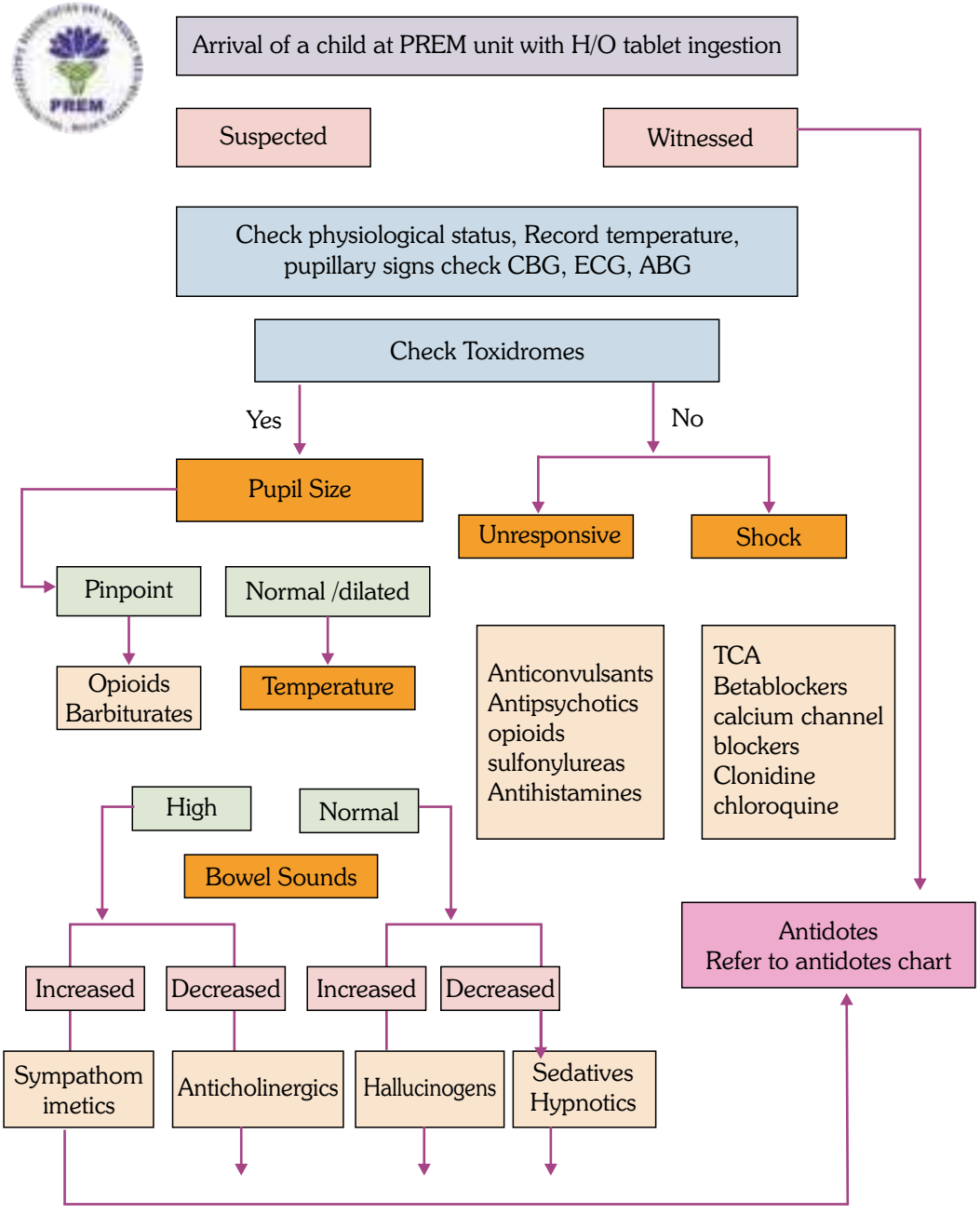
Annexure - 2

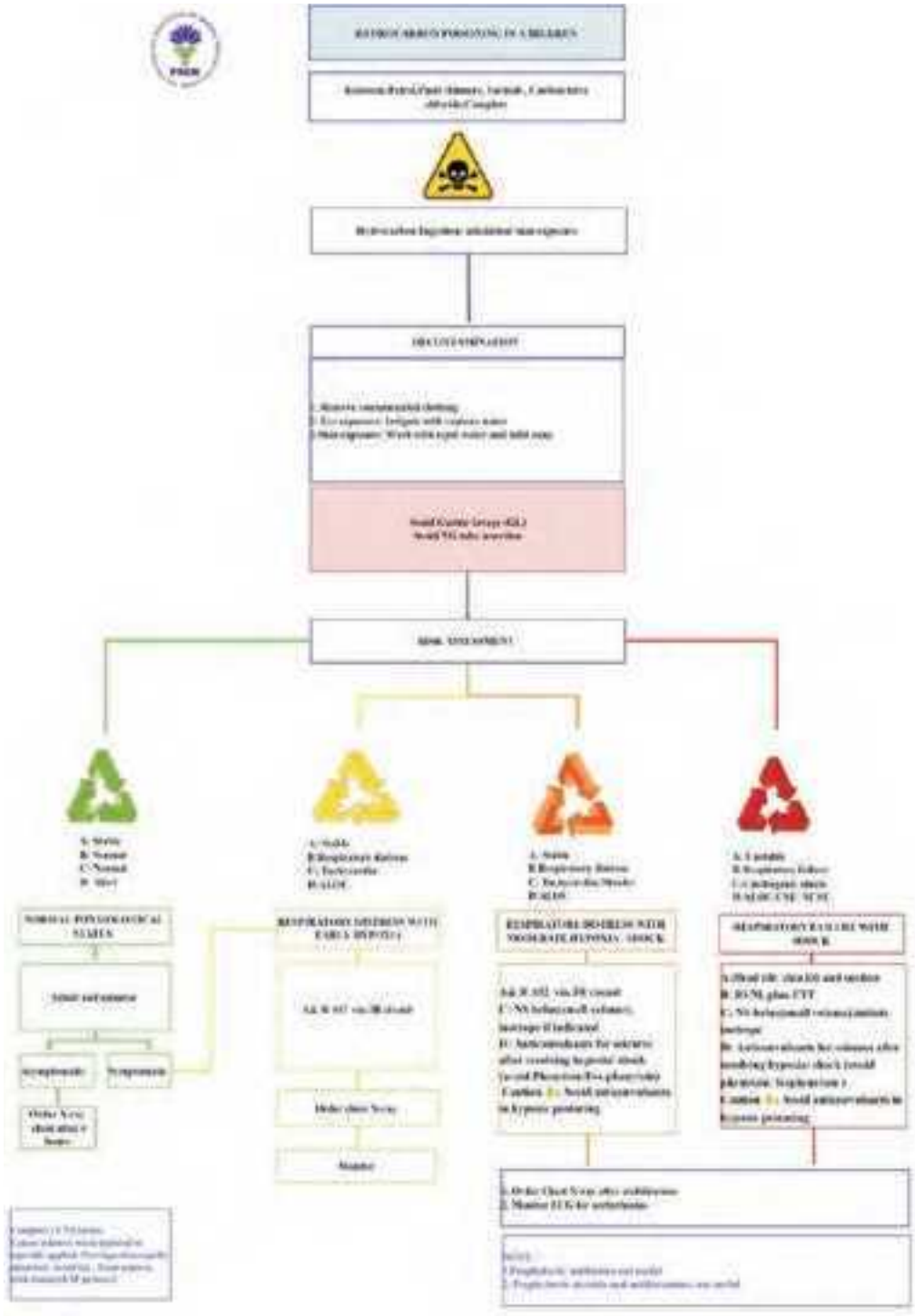
Drugs used in IMCU

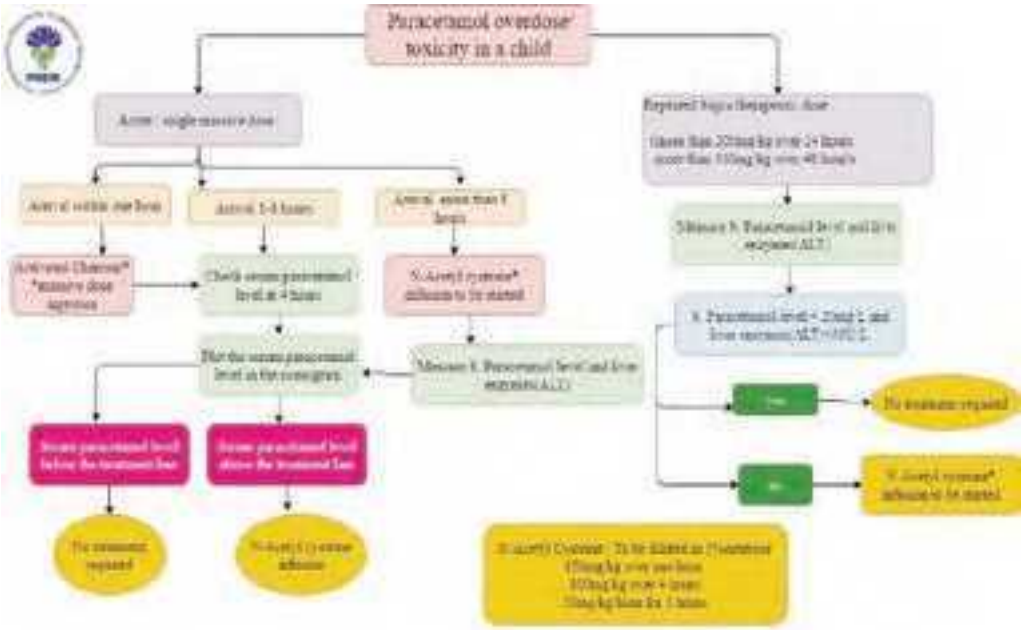
VECURONIUM	0.1mg/kg bolus then 1mcg/kg/min	1ml = 4 mg , 1 ampule=1 ml -4 mg	1 ampule bolus then 5 ampule in 5oo ml
PANCURONIUM	0.1mg/kg bolus then 1mcg/kg/min	1 ml =2 mg, 1 ampule = 2 ml-4 mg	1 ampule bolus then 5 ampule in 500ml NS 16 drops/min
PROPOFOL	0.5 -1 mg/kg bolus then 50mcg/kg/min	1ml = 10 mg , 1 vial = 10 ml - 100 mg	5 ml bolus then 3v in 500ml NS 12 drops/min
MIDAZOLAM	0.1-0.2 mg/kg bolus then0.1 - 2Mg/kg/hr	1 ml =1 mg, 5 ml vial =5 mg	5 ml bolus then 10 ml in 500ml NS(50 ml= 1 mg) 10 drops/min
FENTANYL	1 – 2 mcg/kg bolus then 1-10mcg/kg/hr	1ml ampule = 2ml = 100 mcg	2 ml bolus then 1 ml hrly
KCL	Not >20mEq/hr maximum 20 mEq/l through peripheral vein,100mEq/l through central line	1 amp = 10 ml= 20 mEq	5 ampule(100 mEq)in 500 ml NS =100 ml/hr
GLUCOSE INSULIN	2×5U regular insulin in 25%D over 30 min		100 drops/min
3% NACl	0.6×body weight ×(desired actual sodium),max 10mEq/day,1-2 mEq/hr	1 vial = 100 ml = 51.3 mEq	6-7 bottles/day, 6 -7 drops /min
CALCIUM GLUCONATE	10 ml 10% in 100 ml 5%D over 20 min	10 ml = 1 gm- 90 mg elemental calcium	10 ml in 100 ml 5%D = 150 drops/min
SODIUM BICARBONATE	0.5×body weight×(24 actual NaHCO3)	1 amp = 10 ml -10 mEq	In 5%D, 50% calculated dose over 4 hrs , remaining over next 18 hrs
MAGNESIUM SULPHATE	2 gm in 200 ml 5%Dor NS over 20 min	1ml = 500mg , 1 ampule=2 ml -1g	2 amp in 200 ml 5%D or NS = 150 drops /min
N-ACETYL CYSTEINE	150mg/kg over 15 min then 50mg/kg over 4 hrs then 100mg/kg over 20 hrs	1 ml =200 mg, 1 ampule = 2 ml-400 mg	18 amp in100 ml NS – 100 drops/min, 6 amp in 500 ml NS – 30 drops/min,12 amp in 500 ml NS – 5 drops/min
HEPARIN	5000 U bolus then 600 U /hr (12 U/kg/hr)	1 ml = 5000 U , 1 vial -5 ml - 25000 U	1 ml bolus then 3 ml in 500 ml NS 5 drops/min
L ORNITHINE ,L ASPARTATE(LOLA)	20 gm (4 amp) in 250 ml NS over 4 hrs	(OR) 5 gm (1 amp) in 300 ml NS 8 hrly	
MILRINONE(like dobutamine)contraindi-hyotension with hypovolemia	50mcg/kg over 10 mins then 0.25 -0.75 mcg/kg/min	10 ml vial = 1 ml = 1 mg	2.5 in 5 %D over 10 mins then 10 ml (10 mg) in 500 ml 5%D (1ml = 20 mcg) 1-1.5 ml/min
Forced alkaline dieresis(FAD)	NS 500 ml over 30 mins then 400 ml 5%D with 100 ml soda bicarbonate over 30mins then NS 10 Meq KCL	Urine output should be 3 ml/kg /hr otherwise give frusemide	3 cycles day Contraindication- renal ,cardiac failure, hypotension
NORADRENALINE	1-30mcg/min or 0.05-1mcg/kg/min	1ml=2mg, 1amp=2ml-4mg	1amp (4mg) in 500ml 5%D, 1ml=8mcg
DOPAMINE	5-20mcg/kg/min	1ml=40mg, 1amp=5ml-	2amp (400mg) in 500 ml 5%D,

		200mg	1ml=20mcg/kg/min
DOBUTAMINE	2-10mcg/kg/min	1ml=50mg,1amp=250mg	1amp(200mg)in 500ml 5%D, 1ml/min=5mcg/kg/min
ADRENALINEE	0.05-0.5mg/kg/min	1ml=1mg, 1amp=1ml-1mg	2.5ml(2.5mg) in 500ml 5%D, 1ml=5mcg, 1ml/min=0.1mg/kg/min
VASOPRESSIN	0.01-0.04U/min	1ml=20U-1amp=1ml	1amp in 500ml NS, 4-8drops/min
NITROGLYCERINE	10-200mcg/min	1ml=5mg, 1amp=5ml-25mg	1amp (25mg) in 500ml 5%D/NS, 1ml/min=50mcg/min
NITROPRUSSIDE	0.5-10mcg/kg/min	1amp=50mg	1amp (50mg) in 500ml 5%D/NS, 1ml=2mcg/kg/min
LABETOLOL	20mg every 10 min max 300 mg then 2 mg/min	1ml=5mg,1V=20ml	4ml bolus then 4ml in 500ml NS, 12 drops/min
AMIODARONE	150-300mg over 10 min then 1 mg/kg/hr for 6 hrs and 0.5mg/kg/hr for next 18 hrs	1ml=50mg,1amp=3ml-150mg	6ml in 5%D over 6 hrs then 0.5ml/hr for 18 hrs
LIGNOCAINE	1mg/kg bolus then 2-4mg/min	1ml=21mg	2.5ml bolus then 150ml in NS over 24 hrs
ISOPRENALINEE	1-4mcg/min	1ml=1mg, 1mp=2ml-2mg	2ml in 500ml in 5%D, 1ml/min = 4mcg/min
FRUSEMIDE	40-120mg bolus then 2-20mg/hr	1ml=10mg- 1amp=20mg	
AMINOPHYLLINE	500mg in 200 5%D-NS over 30 mins then 0.8 mg/kg/hr	1ml=25mg, 1amp=10ml-250mg	2 amp in 5%D, 100 drops/min over 30 min then 10 drops/min
ATROPINE	3-5mg/min (for OPC poisoning)	1ml=0.6mg, 1amp=1.2 mg	20 amp in 500ml NS – 100ml/hr
PRALIDOXIME	1g bolus then 500mg/hr	1amp= 10ml-500mg	5 amp in 400ml NS -100ml/hr
OCTREOTIDE	100mcg bolus then 50mcg/hr (UGI bleed)	1ml= 50/100mcg	5 amp in 500ml NS- 100ml/hr
PANTOPRAZOLE	80mg bolus then 8mg/hr	1V=40mg	1V in 500ml NS – 100 ml/hr
PHENYTOIN	20mg/kg bolus at 50mg/min	1ml=50mg, 1amp=2ml-100mg	10 amp in 200 ml NS – 100 drops/min
PHENOBARBITONE	20mg/kg bolus at 75mg/min	1ml=200mg, 1amp=2ml-400mg	2.5ml in 200ml NS -100 drops/min
THIOPENTONE	1mg/kg bolus then 1-2mg/kg/hr	1V=500mg	Dilute with 10ml NS 1ml bolus then 16 drops/min

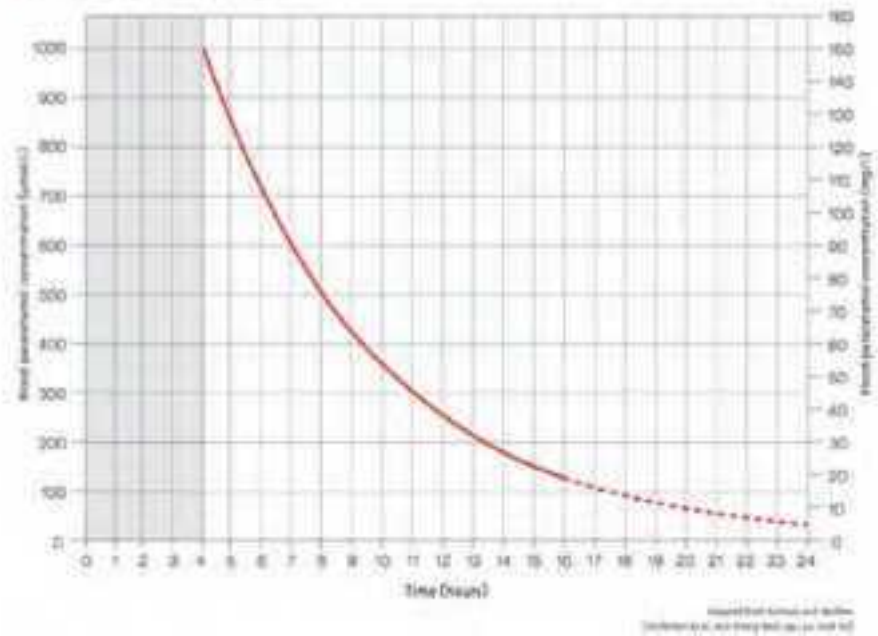
13. REFER TO TERTIARY CARE


Arrival of a child at PREM unit with H/O tablet ingestion
Suspected
Witnessed
Check physiological status, Record temperature, pupillary signs check CBG, ECG, ABG
Check Toxidromes
Yes
No
Pupil Size
Unresponsive
Shock
Pinpoint
Normal /dilated
Opioids Barbiturates
Temperature
Anticonvulsants Antipsychotics opioids sulfonylureas Antihistamines
TCA Betablockers calcium channel blockers Clonidine chloroquine
High
Normal
Bowel Sounds
Antidotes Refer to antidotes chart
Increased
Decreased
Increased
Decreased
Sympathom imetics
Anticholinergics
Hallucinogens
Sedatives Hypnotics





Paracetamol Treatment Nomogram[4]



Approach to button battery ingestion in children

Button batteries (size > 12 mm) can lodge inside the oesophagus (age < 12 years). Burns leading to necrosis can start within 2 hours. Button battery (BB) ingestion is usually not witnessed.

Suspect

1) Suspect BB if a child suddenly presents with unexplained symptoms: Stridor, wheeze, drool, vomiting, pain abdomen, refusal to feed, gagging or choking during feeds.
2) Consider BB INGESTION if the child has consumed coin or any other foreign body.

Confirm diagnosis by radiograph

3) Order x-rays with the following views:
   - Neck and oesophagus
   a) AP view: Look for "double rim"
   b) Lateral view: "step-off"
   - Abdomen: Location of coin shadow in or outside the stomach

Pre-hospital Treatment

- Do not induce vomiting.
- If witnessed, start honey immediately (ideally at home if available enroute to the PREM unit)
- Give 10 mL (2 teaspoons) of honey (commercial) orally once in 10 minutes x 6 doses for children older than 12 months.
  1. Avoid honey in infants less than 12 months
  2. Avoid honey if duration of BB ingestion is > 12 hours; the risk of oesophageal perforation worsens after 12 hours.
  3. Avoid honey if the child cannot swallow.
  4. Give 10 mL (2 teaspoons) of honey by mouth every 10 minutes for up to 6 doses. Do not worry about the exact dose or timing.
  5. Honey DOESN'T replace removal of BB.

Honey Delays Alkaline Burn Within Esophagus

Ensure "nil Per Oral" (exception Honey) until BB ingestion has been ruled out or endoscopically removed.

Check Size Of BB:

Look for imprint code from a companion or replacement battery, battery packaging, or product instructions for size of BB.

Shift To Center With Endoscopic Facilities

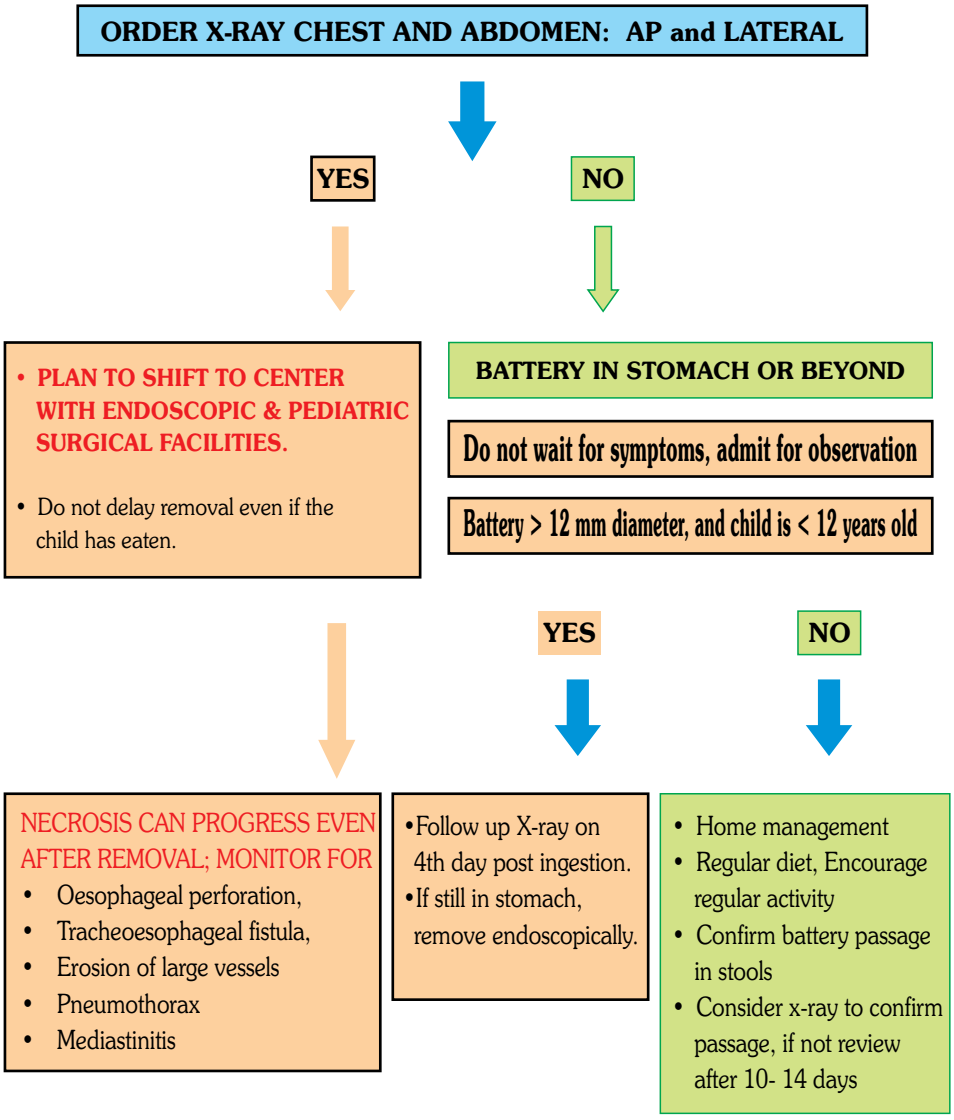
Button Battery Ingestion can Erode oesophagus within 2 Hours

Symptoms:

Noisy breathing (stridor), drooling, vomiting, choking, chest discomfort, cough. Maybe asymptomatic

Give honey (if > 1 Yr) 10 Ml every 10 mins upto 6 doses
if button battery ingestion is suspected

Ref: https://www.poison.org/battery/guideline


ORDER X-RAY CHEST AND ABDOMEN: AP and LATERAL
YES
NO
• PLAN TO SHIFT TO CENTER WITH ENDOSCOPIC & PEDIATRIC SURGICAL FACILITIES.
• Do not delay removal even if the child has eaten.
BATTERY IN STOMACH OR BEYOND
Do not wait for symptoms, admit for observation
Battery > 12 mm diameter, and child is < 12 years old
YES
NO
NECROSIS CAN PROGRESS EVEN AFTER REMOVAL; MONITOR FOR
• Oesophageal perforation,
• Tracheoesophageal fistula,
• Erosion of large vessels
• Pneumothorax
• Mediastinitis
•Follow up X-ray on 4th day post ingestion.
•If still in stomach, remove endoscopically.
• Home management
• Regular diet, Encourage regular activity
• Confirm battery passage in stools
• Consider x-ray to confirm passage, if not review after 10- 14 days

CAUSTICS (ACID/ALKALI) CAN CAUSE NECROSIS AND STRICTURE OF THE ESOPHAGUS

History: What chemical? Disinfectants, toilet cleaners, or dishwashing powders

!lkali. Liquefactive necrosis-Acid. Coagulative necrosis

When did the child ingest?

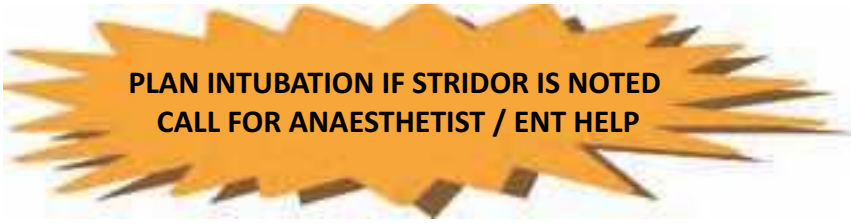
Symptoms? Vomiting, Drooling, Hoarseness, stridor and breathlessness, fever, dysphagia, and refusal of feeds

Local exam. **Tongue erythema, oedema of the lips, oral ulceration**

Systemic assessment. **!ssess physiological status**

MANAGEMENT IN CHILDREN

Do	Don't
Nil per oral	Gastric lavage
IV fluids	Stimulate emesis
Base line x-ray. Lateral and chest !P	Cathartics (agents that cause diarrhea)
Plan endoscopy within 24 hours of ingestion	Activated charcoal
Consider proton pump inhibitor. 0/7-3/5 mg/kg/dose for 2-3 weeks	Naso gastric tube insertion
	Barium swallow
	Steroids
	Antibiotics (unless infection or perforation is suspected)



Even if oro-pharyngeal injury is mild, plan for endoscopy. Esophageal injury can be severe/

PREM Protocol: Management of Scorpion Sting in PED

Scorpion Sting

LOCAL EFFECTS

- ☐ Oral paracetamol 15mg/kg/dose
- ☐ Ice compression at the site of bite

SYSTEMIC EFFECTS (AUTONOMIC STORM)

Profuse sweating, vomiting, cold peripheries, hyper-salivation, Priapism, bradycardia

NO SHOCK

DISABILITY
Alert, Tone, and posture: N, Eyes: No conjugate deviation, No lid twitch, No nystagmus, PERL

AIRWAY
Stable

BREATHING
RR:N/No grunt/No retractions/Thoracic respiration/ No crept
SaO_2 100%

CIRCULATION
HR: N, Perfusion: cool periphery+, pulse pressure N
Liver span: N, BP↑/ MAP: ↑

- ☐ Prazosin 30µg/kg/dose by oral/NGT
- ☐ Maintain supine position to prevent hypotension.

Prazosin can be repeated after 3 hours and 6th hourly till extremities are warm and dry (usually not more than 4 doses are required).

COMPENSATED CARDIOGENIC SHOCK

DISABILITY
Impaired-alert/ Hyperalert/lethargy/ Tone & posture: N /No-conjugate deviation/No nystagmus No lid twitch, PERL

AIRWAY
Stable

BREATHING
RR:↑/No grunt Retractions++/Thoraco-abdominal respiration±/ Crept±, SaO_2 100%

CIRCULATION
HR: Tachycardia/Cold shock (pulse pressure narrow) Liver span: N/hepatomegaly/ SBP: ↑/M!P↑

- ❖ O_2 through Jackson-Rees Circuit
- ❖ [1]NS/RL boluses @5-10ml/kg
- ❖ Prazosin 30µg/kg/dose

- ☐ Shock + no PE (Evidence of hypovolemia) (vomiting+ or severe perspiration+)
- ☐ Continue O2 through JR circuit
- ☐ Continue 5-10 ml/kg until shock resolves (Small volume shock)

- ❖ [1]Shock +PE or hepatomegaly+
- ❖ Stop fluid bolus,[1,2]Initiate dobutamine.
- ❖ Intubate to provide PEEP.

HYPOTENSIVE CARDIOGENIC SHOCK

DISABILITY
Pain responsive /agitation/combative Thirst/Unresponsive Tone and posture: Abn Conjugate deviation± Nystagmus ±/Lid Twitch± /PERL

AIRWAY
Stable

BREATHING
RR:? /? Grunt± /Retraction+/ Thoraco-abdominal respiration±/ Crept± SaO_2 <92%±

CIRCULATION
HR: ? /"N" Relative bradycardia
Gallop± Muffled±, Cold shock, Liver span: N/hepatomegaly, SBP: ?

- ❖ O_2 through JR circuit
- ❖ [1]NS/RL bolus @5 ml/kg
- ❖ Epinephrine infusion @0.3-0.5µg/kg/min
- ❖ [1]Intubate to provide PEEP.
- ❖ Withhold Prazosin until BP normalizes

CPR Alert

x [1]Risk of cardiogenic or non-cardiogenic pulmonary edema complicates shock management due to scorpion envenomation. During fluid therapy (small volume shock), monitor for airway instability, pink froth, increase or decrease in respiratory rates, grunt, retractions, abdominal respiration, fresh rales, gallop, increasing liver span, agitation, fighting the mask and drop in oxygen saturation (i.e., signs of pulmonary edema). If anyone or a cluster of these signs develop, stop further fluid , initiate an inotrope, and prepare to intubate.

PREM Protocol: Approach to Snake Bite

Bite: No hypoxia or Shock

DISABILITY
LOC: Alert
T&P: Normal
Eyes: EOM PERL

AIRWAY **Stable**
BREATHING **RR:** N
Grunt: No
Stridor: No
Retractions: No
Air entry: +
Respiration: Thoracic
Sounds: No
SpO_2: >94%

CIRCULATION
HR: N / ↑ ; **H&S:** No muffling, no gallop
P&C: Warm, pink; **Pulses:** +++/++
CRT: <2 seconds; **Liver span:** N, soft; **SBP:** N
DBP: >50% of SBP; **PP:** 30–40 mmHg; **MAP:** N

OTHERS: Cellulitis (severe, painful progressive swelling, rapidly crossing the joint); **Bite mark**; Tender lymphadenitis
WBCT: N/>20 minutes; **Confirmed snake bite**

- **Reassure, immobilize the affected limb.**
- **Tetanus prophylaxis, antibiotics for cellulitis (not bite mark)**
- **ASV 8-10 vials in Normal Saline over 1–2 hours**

Haemotoxic: No-hypoxia or shock

DISABILITY
LOC: Alert
T&P: Normal
Eyes: EOM PERL

AIRWAY Stable
BREATHING **RR:** N
Grunt: No
Stridor: No
Retractions: No
Air entry: +
Respiration: Thoracic
Sounds: No
SpO_2: >94%

CIRCULATION
HR: N/ ↑ ; **H&S:** No muffling, no gallop
P&C: Warm, pink; **Pulses:** +++/++
CRT: <2 seconds; **Liver span:** N, soft; **SBP:** N
DBP: >50% of SBP; **PP:** 30–40 mmHg; **MAP:** N

OTHERS: WBCT >20 minutes bleed from bite site
Other bleeding manifestations

- **ASV 8-10 vials.**
- **Repeat WBCT 6 hourly after ASV.**
- **If WBCT >20 minutes repeat 8–10 vials (Max 30 vials).**
- **If patient continues to bleed briskly, give ASV within 1 to 2 hours.**
- **Consider FFP or cryoprecipitate or fresh blood after neutralizing dose of ASV.**

Neurotoxic: No hypoxia or shock

DISABILITY
LOC: Alert
T&P: Descending paralysis
Eyes: Diplopia
Ptosis
dysarthria
Dysphagia
PERL

AIRWAY Stable
BREATHING **RR:** N
Grunt: No
Stridor: No
Retractions: No
Air entry: +
Respiration: Thoracic
Sounds: No
SpO_2: >94%

CIRCULATION
HR: N/ ↑ ; **H&S:** No muffling, no gallop
P&C: Warm, pink; **Pulses:** +++/++
CRT: <2 secs; **Liver span:** N, soft; **SBP:** N
DBP: >50% of SBP; **PP:** 30–40 mmHg; **MAP:** N

- **ASV 8-10 vials over 1 hour. Repeat 10 vials if no improvement.**
- **Atropine 0.02–0.05 mg/kg, ½ hourly**
- **Neostigmine 40 µg/kg ½ hourly until neurological recovery. Subsequent doses given (10-40 µg/kg) at 1, 2, 6 and 12 hours. (Improvement is noted only in cobra bite)**
- **If no improvement after 3 doses of Neostigmine + atropine give Inj. Calcium Gluconate 1-2 mL/kg (1:1 dilution over 5–10 minutes). (Max dose: 10 mL). Repeat 6 hourly. (useful for Krait bite).**

UNKNOWN BITE:

- If 20-minute Whole Blood Clotting Time is normal: No ASV.
- Repeat WBCT ½ hourly for 3 hours, then hourly for 24 hours.
- If WBCT is >20 minutes: Start Anti-Snake Venom (ASV)

PRIOR to ASV administration, prepare:

- IM Adrenaline: 0.01 mL/kg of 1:1000 (max 0.5 mL) or 0.1 mL/kg of 1: 10,000 IV
- IV Chlorpheniramine maleate: 0.2 mg/kg
- IV Hydrocortisone: 2–6 mg/kg
- When allergic symptoms resolve/ PREM triangle becomes normal, continue ASV infusion at a slower rate for 10–15 minutes. If child remains stable, continue infusion at normal rate.

SURGICAL ISSUES

- Compartment syndrome is rare in children.
- Early and adequate antivenom is the best strategy to prevent irreversible muscle damage and gangrene

Hemotoxic:

Hypoxia; Cardiovascular Dysfunction ±
Non-Convulsive Status Epilepticus ±

DISABILITY
LOC: Responsive to pain ±
Unresponsive ±
T&P: Abnormal
Eyes: Conjugate deviation ±
Nystagmus ±
lid twitch ±
PERL

AIRWAY: Stable or Unstable
BREATHING RR: ↑
Grunt: ±
Stridor: +
Retractions: +
Air entry: +
Respiration: Thoracic
Sounds: +
SpO_2: <94%

CIRCULATION: HR: HR: ↓ / ↓ ; H&S: Muffling ± gallop ±; P&C: Cool, dusky; Pulses: +++/+
CRT: >2 seconds; Hepatomegaly ±; SBP: N/ ↓
DBP: <50% of SBP; PP: N/ ↓ ; MAP: ↓

- Oxygen via Jackson Rees circuit
- NS 10 mL/kg boluses (small volume)
- Inotropic support if needed.
- ASV maximum 30 vials
- Blood & blood components

Neurotoxic:

Hypoxia; Cardiovascular Dysfunction ±
Non-Convulsive Status Epilepticus ±

DISABILITY
LOC: Unresponsive
T&P: floppy
Eyes: Conjugate deviation ±
Nystagmus ±
lid twitch ±
PERL

AIRWAY
Unstable
BREATHING
Apnoea
SpO_2: <94%

CIRCULATION: HR: ↓ / ↓ ; H&S: Muffling ± gallop ±; P&C: Cool, dusky; Pulses: +++/+
CRT: >2 seconds; Hepatomegaly ±
SBP: N/ ↓ ; DBP: <50% of SBP; PP: N/ ↓ ; MAP: ↓

- Oxygen via BVM ventilation, Plan early intubation. Cardiac massage & epinephrine if cardiac arrest or low BP
- NS 10 mL/kg (small volume shock)
- ASV 8-10 vials maximum 30 vials
- Inj. Atropine, Neostigmine ½ hourly (see above)

Standard Treatment Guidelines. Management of Snake Bite. Ministry of Health and Family Welfare, Government of India. August 2017. National Health Mission–Strengthening of Pediatric Emergency Care System in Tamil Nadu–Establishment of Pediatric Resuscitation and Emergency Units under Tamil Nadu Accidents and Emergency Care Initiative under the name of PREM G.O(D)No. 539, Department of Health and Family Welfare, dated 30.11.19.

Snakes of Medical Importance in Tamil Nadu

BASIC FACTS OF SNAKE BITE

1. All animal bites are medical emergency
2. 30 percent of the Snake bites are Venomous
3. All Snake bites victims have to be admitted and observed for minimum of 24 hours from the time of admission.
4. Before removing the tourniquet, start a lifeline and keep victim in supine posture.
5. Watch for early symptoms and signs of envenomation and complications
6. No need to refer every victim to a higher centre
7. Usually Krait bites do not cause local signs
8. Both Adults and Children require the same dose of ASV
9. Pregnant women are treated in the same way as other victims
10. Possibility of dry bite should be kept in mind

Stepwise clinical approach to snake bite

Step - 1 : Check vitals ? Unstable
? ?
Stable ? Stabilise

Step - 2 : History (Time of Bite, Body site, Local Envenomation, Neuro and Hemotoxic symptoms, Any first aid given, Co-morbids)

Step - 3 : Examination - Local, Neurological and Hemotoxic signs of Envenomation

Step - 4 : Investigations - (As per the guidelines given)

Step - 5 : Management (Observation and As per the guidelines given)

Step - 6 : Referral - (As per the guidelines given)

Dont's of Snake Bite:

The traditional methods such as application of tourniquet, cutting (incision) and suction, washing the wound, snake stone or other methods have adverse effects and hence, they have to be discarded.

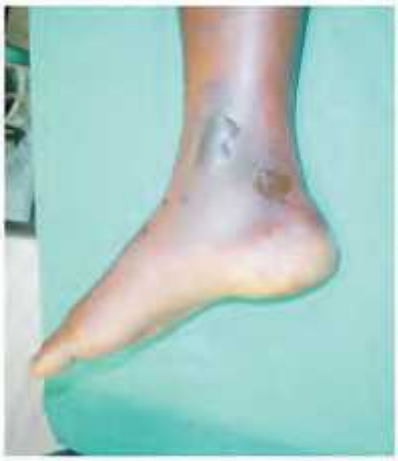
Do's of Snake Bite:

1. Admit the victim immediately.
2. Ask effectively.
3. Assess quickly.
4. Act swiftly.
5. Administer medication meticulously.
6. Address to the wound properly.
7. Anticipate complications keenly.
8. Avoid errors carefully.
9. Ascertain the status repeatedly.
10. Amicable with patients and care givers and show empathy.
11. Advise on follow up accordingly.
12. Arrange for referral early.

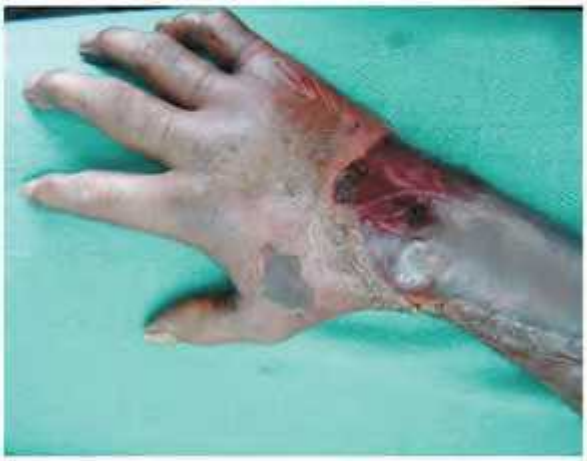
Snakes, clinical aspects and therapeutic response

Feature	Cobras	Kraits	Russells Viper	Saw Scaled Viper	Hump Nosed Viper
Local Pain / Tissue Damage	YES	NO	YES	YES	YES
Ptosis / Neurological Signs	YES	YES	YES!	NO	NO
Haemostatic abnormalities	NO	NO!	YES	YES	YES
Renal Complications	NO*	NO*	YES	NO*	YES
Response to Neostigmine	YES	NO?	NO?	NOT applicable	NOT applicable
Response to ASV	YES	YES	YES	YES	NO

[* If features of renal failure are noted search for other causes / mechanisms]



Typical signs of local envenomation
Edema,blister and joint swelling



Cellulitis with compartmental syndrome

Details of local envenomation

- **P**ain- pain at the site of bite, swelling and regional lymphnode
- **O**ozing- sero / sanguinous oozing from the site of bite
- **N**ode- development of an enlarged tender lymphnode draining the bitten limb
- **D**iscoloration- discoloration at the site of bite
- **S**welling – swelling is seen at the site of the bites on the digits (toes and especially fingers); local swelling develops in more than half of the bitten limb immediately (in the absence of the tourniquet) and swelling extends rapidly beyond the site of bite (eg. beyond the wrist or ankle within a few hours of bites on the hands or feet)

Neurotoxicity

Assess for progressive descending motor paralysis starting from ptosis.

20 Minutes Whole Blood Clotting Test (20WBCT)

Advantages	Requirements	Procedure
• The most reliable test of coagulation. • Can be carried out, at the bedside. • Does not require specialised training.	• Dry glass test tube (clean and new) • 2ml disposable syringe • Cotton • Antiseptic solution • Clean gloves (one pair) • (The test tube must not have been washed with detergent, as this will inhibit the contact element of the clotting mechanism)	• Wash hands with soap and water. • Wear the gloves • Collect 2ml blood from the peripheral vein of the unaffected limb • Remove the needle and transfer the blood along the walls of the test tube • Keep the test tube untouched and unshaken in a safe place near the patient's bedside at ambient temperature for 20 minutes • Note the time • After 20 minutes the test tube is gently tilted and if the blood is still liquid then the patient has incoagulable blood.

Other Useful Tests:

- Clinical test:
  - PR / BP / RR / Postural Blood Pressure
- Laboratory studies:
  - Haemoglobin / PCV / Platelet Count/ PT / APTT / FDP / D-Dimer
  - Peripheral Smear / Blood grouping / Rh typing
  - Urine Tests for Proteinuria / RBC / Haemoglobinuria / Myoglobinuria
  - Biochemistry for Serum Creatinine / Urea / Electrolytes / Oxygen Saturation
- Imaging studies :
  - X-Ray Chest / CT / Ultrasound (whenever required)
- Others
  - Electrocardiogram
  - Special investigations depending upon clinical status.
  - Ocular fundus examination

ASV Administration

ASV should be administered only if a patient develops one or more of the following signs / symptoms of envenomation.

Systemic envenoming

- Evidence of coagulopathy primarily detected by 20 minutes WBCT or visible spontaneous systemic bleeding, bleeding gums, etc.
- Evidence of neurotoxicity: ptosis, external ophthalmoplegia, muscle paralysis, inability to lift the head etc.,

Local envenomation

Purely local swelling, even if accompanied by a bite mark from an apparently venomous snake, is not grounds for administering ASV if a tourniquet or tourniquets have been applied. These themselves can cause swelling. Once they have been removed for 1 hour and the swelling continues, then it is unlikely to be as a result of the tourniquet and administration of ASV may be justified.

Dosage

The initial dose is 10 vials of ASV (Polyvalent) to be administered over a period of 1 hour as an infusion in Isotonic saline (5 to 10 ml per Kg body weight).

Available ASV is polyvalent (Krait, Cobra, Russel l and Sawscale d viper) and marketed in liquid or lyophilised preparations in 10ml vial.

ASV neutralises the unbound venom, hence give it early if indicated.

ASV should not be pushed as IV bolus or IM directly or Local infiltration.

Some may develop allergic reaction / anaphylaxis. Hence careful monitoring is essential.

Maximum of 30 vials can be administered in a victim with Envenomation

Repeat dose of 10 vials of ASV can be considered after 1 to 2 hours, if Neurotoxicity worsens or patient has gone for respiratory failure. For Haemotoxicity repeat 20 minutes WBCT after 6 hours and if prolonged give 5 to 10 vials of ASV.

Referral aspects for snakebite

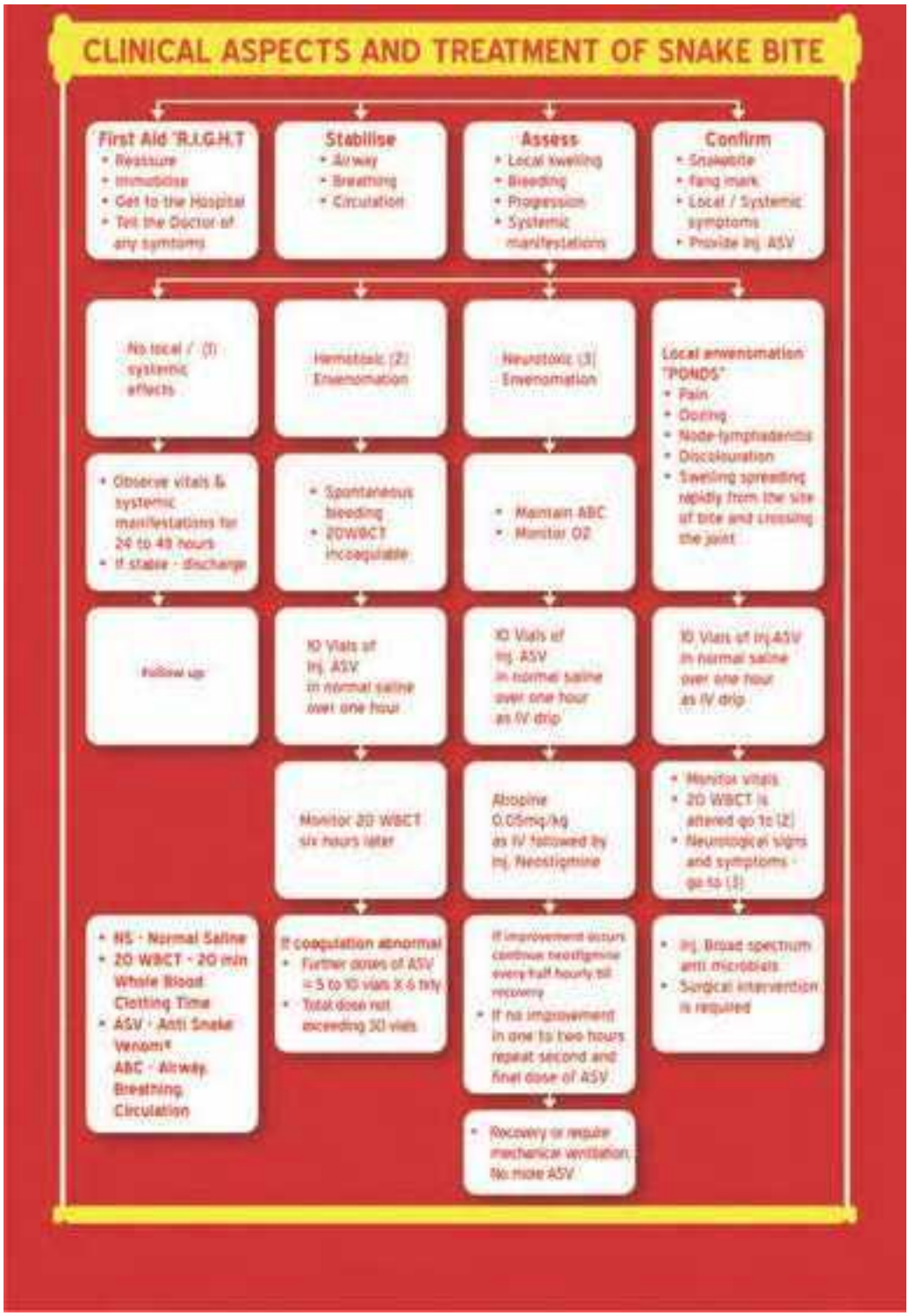
Who needs?	When to refer?	Where to refer?
Patient requiring • Respiratory support • Deteriorating neurologic manifestations • Surgical intervention-Necrosis / Fasciotomy • Spontaneous persistent bleeding • Co-morbid diseases • Acute impending kidney failure	• Refer the patient after stabilising the case and after giving injection ASV	Refer to higher institution having • Ventilator • Dialysis facilities • Measures to provide further supportive treatment.

Discharge Criteria: If there is no symptoms or signs of envenomation after 24 to 48 hours of admission, victim may be discharged and educate on prevention.

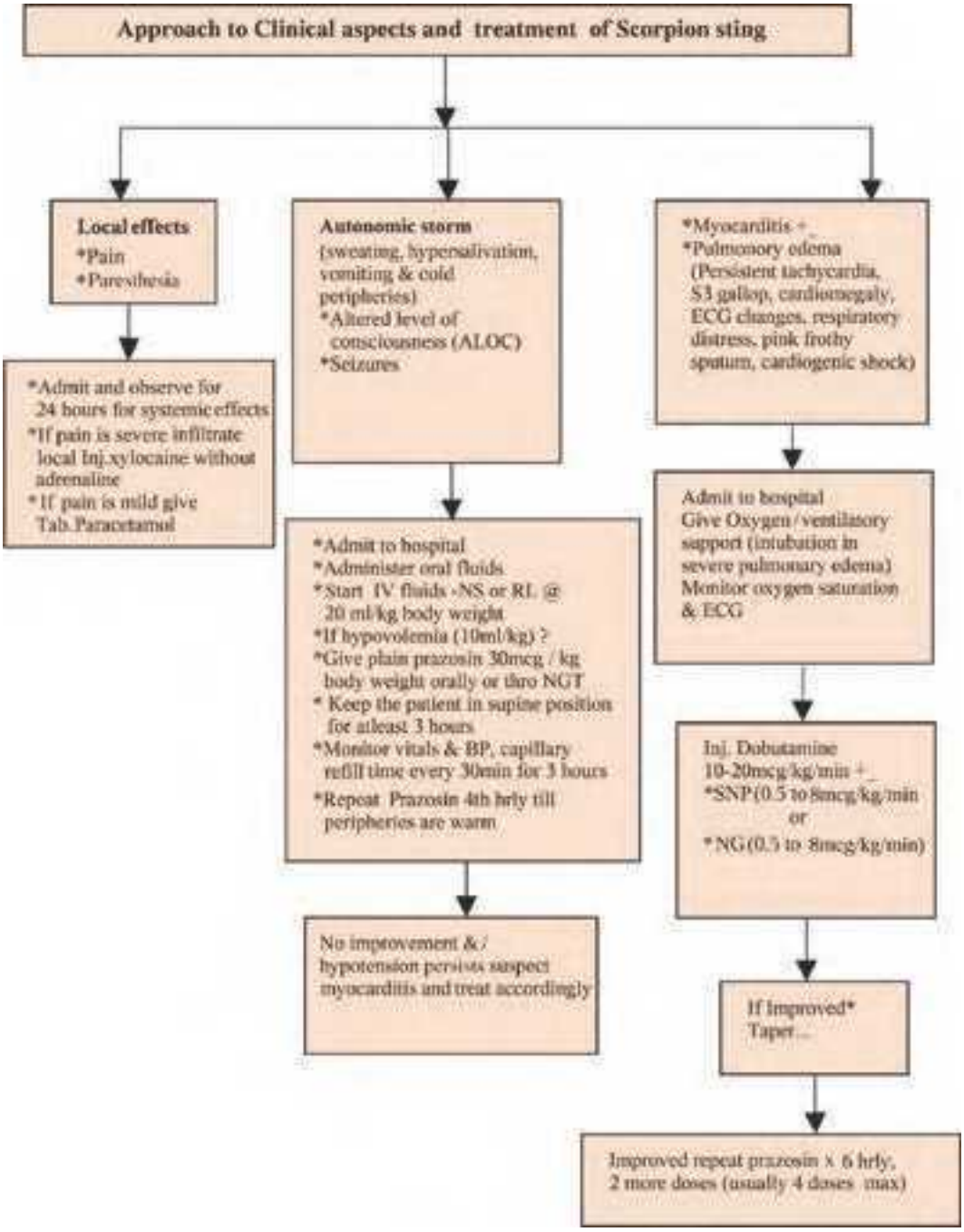
PREVENTION OF SKAKEBITE

At Home	In the Fields	Walking
Do's • Be tidy to discourage rats • Repair / fill in rat holes, cover drainpipes with mesh • Sleep on a cot **Dont's** • Keep rubbish or animal feed on the ground • Pile stones, titles, wood, coconut husks or store crops near the house or paths • Keep plants / pots close to windows & doors	**Do's** • Use a torch • Wear shoes • Watch where you step **Dont's** • Put your feet or hands into places you can't use **Be Careful while** • Grass cutting • Weeding • Picking up piles of grass etc. • Harvesting crops & fruits • Wood collecting, working at the base of trees in mulch, leaf litter or thick vegetation	**Be Careful** • At dusk/dawn or at night • On paths close to / with thick vegetation or leaf litter • Near water & white bathing. Look Out for small snakes at the start of the rainy season

SUMMARY


CLINICAL ASPECTS AND TREATMENT OF SNAKE BITE
First Aid "R.I.G.H.T
• Reassure
• Immobilise
• Get to the Hospital
• Tell the Doctor of any symtoms
Stabilise
• Airway
• Breathing
• Circulation
Assess
• Local swelling
• Bleeding
• Progression
• Systemic manifestations
Confirm
• Snakebite
• Fang mark
• Local / Systemic symptoms
• Provide Inj. ASV
No local / (1) systemic effects
Haemotoxic (2) Envenomation
Neurotoxic (3) Envenomation
Local envenomation "PONDS"
• Pain
• Oozing
• Node-lymphadenitis
• Discolouration
• Swelling spreading rapidly from the site of bite and crossing the joint
• Observe vitals & systemic manifestations for 24 to 48 hours
• If stable - discharge
• Spontaneous bleeding
• 20WBCT incoagulable
• Maintain ABC
• Monitor O2
Follow up
10 Vials of Inj. ASV in normal saline over one hour
10 Vials of Inj. ASV in normal saline over one hour as IV drip
10 Vials of Inj.ASV in normal saline over one hour as IV drip
Monitor 20 WBCT six hours later
Atropine 0.05mg/kg as IV followed by Inj. Neostigmine
• Monitor vitals
• 20 WBCT is altered go to (2)
• Neurological signs and symptoms - go to (3)
• NS - Normal Saline
• 20 WBCT - 20 min Whole Blood Clotting Time
• ASV - Anti Snake Venom
ABC - Airway, Breathing, Circulation
If coagulation abnormal
• Further doses of ASV = 5 to 10 vials X 6 hrly
• Total dose not exceeding 30 vials
If improvement occurs continue neostigmine every half hourly till recovery
• If no improvement in one to two hours repeat second and final dose of ASV
• Inj. Broad spectrum anti microbials
• Surgical intervention is required
• Recovery or require mechanical ventilation, No more ASV

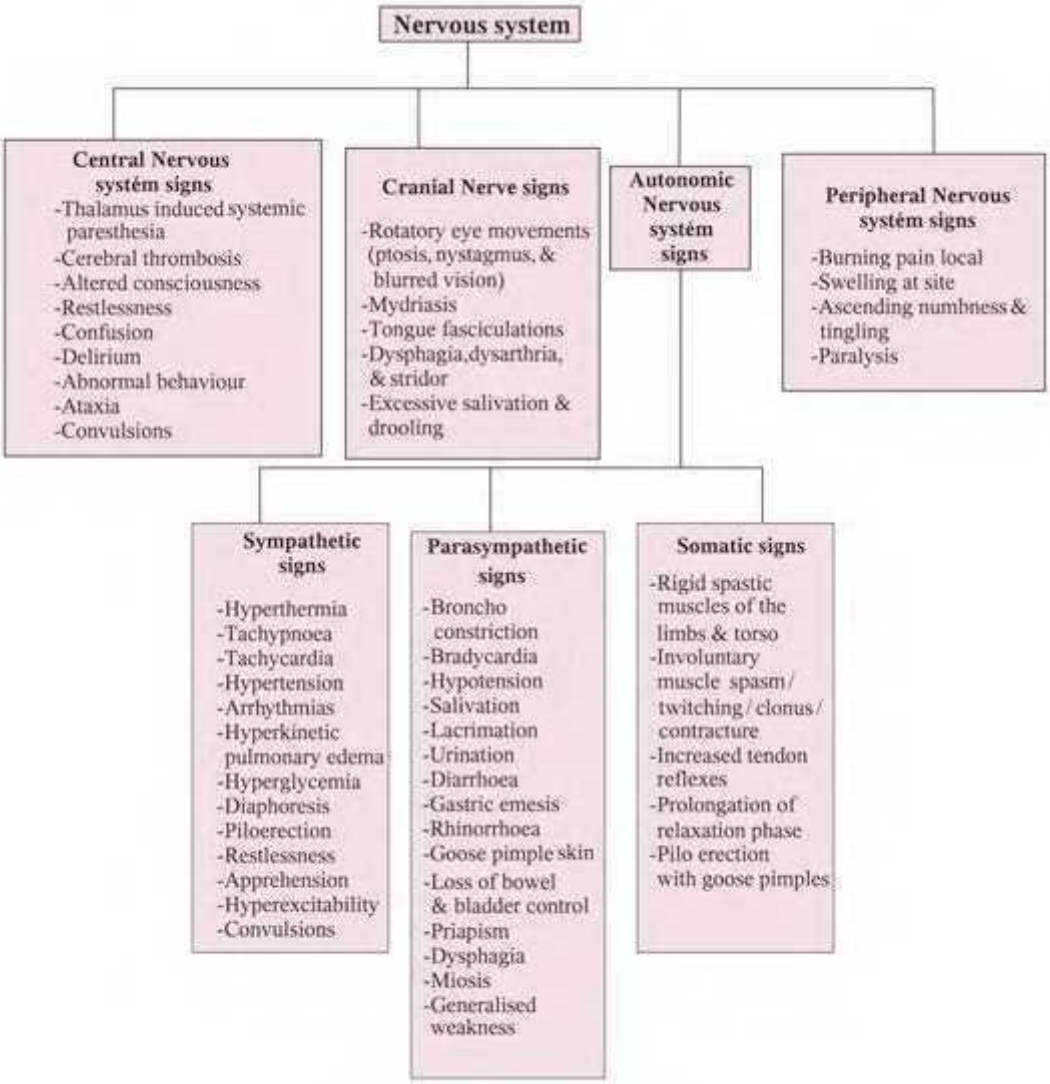
Algorithmic approach to scorpion sting



NGT - Nasogastric Tube; IV - Intravenous; NS – Normal Saline;

RL - Ringer Lactate; NG – Nitroglycerine; SNP - Sodium Nitroprusside

Nervous system signs



Non-neurological signs

* Cardiovascular signs
  - Hypotension
  - Hypertension
  - Tachycardia (bradycardia at times)
  - Cardiac dysrhythmia
  - Transient apical pansystolic murmur
  - Cardiovascular collapse
  - Cardiogenic shock
  - Cardiac dysfunction

* Respiratory Signs
  - Tachypnoea
  - Pulmonary edema
  - Respiratory failure

* Gastro intestinal Signs
  - Dysphagia
  - Excessive salivation
  - Nausea and vomiting

* Hematologic Signs
  - Platelet aggregation
  - Disseminated intra vascular coagulation (DIVC)

* Metabolic Signs
  - Hyperglycemia
  - Increased lactic acidosis
  - Electrolyte imbalance

* Genitourinary Signs
  - Acute renal failure
  - Rhabdomyolysis
  - Priapism

* Allergic Signs
  - Urticaria
  - Angioedema
  - Bronchospasm
  - Anaphylaxis

"Self Harm is preventable; Treatable with early appropriate intervention!
The Secret of successful treatment is close monitoring and appropriate early intervention!!"