RETROSPECTIVE ASSESSMENT OF POISON CASES IN GOVERNMENT DISTRICT HEAD QUARTERS HOSPITAL TIRUPPUR

Dissertation submitted to

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

Chennai

In partial fulfillment of the requirement for the award of the degree of

MASTER OF PHARMACY

In

PHARMACY PRACTICE

Submitted by

S.B.AAMIN

REG. NO: 261540401

Under the guidance of

Dr. R. SENTHIL SELVI M.Pharm., Ph.D.,

PROFESSOR,

DEPARTMENT OF PHARMACY PRACTICE,

THE ERODE COLLEGE OF PHARMACY & RESEARCH INSTITUTE

ERODE.

OCTOBER – 2017.

EVALUATION CERTIFICATE

This is to certify that dissertation work entitled "**RETROSPECTIVE ASSESSMENT OF POISON CASES IN GOVERNMENT DISTRICT HEAD QUARTERS HOSPITAL TIRUPPUR"** Submitted by **REG NO: 261540401** to **THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY, CHENNAI**, in partial fulfillment for the **Degree of MASTER OF PHARMACY** is a bonafide thesis work carried out by the candidate at the Department of Pharmacy Practice, The Erode College of Pharmacy and Research Institute, Erode, was evaluated by us during the academic year **2016-2017.**

Internal Examiner **External Examiner**

Convener of Examination

Examination center: The Erode College of Pharmacy and Research Institute, Erode.

Date:

The Erode College of Pharmacy and Research Institute

Dr. R. Senthil Selvi M.Pharm., Ph.D.,
Professor,
Department of Pharmacy Practice,
The Erode College of Pharmacy and Research Institute,
Erode -638112.

Certificate

This is to certify that dissertation work entitled "**RETROSPECTIVE ASSESMENT OF POISON CASES IN GOVERNMENT HOSPITAL TIRUPPUR"** Submitted by **REG NO: 261540401** to **THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY, CHENNAI**. In partial fulfillment for the award of **Degree of MASTER OF PHARMACY IN PHARMACY PRACTICE,** is the bonafide work carried out under the guidance and direct supervision of **Prof. Dr. R. SENTHIL SELVI M.Pharm., Ph.D.,** department of pharmacy practice, The Erode College of Pharmacy and Research Institute, Erode-638112, during academic year **2016-2017.**

Dr .R.SENTHIL SELVI M.Pharm., Ph.D.,
Professor, Department of Pharmacy Practice.

Place: Erode.
Date:

The Erode College of Pharmacy and Research Institute

Dr. V.Ganesan M.Pharm., Ph.D.,

Principal,

The Erode College of Pharmacy and Research Institute,

Erode - 638112.

Certificate

This is to certify that the investigation described in the dissertation entitled "**RETROSPECTIVE ASSESMENT OF POISON CASES IN GOVERNMENT HOSPITAL TIRUPPUR"** submitted by **REG NO: 261540401** to **THE TAMILNADU Dr. M.G.R.MEDICAL UNIVERSITY, CHENNAI.** In partial fulfillment for the award of **Degree of MASTER OF PHARMACY IN PHARMACY PRACTICE** is the bonafide work carried out under the guidance and direct supervision of **Prof. Dr. R.SENTHIL SELVI M.Pharm., Ph.D.,** Department of Pharmacy Practice, The Erode College of Pharmacy and Research Institute, Erode-638112, during the academic year **2016-2017**

Dr.V.Ganesan M.Pharm., Ph.D.,

PRINCIPAL.

Place: Erode.

Date:

GOVERNMENT DISTRTICT HEADQUARTERS HOSPITAL

TIRUPUR - 641601

This is to certify that Mr. AAMIN.S.B., Post graduate student at The Erode college of pharmacy, Erode was doing a research project under in government district headquarters hospital, tirupur as a part of his M.Pharm(pharmacy practice) curriculum. The project was titled ***"RETROSPECTIVE ASSESMENT OF POISON CASES IN GOVERNMENT HOSPITAL TIRUPUR"*** and was done during the periods of November 2016 to june 2017(7 months). I wish him success in all future endeavors.

21/6/17

Hospital Superintendent
Govt. Head Qrs, Hospital
TIRUPPUR- 641 608

DECLARATION

The Research Work Embodied in this Dissertation Work entitled " **RETROSPETIVE ASSESSMENT OF POISON CASES IN GOVERNMENT HOSPITAL TIRUPPUR"** was carried out by me in the Department of Pharmacy Practice, The Erode College of Pharmacy, Erode, under the direct supervision of **Dr .R.Senthil Selvi M.Pharm., Ph.D.,** The Erode College of Pharmacy and Research Institute, Erode. This Dissertation submitted to **THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY, CHENNAI,** as a partial fulfillment for the award of **Degree in Master of Pharmacy in Pharmacy Practice** during the academic year 2016-2017. The work is original and has not been submitted in part or full for the award of any degree or diploma of this or any other university.

Reg.no: 261540401

Place: Erode.

Date:

ACKNOWLEDGEMENT

The secret of success is motivation, dedication, confidence on self and above all the blessing of god. Success is an outcome of collaborated efforts aimed that achieving different goals. I hereby take this opportunity to acknowledge all those who have helped me in the completion of this dissertation work.

First and foremost I am obliged to **ALLAH "the most compassionate, the most merciful",** without his blessings this project work would never have been veracity.

I consider it as a great Honour to express my deep sense of gratitude and indebtedness to **Dr. R. Senthil Selvi M.Pharm., Ph.D.,** Professor, Department of Pharmacy Practice, for her valuable suggestions, remarkable guidance, consent encouragement and every specific and personal concern, throughout the course of investigation and successful completion of his work.

I take this valuable time to express my sincere thanks to **Dr. J. Nandha kumar M.Pharm., Ph.D.,** Professor, Department of Pharmacy Practice, The Erode College of Pharmacy and Research Institute, Erode, for his providing his valuable time and advices.

I express My Heart Full Gratitude and thanks to **Medical Superintendent and All Staff in Government District Head Quarters Hospital, Tiruppur.** Who had taken the pain to provide me with all the facilities for the completion of My Project Work.

I am elated to place on profound sense of gratitude to Prof. **Dr. V. Ganesan M.Pharm., Ph.D.,** Principal, The Erode College of Pharmacy and Research Institute, Erode. For his support and encouragement.

I express my special thanks to **Mr.S.Rajarajan M.Pharm,Asst professor, Dr. Suthanth.T Pharm.D.,** and **Dr. S. Balamurugan Pharm.D.,** Lecturer, The Erode College of Pharmacy and Research Institute, Erode, for his valuable suggestion and to impart me the gain of his knowledge.

I am hearty thankful for **Mr.P.Royal Frank M.Pharm.,** Assistant Professor, Department of Pharmacology, for his valuable suggestions.

I cannot express what I owe to My Father, Mother, Brother and My Loving Sister always being with me, for making me what I am and for all the love and care they provided me without which I would not have been in a position to write this page.

I take this opportunity to express my gratitude to all those who have helped me directly or indirectly in the successful completion of my dissertation. Although words can't express my gratitude, I am trying to acknowledge their valuable contribution.

Above all I dedicate myself before the unfailing presence of ALLAH and consent love and encouragement given to me by beloved father, mother, sister and brother who deserve the credit of success in whatever work I did.

Place: Erode. Reg.no:261540401

Date:

INDEX

TABLES

FIGURES

ABBREVIATIONS

NSAID	NON-STEROIDAL ANTI-INFLAMMATORY DRUGS
CNS	CENTRAL NERVOUS SYSTEM
IV	INTRAVENOUS
IM	INTRAMUSCULAR
SC	SUB CUTANEOUS
LD	LETHAL DOSE
ANTU	ALPHA-NAPTHYL THIOUREA
DDT	DICHLORODIPHENYLTRICHLOROETHANE
CO	CARBON MONOXIDE
MAO	MONO AMINO-OXIDASE
LSD	LYSERGIC ACID DIETHYLAMIDE
ET	ENDOTRACHEAL
CN	CYANIDE
CHF	CORONARY HEART FAILURE
CAVH	ARTERIOVENOUS
CVVH	VENOVENOUS
ABG	ARTERIAL BLOOD GAS
OPC	ORGANOPHOSPHOROUS COMPOUND
NAC	N-ACETYLCYSTEINE
ATP-ASE	ADENOSINE TRIPHOSPHATASE
MRD	MEDICAL RECORD DEPARTMENT

INTRODUCTION

Poison is a substance that causes damage or injury to the body and endangers one's life due to its exposure by means of Ingestion, Inhalation or Contact. Poisonings and Snake Bites constitute a Major Cause of Hospitalization and Mortality in developed as well as developing nations.

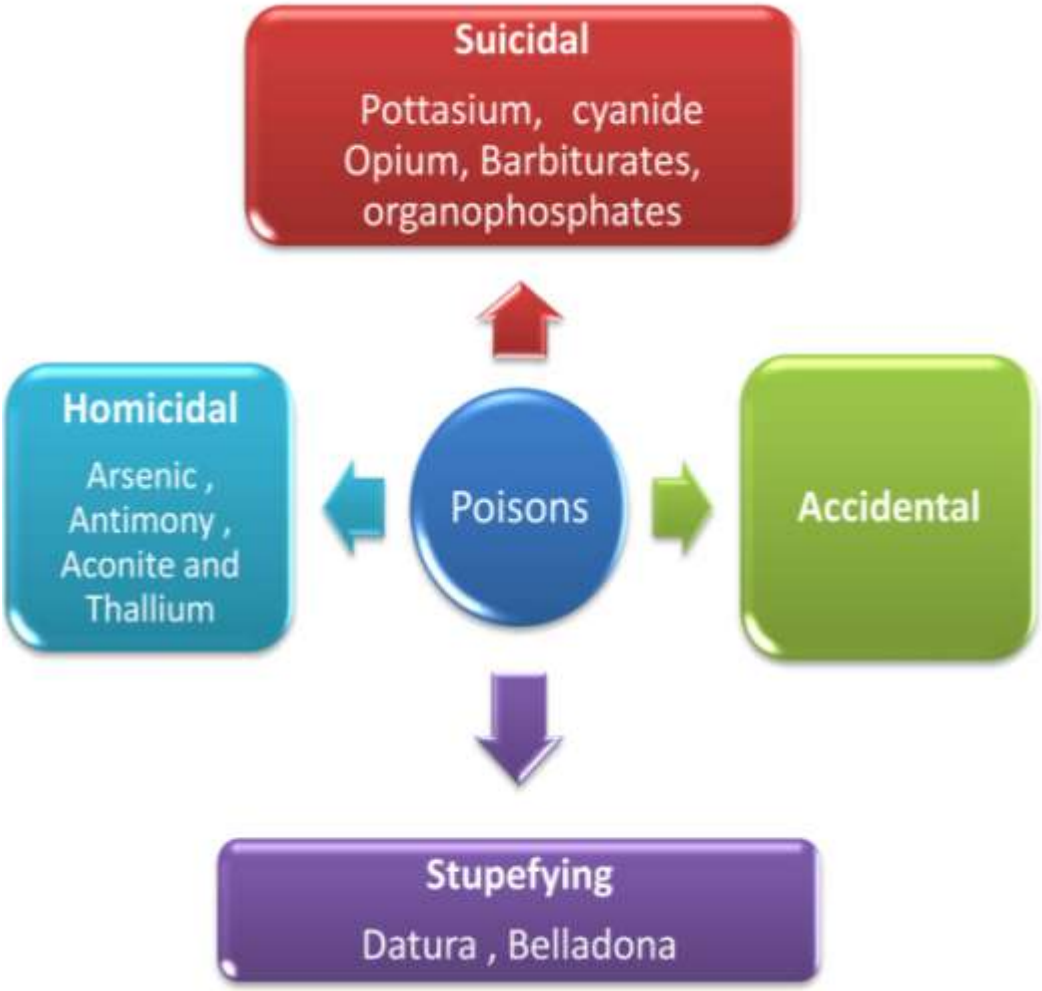


Fig: 1 Poisons are classified according to the Dose Related Adverse Effects [1].

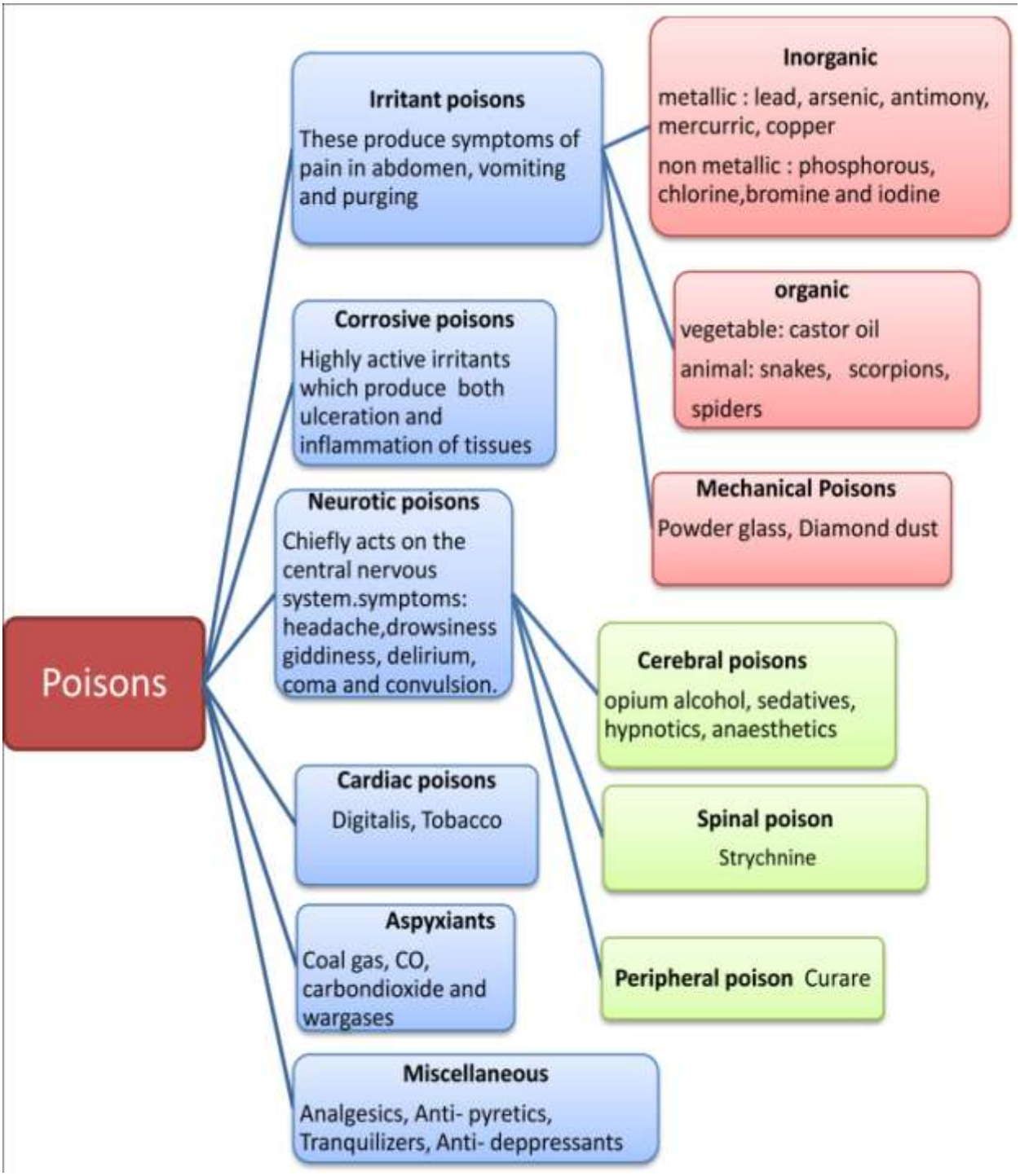


Fig: 2 Poisons are classified according to Mode of Action [2]

Table: 1 Poisons are classified according to Source

➢ DRUGS	**AMOEBICIDES** Carbarosone, Pentamidine, Ronidazole, Emetine..etc **ANAESTHETICS** Ketamine, Benzocaine, Lidocaine, Enflurane etc. **ANTICONVULSANT** Beclamide, Carbamazepine, Lacosamide, Valproic acid etc. **ANTIBIOTICS** All preparations and their salts, Avopracin, Ertapenam sodium, Teicoplannin, Telithromycin etc **ANTI- LEPROTICS** Clofazimine, Dapsone, Thiambutasine etc. **NSAID'S** Alclofenac, Azapropazone, Butorphanol, Celecoxib, Diclofenac, Etodolac, Flufenamic acid, Ibuprofen, Indomethacine, Nalbufine etc. **ANTI –ASTHMATICS** Aminophyline, Etophyline, Doxophyline, Formoterol, Salbutamol etc. **ANTI –CHOLINERGIC** Atropine, Belladona, Benzhexol, Benztropine, Cyclopentolate, Dicyclomine, Ethopropazine, Fenpipramide, Fesoteradine, Glimepiride, Glycopyrrolate, Homatroine, Ipratropium etc. **ANTI-DEPRESSANTS** Agomelatine, Bupropion, Citalopram, Deanol. Escitalopram, Fluoxetine, Hydrazines, Phenoxyethyl,

	ANTI-DIABETICS Actohexamide, Carbutamide, Chlorpropamide, Dapagliflozin, Exenatide, Gliclazide, Glimepiride, Lixisenatide, Metformin, Miglitol, Rapaglinidc etc **ANTIHISTAMINES** Antazoline, Bilastine, Cetrizine, Doxylamine, Emedastine, Loratadine etc. **ANTI-HYPERTENSIVES** Alseroxylon, Aliskiren, Azilsartan, Benazepril, etc.
➢ METALS	Aluminium, Arsenic, Beryllium,Cadmium Copper, Iron, Lead, Lithium, Manganese, Mercury, Silver, Thallium, Tin, Zinc..etc
➢ FOOD MATERIALS	Dairy products such as cheese, raw meat, chicken, Contaminated foods, bread, coconut milk, containing foods.
➢ BITES/STINGS	Bees, Spider, Snake and Scorpion
➢ HOUSEHOLD PRODUCTS	Acids, alkalis, camphor, carbon monoxide, bleach, drain cleaner, rug cleaner, wallpaper cleaner, Glass, laundry ink, disc batteries, moth balls, cosmetics, essential oils, detergents.

➢ ARAGES	Kerosene/paraffin, fire lighters, fire starting tablets,fire extinguishers, paints, painting supplies, Slug, weed killers, pellets, petrol, arsenic, lead, swimming pool chemicals, antifreeze, fumigants ,acids, alkalis, camphor, carbon monoxide, bleach, drain cleaner, rug cleaner, wallpaper cleaner, laundry ink, disc, batteries, moth balls, cosmetics, essential oils, detergents, car cleaning products, hobby chemicals
➢ INSECTICIDES	Pyrethroids, organophosphorus, carbamates, Organochlorine, manganese compounds.
➢ RODENTICIDES	Warfarines, indanodiones
➢ INSECT REPELLANTS	Diethyltoluamide.
➢ HERBICIDES	Bipyridyls, Chlorophenoxy glyphosate, Acetanilides, Triazines
➢ FUNGICIDES	Thiocarbamates, Dithiocarbamates, Cupric salts, Tiabendazoles, Triazoles, Dicarboximides, Dinitrophenoles, Organotin compounds, Miscellaneous.

➢ FUMIGANTS	Aluminium & Zinc phosphides, Methyl bromide, Ethylene dibromide.
➢ PESTICIDES	Aldrin, Dieldrin, Chlordane, DDT, Endrin, Heptachlor Mirex,Toxaphene
➢ PLANTS	Oleander, Poison ivy, Mushrooms, Thorn apple, Datura, Belladona,
➢ INDUSTRIAL& LABORATORY POISONS	Acetic anhydride, N-acetyl anthranilic acid , chloroform, Acetyl bromide, Acetyl chloride, Ammonia, Anthranilic acid, Formaldehyde, Hydrochloric acid, nitric acid[3].

Mechanism of action of poisons:

1. **Local action**:

Poisons act directly on the Tissues and Cause Corrosion, Irritation and Inflammation.

2. **Remote action**:

As the Poison gets absorbed systemically. It produces both Specific CNS, Spinal Cord, Cardiac and Non Specific Shock.

Factors modifying the action of poisons

1. **Dose**:

Small dose usually produce no toxic effects whereas large doses produce toxic effects on the body. Some individuals also exhibit phenomena like idiosyncrasy, allergy and synergism. The presentations are different with single or chronic exposure and with frequency of exposure.

2. **Form of poison**

(a) Physical state:

Gases and vapours act more quickly than fluid Poisons because they are absorbed immediately. Fluid Poisons act faster than solid ones.

(b) Chemical combination:

Some substances in certain combination become Inert like Nitric Acid and Hydrochloric Acid, and certain other combinations become Poisonous like lead Carbonate and Copper Sulphide.

(c) Mechanical combination:

The Action of a Poison is considerably altered when combined mechanically with inert substances.

3. **Method of administration**

A poison acts most rapidly when inhaled in gaseous or vapours form or when injected I.V followed by I.M. /S.C. and least rapidly when swallowed.

4. Condition of **the body**

(a) **Age:**

Children are more susceptible than adults to toxins. In old age poisons have greater effects.

(b) Sleep and intoxication:

The body functions are lowest during sleep, so the poisons are absorbed slowly during sleep[4].

COW DUNG POISON

Auramine-O is a diarylmethane dye used as a flourescent stain. In its pure form, auramine-O appears as yellow needle crystals. It is very soluble in water and soluble in ethanol although the requirement for metabolic activation regarding, Auramine is unresolved. It is considered to be a Pro-Carcinogen.

Cow Dung Poison is commonly known as Sani Powder Poison in local Tamil Language in South India it is a Lethal Poisonous Synthetic Chemical. Cow Dung was traditionally used as a germicide and insect repellent to clean homes, courtyards and temples in the state of Tamil Nadu, since the unavaibility of Cow Dung Synthetic Chemicals are used to prepare this Sani Powder. Sani Powder Poison is available in two Colours that are yellow and green.

Sani Powder is available in two varieties

1. Yellow Powder: Auramine-O chemically known as diarylmethane dye
2. Green Powder: malachite green, chemical constituent being Triphenyl methane.

In rural Tamil Nadu (South India), especially in the districts of Coimbatore, Erode and Tiruppur. Cow Dung Powder is commonly used as a Suicidal Poison. There is no specific antidote for these dyes. It is very toxic due to which death occurs within hours of ingestion. Many deaths have been reported due to this Cow Dung Poison [5].

The lethal effect of this agent is most commonly accomplished by the liver the reason behind preponderance liver toxicity is established by fact that within several hours of ingestion of the total ingested dose is concentrated in the liver. Over half the deaths due to cow dung toxicity occur in the first day following which liver damage, hepatic fulminent hepatic failure.

In such a case a huge population in our country is left with no choice other than to suffer death. The morbidity and mortality following toxic exposures of sani Powder poison continues to rise[6].

Fig: 3 Cow Dung Powder in Solid & Liquid form.

RODENTICIDES

Rodenticides are a heterogeneous group of substances that exhibit markedly different toxicities to humans and rodents. They are among the most toxic substances regularly found in homes. The varieties of Rodenticides used over the years [7].

Classification of Rodenticides based on toxicity

- **Highly toxic Rodenticide**

  Highly toxic Rodenticide are those substances with a single dose LD_{50} of less than 50mg/kg body weight. Some of these compounds have largely been abandoned because of serious human toxicity. This group includes:

  1. Aluminium Phosphide
  2. Sodium Monofluoroacetate
  3. Strychnine
  4. Zinc Phosphide
  5. Yellow phosphorous
  6. Arsenic
  7. Thallium.

- **Moderately toxic Rodenticides**

Among the moderately toxic Rodenticides, those with LD_{50} of more than 500mg/kg

Body weigth are

1. Alpha-napthyl-thiourea (ANTU) and
2. Dichlorodiphenyltrichloroethane (DDT).

Patients who ingest large quantities of ANTU may develop Dyspnea, Rales and Cyanosis (secondary to pulmonary edema), and hypothermia. Poisoning from Exposure to DDT can result in symptoms such as vomiting, tremors, and Convulsion. How much exposure is required to cause severe illness or even death Is, however, not certain[8].

- **Low toxicity Rodenticides**

Low toxicity Rodenticides are those with LD_{50} between 500 and 5000mg/kg body weight and include:

1. Red squill
2. Norbimide
3. Anticoagulants Warfarin-type Rodenticides[9].

Fig: 4 Rodenticide.

SNAKE BITE

A Snake will sometimes bite in self defense if disturbed or provoked. Some Snakes are Venomous & can Inject Venom (Toxin) as they Bite. A Bite from a Venomous Snake is a Medical Emergency as they can be dead if not treated quickly.

SYMPTOMS OF SNAKE BITES

- ✓ Pain, Redness & Swelling in the area of the Bite.
- ✓ Nausea (feeling sick) & Vomiting.
- ✓ Dizziness & Fainting.
- ✓ Blistering & Eventually, Gangrene in the area of the Bite.
- ✓ Shock.
- ✓ Muscle Paralysis (An inability to move one or more muscles of the body) leading to Difficulties, Swallowing & Breathing.
- ✓ Bleeding.
- ✓ Swelling of the Lips, Gums & Tongue.
- ✓ Irregular Heart Beat[10].

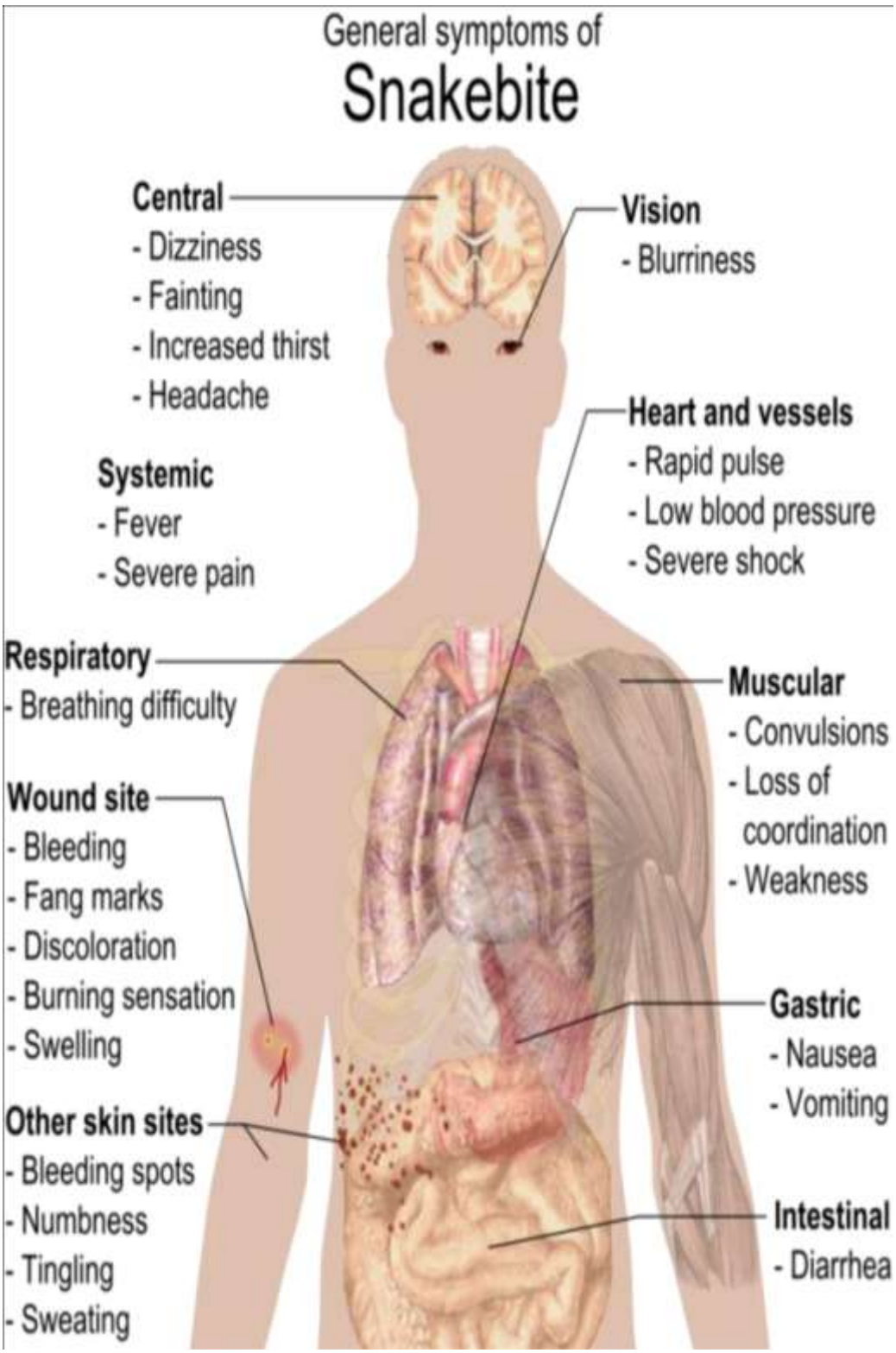


Fig: 5 Symptoms of Snake Bite

Fig: 6 poisonous snakes in india

KING KOBRA

INDIAN COBRA

INDIAN KRAIT

MALABAR PIT VIPER

RUSSELL'S VIPER

SAW SCALED VIPER

MANAGEMENT OF SNAKE BITE

- First aid treatment.
- Transport to hospital.
- Rapid clinical assessment and Resuscitation.
- Detailed clinical assessment and species diagnosis.
- Investigations/laboratory tests.
- Anti venom treatment.
- Observing the response to anti venom.
- Deciding whether further dose(s) of anti-venom are needed.
- Supportive/ancillary treatment.
- Treatment of the bitten part.
- Rehabilitation.
- Treatment of chronic complications [11].

OLEANDER POISONING

Oleandrin is a Toxic Cardiac Glycoside found in Oleander (*nerium oleander L.*) along with neandrin it is primarily responsible for the sap of Oleander. Oleandrin has been used many years in China and Russia for its properties as a Cardiac Glycoside, for both suicidal and therapeutic purposes as in treatment of Cardiac Insufficiency [12].

Available forms:

Oleandrin is apart from its pure form, also closely related to structural similar glycoside, which all have more or less the same characteristics as Oleandrin:

- Oleandrigenin is a Deglycosylated metabolite of Oleandrin. It has however a more mild effect.
- Neandrin
- Neritaloside
- Odorside[13]

Mechanism of Oleandrin:

Because of its properties as a cardiac glycoside, Oleandrin interferes in some essential processes within the cell, the most important of these being the inhibition of the Na-K ATPase. This protein enables the cell to exchange the Cations Na+ and K+ between the intercellular and extracellular spaces by which, for instance, electronic signaling is made possible in nerve cells. Oleandrin binds to specific amino acids in the protein, causing it to lose its function. After depolarization of the cell in which Na+ flows into the cell, the Na+ cannot be transported back into the extracellular membrane, causing the sodium gradient to disappear. This gradient is the driving force for other transport proteins, such as the sodium-calcium exchanger, which plays an important role in Cardiomyocytes[14].

To make muscle contraction possible, a calcium influx from the extracellular fluid into the cell is crucial. After the muscle contraction, the calcium is normally pumped out of the cell and exchanged for sodium. When the sodium gradient is depleted, calcium cannot be pumped back and, as consequences, accumulates in the Cardiomyocytes. [15].

As a result of the high Calcium Concentration, actin and myosin filaments will bind stronger, unable to relax properly to make a new contraction possible. This may result in cardiac arrhythmias, in the worst case decreasing cardiac output and causing a shortage in oxygen supply in vital tissues. Apart from being a potent toxic compound, it may also be used in the therapeutic ways. Both Oleandrin and Oleandrigenin, as well as their relatives, may be

able to inhibit proliferation of tumor cells and stimulate their apoptosis as a result of the high concentration of intracellular calcium. In addition, it inhibits excretion of fibroblast growth factor 2 through membrane interaction and through inhibition of the Na,K-ATPase pump. However, there are no results from clinical testing on humans that support any used as a cancer treatment.

Toxicity of Oleandrin:

Oleandrin has been reported to be Lethal, but exact dosages are not fully documented. The fatal blood concentration of Oleandrin has been estimated for humans to be approximately 20ng/ml in decreased blood by extrapolation of intoxication symptoms. In practice, there have been adult cases where in 14-20 Oleander leaves (of unknown Oleandrin concentration) proved not to be fatal, but also a lethal case of a child that consumed only one leaf[16].

Fig: 7 Oleander Seeds

ANT CHALK

Ant Chalk also known as Chinese Chalk or 'Miraculous Insecticide Chalk' is an Insecticide in the form of normal looking chalk. It contains the pesticides Deltamethrin and Cypermethrin [17].

While the active ingredients are legal in the United States, the chalk is not legal there. Labeling often claims the chalk is "harmless to human beings and animals" and "safe to use." Chalks have been found to cause serious health problems and deaths [18]. Packaging, often containing lead- based inks, generally does not list ingredients. Despite its Illegal Status, "Chinese chalk" is illegally imported from China and sold in corner stores in the United States [19].

Deltamethrin

Deltametharin is a Pyrethroid Ester Insecticide. It is sold as "Deltagard" in Canada.

Since Deltamethrin is a Neurotoxin, it temporarily attacks the Nervous System of any animal with which it comes into contract. Skin Contact can lead to Tingling or Reddening of the Skin Local to the application. If taken in through the Eyes or Mouth, a common symptom is facial Paraesthesia, which can feel like many different Abnormal Sensations, Including Burning, partial numbness, "pins and needles", skin crawling , etc. there are no reports indicating that Chronic intoxication from Pyrethroid Insecticides causes motor neuron damage or motor neuron disease[20].

Recently, in South Africa, Residues of Deltamethrin were found in Breast Milk, together with DDT, in an area that used DDT treatment for malaria control, as well as Pyrethroid in Small-Scale agriculture [21].

There are No Antidotes and Treatment must be Symptomatic, as approved by a Physician. Over time, Deltamethrin is metabolized, with a Rapid Loss of Toxicity, and passed from the

body. A Poison Control center should be contacted in the event of an Accidental Poisoning [22].

Cypermethrin

Cypermethrin is a Synthetic Pyrethriod used as an Insecticide in large scale commercial agricultural applications as well as in consumer products for domestic purposes. It behaves as a fast-acting neurotoxin in insects. It is easily degraded on soil and plants but can be effective for weeks when applied to indoor inert surfaces. Exposures to sunlight, water and oxygen will accelerate its decomposition. Cypermethrin is highly toxic to fish, bees and aquatic insects, according to the national pesticides telecommunication network (NPTN). It is found in many Household Ant and Cockroach Killers including raid and Ant Chalk [23].

Cypermethrin is moderately toxic through Skin Contact or Ingestion. It may cause Irritation to the Skin and Eyes. Symptoms of Dermal Exposure include Numbness, Tingling, Itching, Burning Sensation, Loss of Bladder Control, in coordination, seizures and possible Death. Pyrethroids may adversely affect the central nervous system. Human volunteers given dermal doses of 130ug/cm^2 on the earlobe experienced local tingling and burning sensations. One man died after eating meal cooked oil. Shortly after the meal, the victim experienced nausea, prolonged vomiting, stomach pains, and diarrhea which progressed to convulsions, unconsciousness and coma. Other family members exhibited milder symptoms and survived after hospital treatment. Cypermethrin is not a skin or eye irritant, but it may cause allergic skin reactions. Excessive exposure can cause nausea, headache, muscle weakness, salivation, shortness of breath and seizures. In humans, Cypermethrin is deactivated by enzymatic hydrolysis to several carboxylic acid metabolites, which are eliminated in the urine. Worker exposure to the chemical can be monitored by measurement of the urinary metabolites, while severe over dosage may be confirmed by Quantitation of Cypermethrin in blood or Plasma [24].

Fig: 8 Ant chalk.

Diagnosis of poisoning

The diagnosis in a case of poisoning can be made from the

1) History

2) Physical Examination

3) Laboratory Evaluation

4) Toxicological Screening

1. History

- Most important indicator of toxic ingestion. Careful history regarding involved toxins, amount of drug and timing should be recorded.
- Information regarding prescription medication, over the counter drugs and illicit substances of abuse should be obtained.
- Friends, relatives and other involved health care providers should be questioned and medications identified.
- Medication found on or near the patient should be examined and pharmacy on the Medication label should be called to determine the status of all prescription Medication [25].

2. Physical Examination

- Evaluation of airway patency, Respiration, Circulation.
- Rapid assessment of mental status, temperature, pupil size, muscle tone, reflexes, skin and peristaltic activity.
- Separate patients into two groups
    1) Depressed Status
    2) Agitated Status

Drugs causing the **depressed** status are Sympatholytic like Adrenergic Blockers, Anti-Arrhythmias, Anti-Hypertensive, Anti-Psychotics, Cyclic Antidepressant, Cholinergics like Nicotine, Carbamates, Organophosphates, Physostigmine, Pilocarpine, Sedative-Hypnotics like Alcohols, Barbiturates, Benzodiazepines; Narcotics like Analgesics, Anti-Diarrheal Agents; and others like CO, Cyanide, Hypoglycemic Agents, Lithium and Salicylates[26].

Drugs causing an **agitated status** are Sympathomimetics like Adrenergic Agonists, Amphetamines, Caffeine, Cocaine, Ergot Alkaloids, MAO inhibitors, Theophylline, Anticholinergics like Anti-Histamines, Anti-Parkinsonism drugs, Anti-Psychotics, Anti-Spasmodics, Cyclic Antidepressant, Cyclobenzapine; Drug withdrawal like a blockers, Clonidine, Ethanol, Opioids, Sedatives-Hypnotics, Hallucinogens like LSD, Marijuana.

• Pupillary Size and Reaction to light, with the Patient's Physiological Status gives a Rapid Clue regarding Dominant Ingestion.

Pinpoint pupils with	-	Phencyclidine agitation intoxication
Pinpoint pupils with Lethargy	-	Narcotic overdose
Dilated pupils reacting	-	Cocaine intoxication to light
Dilated pupils not reacting	-	Anti-cholinergic to light intoxication [27]

Labororatory Evaluation

Clinical laboratory data include assessment of the three gaps of toxicology

1. The Anion gap
2. The osmolal gap
3. The arterial oxygen saturation gap.

Unexplained widening of the difference between calculated and measured determination of these values raises the suspicion of toxic ingestion [28].

1. **Anion Gap:** refers to the difference between measured Cations and measure Anions

AG = $[Na^{+}]$ - [Cf] - [HC03"]

Normal Value = 12 ± 4 $^{meqL-}$

The presence of anion gap indicates that there are more unmeasured anions than cations, since total serum cations equals total serum anions. Unmeasured cations include K^{+}, Mg^{++} and Ca^{++} totaling about 11 $^{meqL-}$ under normal conditions, and the concentration of unmeasured anions including protein (mainly albumin), sulfates, phosphates and organic acids is about 23 $^{meqL-1}$.

The anion gap falls by 2.3 $^{meqL-1}$ for every 1 gmL-1 decrease in plasma albumin concentration[29].

2. Osmolal gap: (measured - calculated osmolality)

Certain drugs and toxins of low molecular weight produce a discrepancy between measured and calculated plasma Osmolality, commonly referred to as the Osmolal gap. (Osmolal gap equals measured minus calculated Osmolarity).

Normal Plasma Osmolality=285-295 mosmlL-1. and is calculated as:

Calculated Osmolality = 2[Na^+] + [BUN]/2.8 + [Glucose]/18 + [ethanol] /4.6

Where Na+ (in mmolL-1) is multiplied by 2 to account for anions (Cl- and HCO-and BUN and glucose are divided by 2.8 and 18 to convertmgdL-1 to mmolL-1 and ethanol is divided by 4.6[30].

3. Oxygen saturation gap: Toxins associated with an elevated arterial oxygen saturation gap [>5% difference between saturation calculated from ABG determination and saturation measured by co-oximetery] including carbon monoxide and methemoglobin.

These toxins interfere with oxygen binding to hemoglobin and thereby significantly decrease oxygen content without lowering PaO_2. It is important to note that Oxygen saturation measured by pulse Oximetry is also falsely high in the setting of these toxins [31].

H_2S and Cyanide interfere with cellular utilization of oxygen leading to an abnormally high venous Oxygen saturation or "Arterializations of venous blood". Obtain serum electrolytes, BUN, Blood glucose and Serum Osmolality.

- Calculate anion and Osmolal gaps.
- Obtain ECG; look for widened QRS, and QT intervals and AV block.
- Obtain CXR to look for pulmonary edema or inflates.
- Obtain Abdominal X-Ray to look for radio opaque pills.
- Obtain urine for toxicological screening and routine analysis. Look for calcium oxalate crystals.
- Pregnancy test in women of child bearing age [32].

4. Toxicological Screening

It provides direct evidence of ingestions, but it rarely impacts initial management and initial supportive measures should never await results of such analysis. It is used to

- provides ground for treatment with specific antidote or method for enhancing drug elimination and
- Also identifies drugs that should be quantified to guide subsequent management. Also look for characteristic signs of various kinds of poisoning while immediate treatment measures are being started [33].

Management of Poisoning

Treatments goals include support of vital signs, prevention of further poison absorption, enhancement of poison elimination administration of specific antidotes and prevention of re exposure. Majority of poisoned patient require only supportive treatment [34].

Initial Therapy

Immediate management of life threatening conditions in victims of poisoning with coma, seizures or marked airway obstruction should be as follows.

A. Keep Airway open: Establish and maintain an adequate airway and ventilation.

• Begin supplemental oxygen 5-10, Its by nasal prongs or mask.

• If the patient has no gag reflex, Intubate the Trachea with a cuffed Endo tracheal tube as soon as possible to

a) protect the airway

b) facilitate oxygenation and ventilation

c) Helps removal of secretions [35].

Indications for Endo Tracheal Intubation

1. Patients in coma or with markedly depressed gag reflex.

2. Awake patient with normal gag reflex.

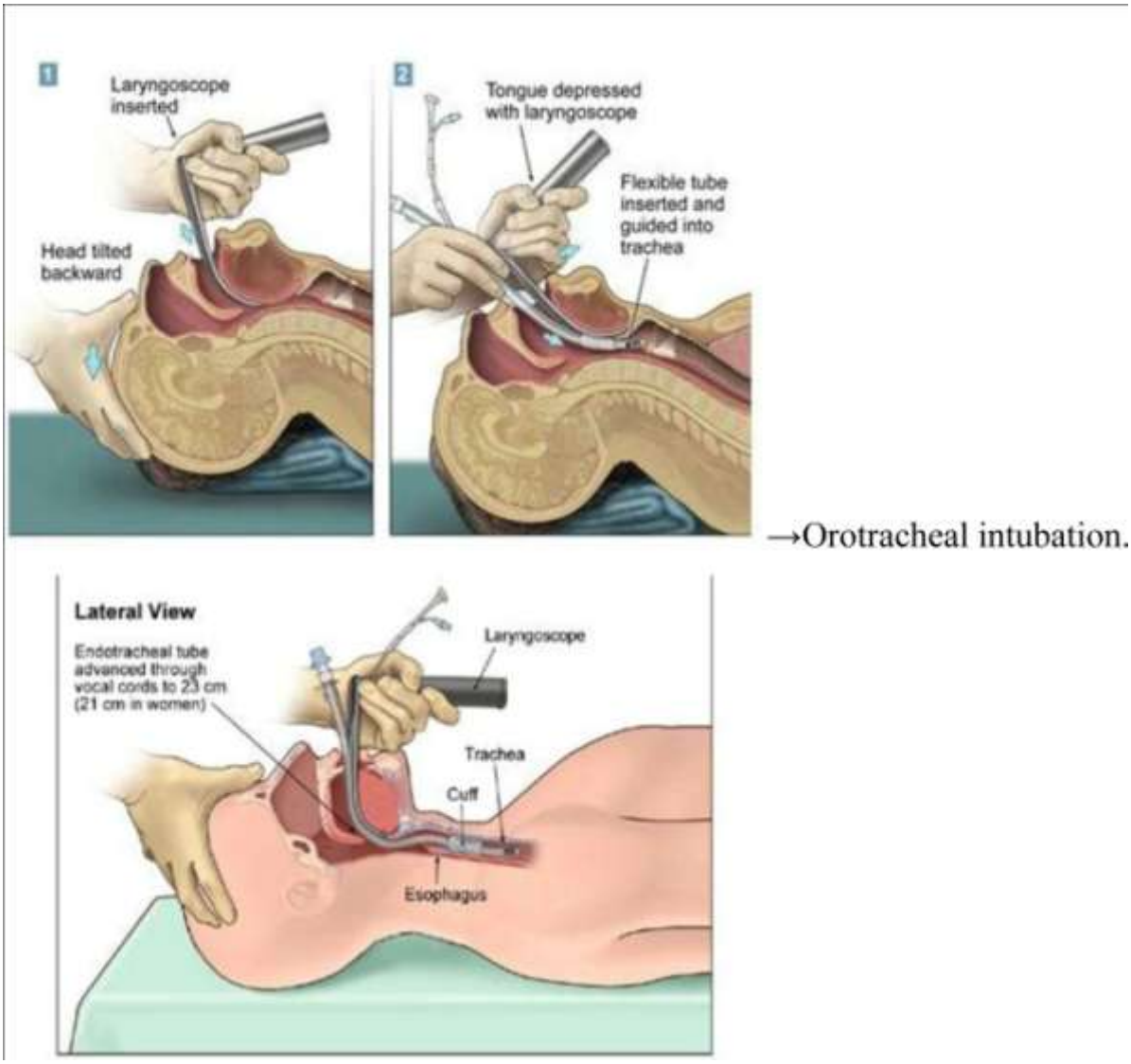
3. Lethargic patient with fluctuating mental status and a variable gag reflex [36].

Choice of Intubation Technique.

a) Orotracheal intubation.

b) Nasotracheal intubation.

a) Orotracheal intubation - This technique is useful for the comatose patients, since it is rapid and the location of E.T. tube can be verified by direct vision.



→Orotracheal intubation.

Fig: 9 Orotracheal Intubation

2. Give Naloxone, 0.4 - 2 mg I.V. If patients response is weak or if narcotic overdose is suspected, give repeated doses of 2 mg every 1-2 min up to a total of 10-20 mg. Patients responding to Naloxone must be observed for at least 3 hours after the last dose of Naloxone.

3. If Alcoholism or Malnutrition is suspected, give Thiamine, 100mg I.V/I.M. The level of consciousness of all intoxicated patients should be assessed and the time of assessment should be recorded.

4. Give Flumazenil, 0.2-0.5 mg. I.V. repeated every 30 sec. up to 3 mg. in case of suspected benzodiazepine poisoning.

E. **Maintain Circulation:** Restore the intravascular volume by intravenous infusion of crystalloids. If the administration of more than 20-30 mlKg-1 of crystalloid solution and usual doses of dopamine (5-15 mgkg-lmin-1) fail to restore blood pressure, insert a pulmonary artery catheter to obtain pressure reading and help guide further with fluids and pressure agents.

F. Treat Seizures: Give Diazepam 0.1 to 0.2 mg/Kg, lV over 1-2 minutes. Followed by Phenobarbital l5 mg/kg, IV if there is no response to Diazepam.

G. Start ECG monitoring-Obtain 12 lead ECG note the rate of rhythm, presence of arrhythmias and PR, QRS and QT intervals.

H. Perform Gastric lavage: Collect sample for future toxicological analysis if required.

• **Nasotracheal intubation.** - This technique is useful in the agitated patient. It doesn't require jaw relaxation and has the advantage of not requiring neck manipulation in a patient who may have cervical spine injury.

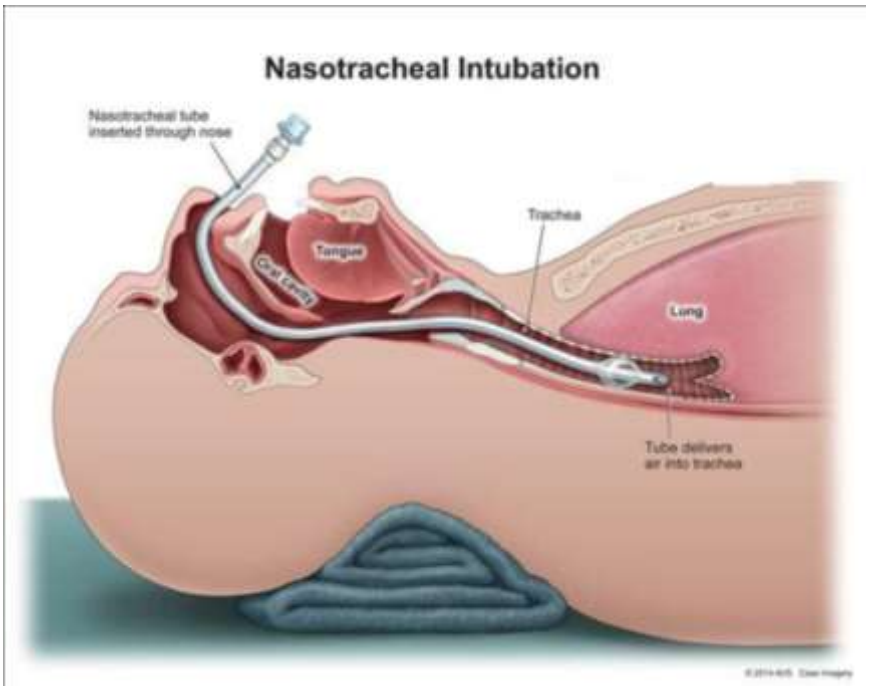


Fig: 10 Nasotracheal intubation

B. **Obtain Arterial Blood Gas measurements:** To determine adequacy of ventilation and perfusion.

C. Gain Intravenous Access: Insert a 18G peripheral or central line and draw blood for complete blood count, serum electrolyte and blood glucose measurements and tests of hepatic and renal function [37].

D. **Treat Coma** (COMA COCKTAIL)

1. Give glucose, 50ml of a 50% solution (25g glucose) IV over 3-4 minutes.

I. Search for associated Illness:

Look for cause of coma or seizures look for

a) Head trauma

b) Hemorrhage or shock

c) Infection

d) Metabolic disorders

e) Hypothermia

f) Hyperthermia[38]

J. Document: The available history and the level of coma before ambulance personnel, relatives etc. leave the hospital [39].

♦ FUNDAMENTALS OF POISONING MANAGEMENT

I. SUPPORTIVE CARE

- Airway protection.
- Treatment of arrhythmia.
- Oxygenation / Ventilation-
- Hemodynamic support.
- Treatment of arrhythmia.
- Correction of metabolic derangements.
- Prevention of secondary complications.

2. PREVENTION OF FURTHER POISON ABSORPTION

- Gastrointestinal decontamination.
- Decontamination of other sites.
- Induced emesis, Gastric lavage.
- Eye decontamination.
- Activated charcoal.
- Skin decontamination.
- Whole bowel irrigation.
- Body cavity decontamination.
- Catharsis.
- Dilution.
- Endoscopic/surgical removal.

3. ENHANCEMENT OF POISON ELIMINATION

- Multi dose Activated Charcoal.
- Extracorporeal removal**.**
- Forced Diuresis.
- Peritoneal dialysis.
- Alteration of P^R
- Hemodialysis.
- Chelation.
- Hemoperfusion /Hemofiltration.
- Exchange transfusion.
- Hyperbaric oxygenation.

4. ADMINISTRATION OF ANTI-DOTES

- Neutralization by antibodies
- Metabolic antagonism
- Neutralization by chemical binding
- Physiologic antagonism

5. PREVENTION OF RE-EXPOSURE

- Adult education.
- Notification of regulatory agencies.
- Child proofing.
- Psychiatric referral.
- Prevention of further drug absorption.

Inhaled Poisons

- Remove the patient from the source of the poison. Give oxygen by mask (CN poisoning).
- Inhalation of Water Aerosol may Dilute Inhaled Irritant in the Nasopharynx.
- Check for hoarseness and singed Nasal Hairs.
- Be Alert for Delayed Development of Upper Airway Obstruction and Pulmonary Edema[40].

Contaminated Eyes

- Wash the Eyes with the copious amounts of plain water or (hang a bottle of 500-1000 ml of NS) above the patient and dribble the solution slowly into the corner of the eye through the I.V tubing.
- Check the tears with P_H paper after the eyes have been washed to make sure that all toxic material has been removed.
- A careful eye examination is done following irrigation.

Contaminated skin

- Wash the skin immediately with plenty of water and dilute soap solution.
- Discard contaminated clothes in a marked plastic bag. Organophosphate compounds are well absorbed through the skin and are difficult to remove [41].

Ingested Poisons

A. Emesis is recommended for emergency treatment of drugs not adsorbed by activated charcoal. It is induced with syrup of Ipecac. Dose is 15ml orally for children (5-10 ml for 6m-lyr) and 30ml for adults.

Give2-3 glasses of plain water following this. This induces the vomiting within 20-30 min. and can be repeated after 30 min. The vomits should be inspected for remnants of Pills or Toxic substances, its appearance and Odour should be noted. Apo Morphine is a Parentral emetic, and can be used with caution because of its CNS and Cardiac side effects. Do not induce emesis in comatose patients.

Contraindications to the induction of emesis are

- Caustic (alkali) or corrosive (acid) ingestion.
- Agents that rapidly produce coma or convulsions in less than 30 min. and may predispose to aspiration during emesis.
- Prior significant vomiting.
- In infants less than 6 months of age.
- In foreign bodies.
- Absence of bowel sounds.

B. Gastric Lavage: It is done a) after suspected serious ingestion when attempt to induce emesis fail and when patients are uncooperative or lethargic, or when gag reflex is markedly depressed.

Place the patient in left lateral deceits position with head down. It is performed with a large bore Nasogastric Tube. Use tap water or saline at body temperature in 250ml increments and continue lavage until fluid returns clear. Airway must be protected if gag reflex is depressed.

C. Activated Charcoal: It has powerful adsorption capacity and is given orally or viagastric tube. It irreversibly binds the drugs within the bowel and reduces the blood concentration by reducing drug absorption and by creating a negative diffusion gradient between the gut lumen and blood (gastrointestinal dialysis).The dose of activated charcoal is 1gmKg-1 orally with a

minimum of 15 gm. The usual dose is 60-70 gm. It can be mixed with 4 parts of water and given every 4 hourly as long the bowel sounds are present. It is used for most poisons accept alcohol, potassium, Fe and Li[42].

Contraindication to the use of activated charcoal

a. It should not be given before, concomitantly or just after ipecac because it may absorb the ipecac and interfere with its emetic properties.
b. It should not be given before, concomitantly or just after oral antidotes unless proved not to interfere significantly with their absorption.
c. It does not effectively adsorb caustic and corrosive and may produce vomiting or stick to the mucosa of oesophagus or stomach and may look like burn on endoscopy.
d. It should not be given if no bowel sounds are present.
e. Activated charcoal is a stool marker, indicating that the toxin has passed through the GIT and no further significant absorption from the original ingestion will occur.

D. Catharsis: Cathartic salts (disodium phosphate, magnesium citrate and sulphate, sodium sulphate) or saccharide (mannitol, sorbitol), promote the rectal evacuation of gastrointestinal contents. The dose of sorbitol is 1-2 gkg-1. Their aim is to prevent constipation following charcoal administration. They are contraindicated in corrosive poisonings and pre-existing diarrhoea.

E. Whole bowel irrigation: It is performed by administering a bowel cleansing solution containing electrolytes and polyethylene glycol orally or by gastric tube at a rate of 0.5 Lhr-1 in children and 2Lhr-l in adults until rectal effluent is clear. The patient must be in sitting

position. It is useful in patients who have ingested foreign bodies, packets of illicit drugs, slow releasing or enteric coated medicines or heavy metals. It is contraindicated in patients with bowel obstruction, ileus, hemodynamic instability, and compromised unprotected airway.

Enhancement of the absorbed drug

The decision to use measures to enhance drug elimination should be based on a rational understanding of drug properties and the clinical condition of the patient.

The various methods are:

- ✓ **Diuresis and P^H manipulations:** Since many toxins are weak acids or bases, they can be ionized in solutions of varying P^H. In the Ionized state, they are less likely to cross cell membranes and their re absorption by renal tubular epithelium is decreased.

(a) Weak acids such as Salicylate and Pheno Barbital are more fully ionized in basic solutions, so that alkalinizing the urine may serve to trap them in the tubular lumen, thus increasing excretion of the drug in the urine

(b) Weak bases such as amphetamines, strychnine and phencyclidine are more ionized in acid medium; acidification of urine has been proposed to enhance their removal. Contraindication to forced Diuresis includes severe CHF, Cerebral Failure and Pulmonary Edema [42].

1) Extra Corporeal Removal of Toxins:

(a) Hemodialysis: During hemodialysis, toxin is removed from the blood into a dialysate solution across a semi permeable membrane. The toxin must be relatively water soluble and not highly protein bound. It should have a small volume of distribution and slow rate of intrinsic elimination(a long t 1/2*).* It is effective in removing methanol, ethylene glycol, salicylates and lithium. It is also of value in correcting pH and electrolyte imbalances especially in anuric patients.

Criteria for potential dialyzalibility include

a) Water solubility

b) Low molecular weight

c) Protein binding

d) Volume of distribution and

e) Intrinsic clearance of the substance.

Complications of hemodialysis include intravenous access complications, hypo phosphatemia, alkalemia, dies equilibrium syndrome and hypotension.

(b) Peritoneal Dialysis: Peritoneal Dialysis is only 1/8-1/4 as efficient as hemodialysis and is not a preferred method.

c) Hemoperfusion: is defined as direct contact of blood with a sorbent system. In Hemoperfusion, blood is pumped through a column of adsorbent material (charcoal or resin) and returned to the patient's circulation. Vascular access similar to that for Hemodialysis is required. The kinetic conditions are the same as in Hemodialysis. It is commonly associated with thrombocytopenia. lt will not correct P^H or electrolyte imbalance.

d) Hemofiltration: Hemofiltration is potentially useful method for removal of substances with a large V_d slow inter compartmental transfer and vivid tissue transfer. Arterio venous (CAVH) or veno venous (CVVH) methods are used.

Antidotes

An antidote is any substance that increases the mean lethal dose of a toxin, or that can favorably affect the toxic effects of a poison

Table: 2 ANTIDOTES

POISONS	ANTIDOTES	DOSAGE REGIMEN
Anti-chollinergic agents	Physostigmine	2 mg iv over 5 minutes, continue agents with an infusion of 4-6 mg hourly (adult dose)
Anticholinesterase	Atropine	1-2 mg i.v.repeated 2-4mg every 5-10 min. or atropine drip.
Anti-coagulants (warfarin type)	Vitamin K	2-5 mg iv adult, 0.4 mg/kg child
Organophosphates	Atropine Pralidoxime	2 mg iv (IM or SC in less severely poisoned patients) followed by further two doses at 5-10 minutes interval until full atropinisation.
Benzodiazepines	Flumazenil	Initially 0.2 mg IV over 30 seconds. Further doses of 0.5 mg can be given over 30 seconds at 60 seconds interval to a total dose of 3 mg.

Narcotic analgesics	Naloxone	0.8-1.2 mgi.v. (children 0.2 mg)Repeat if respiratory depression not reversed within 1-2 mins.
Carbon monoxide	Oxygen (normobaric hyperbaric)	Administer as high as inspired or oxygen as possible until carboxyhaemoglobin concentration falls below 5% -hyperbaric oxygen in severe cases.
Methaemoglobin	Methylene blue	0.2 $mlkg^{-1}$ of 1% solution. Slowly I.V. over 5 minutes. Repeated as necessary upto 6 mgkg-1.
Acetaminophen	n-acetylcysteine	140 $mgKg^{-1}$ orally followed by 70 mgKg-1 every 4 hourly for 17 doses or 6 doses if no Hepatotoxicity.
Paracetamol	Acetylcysteine	300 $mgkg^{-1}$ over 16 hours
	Methionine	2.5 gm orally every 4 hours for 12 hours.

Cyanide	Dicobalt edentate Sodium nitrite Sodium Thiosulphate Oxygen	300 mg iv over 3 minutes 10 ml of 30% iv over 10 minutes. 50 ml of 25% solution in over 10 minutes in may be upto 4 gm I.V. Administer inspired oxygen till clinical recovery occurs.
Thallium	Berlin blue	250 $mgkg^{-1}$ per day in divided Doses till thallium level is < 10mgL-l in blood and urine.
Ethylene Glycol	Ethanol	Dose given should be sufficient to Maintain plasma ethanol levels at 1-2 gL^{-1}.
Methanol	Ethanol	Dose given should be sufficient to maintain plasma ethanol levels at 1-2 gL^{-1}.
β-Blockers	Glucagon Isophrenaline	5 mg iv over 1 minute followed by an infusion of 1-10 mgh^{-1},10-50 mgmin-1 I.V
Digoxin Digitoxin	Fab antibody fragments	Dose should match estimated Digitoxin .
Ca- channel Blockers	$CaCl_2$, Ca gluconate	10 ml of 10% $CaCl_2$ or30 ml of 10% Ca gluconate over 2 min.0.2 $mlKg^{-1}hr^{-1}$10mlKg-1hr^{-1}.

Heavy metals (lead, Mercury, arsenic)	DMSA(Dimercapto succinic acid)	30 mgkg-1 8 hourly for 5 days (then 20 mgkg-1 12 hourly for 14 Days
	DMPS(Sodium 2,3 Dimercaptopropane Sulphonate)	Chronic: 100 mg 3 times a day. Acute: 250 mg every 4 hours for 24 hours then 250 mg every6 hours for the next 24 hours
	Sodium calcium edetate	Upto 40 $mgkg^{-1}$ twice daily by iv Infusion repeated every 48 hours until level falls below toxic levels.
	Dimercaprol	Mercury: 2.5-3 $mgkg^{-1}$ deep IM injection 4-hourly for 2 days, 2-4 times on third day. .
	Pencilamine	Lead: 0.5-1.5 g per day orally for 1-2 months or until lead levels falls below toxic level.

Iron salts.	Desferoxamine	In severe iron poisoning (> 90 mmolL-1) up to 15 $mgkg^{-1}$ per hour reduced to keep the total iv dose under 80 $mgkg^{-1}$ in each24 hours.
Coral snake Rattle snake bite	Anti-venom	Loading dose $lmgKg^{-1}$ upto 100 mg. Anti-venom is diluted in 1000 ml of saline for adults or 20 ml for child[43]`

Supportive care

The goal of Supportive therapy is to maintain physiologic homeostasis until detoxification is accomplished and to prevent and treat secondary complications.

Respiratory Complications - are the commonest cause of death after acute poisoning and immediate management must be given priority to airway ventilation and prevention of aspiration. Hypoventilation, hypoxia and pulmonary edema should be treated. Treatment consists of O_2, fluid restriction diuretics, mechanical ventilation with PEEP, dialysis.

Cardiovascular complications - include hypotension, cardiac arrhythmias and cardiac arrest. Treatment should be on the lines of peripheral perfusion and maintenance of myocardial function.

Renal Failure - May be due to tubular necrosis because of hypotension hypoxia or direct effect of poison on tubular cells. Haemoglobinuria or myoglobinuria may further precipitate renal failure. Patient should be catheterized to maintain a urine output of 0.5 ml/kg-lhr^{-1} with volume resuscitation or dopamine infusion (2-4gm/kg^{-1} min-l).

Neurological complications - include depression of consciousness level, seizures, cerebral edema and peripheral nerve injuries as a result of prolonged pressure. Therapy consists of correction of ABG and metabolic abnormalities and hypotension, reduction in intracranial pressure, hyperventilation, elevation of head and fluid restriction. Seizures may be because of metabolic disturbances and cerebral hypoxia and direct toxic effect. Treatment by I.V. diazepam /Phenobarbitone or infusion of Thiopentone.

Hypothermia - should be treated by passive re warming, I.V. fluid warming, and warm water humidifier in artificial ventilation. Temperature should be monitored centrally via esophagus or nasopharynx and ECG should be monitored for arrhythmias.

Hyperthermia - may occur with Tricyclic antidepressant cocaine or amphetamine etc. Active cooling with sedation, paralysis and ventilation may be required to control core temperature.

Metabolic complications - The concentration of urea and electrolytes and blood glucose, ABG should be checked routinely. Sodium bicarbonate may be needed if p^H falls below 7.1.

Hematemesis - can be produced by caustics, corrosives, iron, mercury, arsenic, lithium, phosphorous, fluoride mushroom, plant poison and organophosphates. The therapy consists of iced saline lavage, fluid and blood replacement antacids H_2 blockers may be used.

Hepatic and gastrointestinal complications -

Gastric stasis may occur in comatose patients or in anticholinergic or opioid poisoned patient. Early decompression with Ryles tube and stress ulcer prophylaxis may be used [44].

REVIEW OF LITERATURE

M.G.Rajanadh *et al.* (2014) - the Present Study aimed to evaluate the pattern of Poisoning at a tertiary hospital in South India and to study the Socio-Demographic profile of the same. Patients who were admitted to the intensive care unit at SRM Medical College Hospital and Research Center were included in the study. Patients were studied for a period of Eight months retrospectively to record the Incidence, Age, Sex, Domicile Distribution, Education, and Occupation, marital and socioeconomic status. Type of Poisoning, various reasons and Nature of Poisoning were noted. Ethical clearance obtained before the study and then, data was collected. The data was analyzed using descriptive statistics. The Total Number of Poisoning cases was 261. Demographic details were examined. The Type of Poison was founded to be Organophosphate, Oleander Seeds, Snake Bite, Nail Polish, Rodenticide, Alcohol (Methanol), Antifungal Drugs, Antipsychotic drugs and Killer, Endosulphan Food, Hair dye, Kerosene and Miscellaneous. The Study Concludes that Poisoning is a communal cause of Hospital Admission. The most Poisoning cases were Observed with Pesticides handled by the farmers in the Agriculture Fields [45].

O.Gambhir Singh *et al.* (2014) - Poisoning cases is a significant contributor to morbidity all over the world. Acute Poisoning cases form one of the commonest Causes of Emergency Hospital Admission. It is a Retrospective Study of 106 Poisoning Cases Admitted in M.A.P.I.M.S. which is a tertiary health care centre in Tamil Nadu, India from January 2010 to December 2012. The Incidence of Poisoning was highest in the Age Range from 20-50 years. Most Common Poison was Insectide, Rodenticide and Cases were mostly Suicidal in Nature. Trends of Poisoning Cases in Melmaruvathur region of Tamil Nadu are most or less similar to other parts of India [46].

B.Maharani *et al.* (2012) – Poisoning is an important Public Health Problem causing significant Morbidity and Mortality throughout the World. Information available with regard to Acute Poisoning in Adults is limited at Salem, Tamil Nadu. Hence this Study was done with the Objective. It is Retrospective Study conducted during Jan 2009- Jan 2012 in Tertiary Care Hospital.150 cases of Acute Poisoning in Adults due to Drugs and Chemicals were included. Data on Age, Sex, Marital Status, Occupation, Religion, Locality, Type of Poison, Time and Month of Intake, Route of Exposure, Associated Co-Morbid Conditions and Outcome of Poisoning were recorded and analyzed by descriptive method. Among 150 cases, 148 cases were of Intentional Poisoning and two cases were of Accidental Poisoning. In all the Cases the Route of Exposure was Oral. Males (92 cases) Outnumbered Females (58 cases) and 101 Cases were Married. Peak Occurrence was in the Age Group of 21-3- years (47 cases). Occupation Wise Poisoning was commonly found among Male laborers (18.66%) and Farmers (13.33%) followed by House Wife's (28%) and Students (16.66%). 147 cases were Hindus. More Cases reported during summer Season (36%) and day time (80%). Organophosphorus was the commonest agent (58.6%). Associated Co-Morbid Conditions were found in 16 cases. The Incidence of Poisoning and its Morbidity and Mortality can be reduced by developing and Implementation of effective prevention strategies [47].

Shreya.M.Shah *et al.* (2016) - the present study was conducted with aim to generate the Clinico-Epidemiological data of Acute Poisoning Cases presented at Hospital which in turn would be helpful in planning Rational use of available resources for prevention and management of Poisoning Cases. Observational Cross-Sectional Study was carried out from October, 2013 to March 2014. Patients of either Sex of above 12 years of Age of Acute Poisoning admitted in Medicine Emergency Ward were included. Obtained data were analyzed using descriptive statistics and results were expressed as percentage and Mean. Of 340 cases, Male and Female patients were 216(63.53%) and 124(36.47%) respectively. Male:

female ratio was 1.74: 1. Most of the cases belonged to the Age Group of 21-30 years (38.82%). Ingestion was the most common Route of Exposure (71.47%). Intentional (Suicidal) Poisoning was recorded in 62.06% Cases followed by Accidental Poisoning (37.94%). Common causes of Poisonous Bites (25.88%) followed by Organophosphate (19.41%) and unknown compound (19.41%) Ingestion. Commonly observed symptoms were Vomiting, local symptoms in such cases of Bites, altered Sensorium, Giddiness and Breathing Difficulty. Average number of days of Hospitalization was 5.39 days. Complete Recovery and Mortality were seen in 66.47% and 16.47% cases respectively. Acute Poisoning is one of the leading cause of hospital admission and Mortalities high costs of treatment and Intensive Care Burden makes poisoning an important area for further research. The current study has managed to contribute sub-stantial additional information regarding the epidemiology and outcome of poisoning in a tertiary care hospital at a district level [48].

A.Muruganathan *et al.* (2017) – Auromine is a florescent Yellow, Tasteless, Odour less, water soluble highly lethal, easily available household poison which kills about thousands of people in Coimbatore, Tiruppur and Erode districts and southern parts of Tamil Nadu. This a prospective study to analyze the clinical presentations, symptoms, signs, biochemical changes in a patients with yellow cow dung powder poisoning, and strategies of management as there is no protocol of management available except symptomatic treatment and it is highly toxic, only few case studies are published and no detailed research papers available. All the patients admitted in Govt Medical College Hospital with history of alleged Yellow Cow Dung Powder Poisoning are taken for study. Those patients with per existing liver diseases are excluded from this study of the 1183 No of Patients studied many 23% of them exhibited signs of Liver Cell Failure, 28% Bleeding Manifestations, 43% Neurological Manifestations 6% had other complication, conclusion; early gastric lavage, in conscious patients, followed by continuous ryles tube aspiration washing the skin and eyes, symptomatic treatment for

hepatitis prevention of hepatic encephalopathy, convulsions and ventilator support for patients with respiratory failure, treatment of arrhythmias are the main treatments [49].

K.Suneetha *et al.* (2016) - Rat killer poison consumption cases are among the second most common poisoning in developing countries. It is associated with significant mortality and morbidity. However, some of the cases get discharged without any effects. This is because of the variability in content. So, the chemical content of the rat killer poison decides the mortality and morbidity. The purpose of this study is to evaluate the clinical outcome of the rat killer poisoning cases with its relation to the chemical content of the Poison. It is a retrospective study conducted on patients admitted to K R Hospital, Mysore. As per inclusion criteria and exclusion criteria cases are included and excluded and a prestructed Performa was used, data were entered. The study is approved by the institutional ethical committee. Most of the cases were young adults. Both the genders were equally affected. High mortality rate found in aluminum phosphide and zinc phosphide containing compounds consumption with cardiogenic shock. Those cases with yellow phosphorous poisoning were stable on day 1 or 2, worsened 3 or 4 with multiple organ dysfunction syndromes. So the chemical content of poison is important for the prognosis and also intensive monitoring and early interventions[50].

Achinta Mandal *et al*. (2016) – Poisoning is a common medical emergency in childhood and one of the important causes of hospital admission and also of death. An institution based cross-sectional observational study was conducted in B S Medical College and Hospital, Bankura, West Bengal, India. Among 89% cases of poisoning studied, 62 cases were between 1 to 3 years of age. Overall mortality was 6.67 % and 8.89% cases required intensive care

support. This study helps us to know the clinico-epidemiological profile of poisoning in children in this part of the country[51].

C.Arulmurugan *et al.* (2015) - acute poisoning is a medical emergency. It is important to know the nature, outcome and severity of acute poisoning cases in order to make appropriate prevention and treatment. This study is conducted to assess the paradigm and outcome of acute poisoning cases in a tertiary care hospital in Tamil Nadu. This is a retrospective study conducted in a tertiary care hospital in Tamil Nadu. The study included 169 cases and data regarding age, sex, time elapsed after intake, name of the poisons, chemical type; duration of hospital stay; outcome and severity were collected in the structured Performa. Incidence was high among males (60.36%). Most of the cases of acute poisoning were in the age group of 0 to 30 years (60.95%) followed by 30 to 50 years age group (30.77%). A majority of poisoning cases (27.2 %) were due to Orgnophosphorous (OPC) insecticide. Total mortality was found to be 5.32%. mortality rate due to paraquat, abrus precatorious seeds was significantly high compared with OPC because ther is no specific antidote. Time lapse had a very significant role in the mortality in cases of poisoning. Poisoning is common with young males. The mortality is high, in cases of self-poisoning with Paraquet and abrus seeds. Despite the highest consumption rate, no mortality was observed with Orgnophosphorus(OPC) because of early medical intervention and specific antidote. Early medical care hospital will help to reduce significant mortality in India[52].

K.Vivekanadan *et al.* (2011) - To determine the incidence and severity of poisoning cases in Meenakshi Medical College and Research Institute. Retrospective and Prospective Observational study. All the poisoning cases due to various agents who attend Emergency from Meenakshi Medical College and Research Institute over a period of Jan 2007 to march 2010 were evaluated retrospectively and prospectively. A total of 232 poisoning cases were attended emergency Meenakshi Medical College and Research Institute over a period of 39

months. The overall male to female ratio was 1.5: 1. The majority of the poisoning cases were found in the age groups of 16-30 years (42.2%). Organophosphorus(OPC) was the most commonly used for self poisoning 31%. Farmers 32.35% , service holders 22.41% and students 20.3% were commonly involved in self poisoning. Intentional poisoning comprised 46.5% of all poisoning. Majority causes of intentional / accidental have been identified and factors contributing occupation related agro chemical poisoning are discussed[53].

N.Malangu *et al.* (2009) - The aim of this study was to characterize acute poisoning cases to a number of selected hospitals in South Africa. All cases admitted to eight hospitals from January 2005 to June 2005, were evaluated retrospectively. Dated obtained from the hospital medical records included the following: demographic characteristics, toxic agents, length of hospital stay, and circumstances of poisoning, morbidity and mortality information on the poisoned patients. From a total of 424 patients admitted for treatment, whose median age was 17.6yrs, 57.8% were females, and 89.6% black Africans, fifty-nine percent of the Poisonings were accidental, and the involved toxic agents were, in descending order; household chemicals (45.7%), modern medicine (17.5%), animal/insect bites (15.8%), agrochemical chemicals(9.7%), Food Poisoning(5.4%), drugs of abuse (3.3%), traditional medicines (2.4%) and plants (0.2%). Poisoning by drug of abuse was commoner in males than females, but the percentage of Females Poisoned by all other toxic agents was higher than in males. Most patients spent less than two days in hospital, but more females (70.1%) than males (29.9%) stayed for more than two days. The overall case fatality rate was 2.4%. of those who died, 80% were black Africans, aged 3 to 19 years and it was delibrate poisoning through drugs of abuse ,carbon monoxide and agricultural chemicals. Acute poisoning reviewed in some selected hospitals in south africa revealed that more black africans females were involved, who spent more than two days hospitalised. The case fatality rate was 2.4% mainly due to drugs of abuse, carbon monoxide and agricultutral chemicals. These findings suggest that

further studies are needed to understand the motivation(s) for this emerging problem and that these should focus primarily on the female black African[54].

Avinash *et al.* (2016) - cow dung poison is commonly known as sani powder poison in local tamil language in south india , it is a lethal poisonous synthetic chemical. Cow dung was traditionally used as a germicide and insect repellent to clean homes, courtyards and temples in the state of tamil nadu, since the unavaibility of cow dung synthetic chemicals are used to prepare this sani powder. Sani powder poison is available in two colours that is yellow and green. Sani powder is available in two varities. 1. Yellow powder: auromine , chemically known as diaryl methane dye. 2. Green powder: malachite green, chemical constituent being triphenyl methane. The modern population started using this deadly sani powder instead of cow dung for the same purpose. This was either due to no availability or inaccessibility of the natural cow dung in the urban areas. Inspite of a legal ban on this chemical, it is easily procurable at they grocery shops for a mearge price of 3-5 rupees/packet. Cow dung poison is lethal as it has no antidote. This deadly neurotoxic poison causes severe hepatotoxic, nephrotoxic, ocular and GI damage, to prevent the damage of the multiorgans n-acetylcystein (NAC) drug have been used to counteract on this deadly poison[55].

Subash vijaya kumar *et al.* (2010) - organophosphorous(OPC) compounds constitute a heterogenous category of chemicals specifically designed for the control of pests, weeds or plant diseases. Our review article mainly focused on OP poisoning, especially with pesticides, its severity and management of toxic exposure. So, we searched science direct, medline and pubmed bibliographic databases using the key phrases causes of organophosphorous compounds, diagnosis, management of OP poisoning and drugs under clinical trials. Our review article examines pathophysiology, clinical manifestations, toxicokinetics of OP poison, its prevention and management. In addition to that, our review suggested antioxidants should be administered for OP poisoning patients to reduce severity.

We conclude that in future, the ministry of agriculture of developing countries especially india, should concentrate on the optimization and monitoring of usage of OP compounds as pesticides and furthermore, encouraging the farmers to use natural pesticides rather than chemical pesticides[56].

Thalapillil Mathew Celine *et al.* (2017) - conducted a study on patients admitted with poisoning in a tertiary hospital with 2018 cases. Data has been collected from the registers kept in the medical record department. Most of them were admitted with organophosphorous and insecticides poisoning. Mortality rate was found to be high in males. The easy accessibility of poisonous substances from the nearby shops should be restricted for controlling these types of poisonous deaths[57].

Tanuja R. Brahmankar *et al.*(2017) – carried out the study in government medical college, Miraj, Maharashtra. They reported a record base cross sectional study in which all the information about the patients were collected from the MLC (medical legal cases) record book for a period of 6 months. Majority of the victims were males, young adults and urban inhabitants. Most of common indication for MLC was homicidal followed by medical check-up of prisoners and road traffic accidents[58].

Heethal Jaiprakash *et al.* (2016) - conducted study in rural area of south india in one hospital around one year. They have given a detailed report on the poisoning cases admitted in that hospital and provide advice to establish strict policies against the use of pesticides and drug abuse. Young adults in the economically productive age group were the most common victims with suicidal intention. Pesticides and household products were the agents that were commonly used that mostly dominated group is female and most of them had education between 7^{th} to 10^{th} standard. Data reported that unmarried group Is high number compared to male groups, in which non-steroidal anti-inflammatory drugs are mostly reported and

followed by anti-epileptics. Awareness and counseling can bring the drastic education in the number of suicidal attempts of drug poisoning[59].

Linto Mathew Thomas *et al.*(2016) – performed a prospective study at R.M.M.C hospital Chidambaram during the period of February 2016 to april 2016. Most of the poisoning cases were phsychological stress. Suicidal tendency was in the age group of 21-30 years. The common substance used to cause deliberate self-harm was organo-phosphorous ompounds. Most of the suicides were attempted by the married patients. Majorly, low class of socio-economic status of population committed suicide[60].

Natarajan *et al.* (2016) – studied about 200 cases of snake bite during the year 2002. 15 patients had anti-snake venom (ASV) treatment within 12 hours and mortality rate among them was 2.9%. most of the bites were due to viper bite. Commonest cause of death was acute renal failure. Bleeding phenomena included bleeding from the site of bite, haematuria, hematesis, malena and epistaxis. Clotting time was prolonged in most of the patients. 50-60ml of ASV was administered every 4-6 hours until clotting time normalized. They also analyzed the creatinine level, urine analysis, bleeding time, clotting time are noted[61].

Prashant Gupta *et al.* (2016) – conducted the study in Uttar Pradesh and the results of the suicidal cases were mostly due to family problems maximum cases received help within 4-8 hours of ingestion or exposure to poisonous substance. Maximum cases were reported with married patients. Prevailing treatment protocols require updating on proper guidelines for better management of poisoning. Pattern and magnitude are multidimensional and demand approach for facting this problem[62].

Singh R R *et al.* (2016) – conducted a study in tertiary care hospital in eastern Uttar Pradesh in the period of December 2015. Out of 140 cases reported in the hospital, the results showed that the rural people resort to pesticides and urban people resort to house hold articles and

drugs poison. They tackled the mortality of poisoning cases by type of poison, amount consumed and time taken to health care[63].

Pratik d. Asari *et al* (2016) – designed a study to investigate the poisoning agenst and outcome in patients in vadadhora, Gujarat. They carried the study for around 6 months. The most common treatments were anti-emetics, H_2 blockers and antimicrobials. Atropine, pralidoxime and anti-snake venom were the specific antidotes for the poisoning cases. Over half of drugs were prescribed in generic and nearly one-thirds of drugs were prescribed in brand name. they concluded that educational programs with emphasis on preventive measures for toxic exposures are necessary to create awareness among the general public[64].

Adhitya Prasad Sarkar *et al.* (2015) – conducted a descriptive study in burdwan medical college and hospital, west Bengal. Majority of cases were hindu, housewives in the age group of 20-30 years of age. Two –third of the cases sougth medical care with in 2 to 4 hours after ingestion of poison. Parts of them are spouse followed by quarrel with other family members or friends. Majorly patients are in rural area. Organophosphorous was the most commonly used poison. In this study male and female patients are in equal proportion[65].

Abishek Prayag *et al.* (2015) – conducted a retrospective study 1 year in KLE Hospital, Belgavi.. poisoning rates are seen among the farmers and young population. Intentional poisoning accounted for higher incidence and mostly with organophosphates. Socio-demographic details such as age, sex, occupation, type of poisoning, length of hospital stay, outcome and other details were collected from the patient case notes, treatment charts, nursing notes, laboratory reports, and discharge summaries. Most of the cases are by intentional poisoning by consuming alcohol shows the percentage of (15.6%)[66].

Baraka gupta *et al.* (2015) – conducted a study on Shrada hospital for a period of 2 years and 1214 cases reported in casuality. They conveyed that maximum incidence was reported at

the time of 8 am to 4 pm among the males. Mostly, patients reported to casuality in consciousness. Out of which 40% patient was discharged and mortality rate was 10.2 %. Poisoning by agrochemicals were seen in majority of cases. Part of the cases were reported due to family problems, pshychiatric , illness and study related problems[67].

Naresh *et al.* (2015) – aimed to evaluate the clinical features, management and outcome of poisoning. They conducted the study for a period of 18 months in the tertiary care hospital in south india. They concluded that OPC poisoning were found to be more common followed by herbicides and mostly occurring in males. Drug poisoning with norethisterone, alprazolam, amlodipine are also seen. All the poisoning cases have complete recovery from the non-survival state[68].

Koulapur *et al.* (2015) – conducted a retrospective study in 210 patients for a period of 6 years from 2003 to 2009 at B.M. patil college, Bijapur. Their study conveyed that most of the cases were found to be reported with pesticide poisoning especially organo-phosphorous compounds under the age group of 31-40. The establishment of specialized toxicological units for detection and management of poisoning cases at all hospitals and primary health care center could considerably minimize the morbidity and mortality due to poisoning[69].

Khalid I. Khatib *et al.* (2015) – conducted the prospective observational study for a period of 51 months. They reported that mostly females were admitted and organophosphate area a common poison. The most common used drugs are sedatives followed by antipsychotic, antidepressants and paracetamol. Young adults and females are affected. Death rate was high. This public health menace needs time, money and concerted effort to create iregulations, awareness and information centers and helplines to crub it[70].

Mukul Joshi *et al.*(2015) – conducted a retrospective study to know the pattern of poisoning in a tertiary care hospital, ahmedabad. Patient data were obtained from medical records and

were documented in a pre-structured proforma. Poisoning is more common in young males so they should be emotionally supported in stressful circumstances. Most of them were illiterate class of patients. Mortality was found to be higher in class of organophosphorous poisoning[71].

Vivendra Mahadik *et al.* (2015) – conducted the study in MGM Hospital and Research centre, belapur, navi Mumbai for a period of five years with 234 cases reported. It is seen that female ratio was high in incidence with age group of 16-35 years. The study also reported that young married women show higher risk of poisoning. This is the first study in maharastara in which married women contribute higher ratio in poisoning[72].

Farhanabashmir *et al.* (2014) - conducted the study in Kashmir to know sociodemographic variables and type of poison consumed from jan 2006 to may 2014. It was seen that many unmarried female contribute more in the age group of 18 to 35 years was mostly. Organophosphorous poisoning was predominant. The study also reported that unmarried individuals are more responsive to psychological stress than married individuals [73].

Ajay Risal *et al.* (2013) – determined the pattern and severity of poisoning cases admitted in dhulikhel hospital, kavre. They analyzed various patterns like age, sex, marital status, time of ingestion , month of occurrence, type of poisoning, outcome and duration of treatment. They encouraged decresing the incidence and mortality by restriction in free sale of the poison and promoting information center[74].

Jesslin *et al.* (2010) – performed a retrospective study in mysore hospital for a period of one year. Most of the poisonous cases were with pesticides followed by rodenticide, kerosene and hair dye. Household products poisoning were seen with cases below the age group of 5 years. It was also observed that incidence of poisoning was found to be decreased with the increase in age. In children, most of the poisoning cases were seen in boys compared to the girls[75].

Shivani Patel *et al.*(2016) - Pyrethrins are naturally-occurring compounds with insecticidal properties that are found in Pyrethrum extract from certain chrysanthemum flowers. The Pyrethrins are often used in household insecticides and products to control insects on pets or livestock. Pyrethroids are manufactured chemicals that are very similar in structure to the Pyrethrins, but are often more toxic to insects as well as to mammals and last longer in the environment than the Pyrethrins. More than 1,000 synthetic Pyrethroids have been developed, but less than a dozen of them are currently used. Permethrin is the most frequently used Pyrethroid insecticide.

AIM AND OBJECTIVES

AIM

- The aim of the study is to characterize Poisoning Cases admitted to the Government District Head Quarters Hospital, Tiruppur.
- To access the Prevalence & Mortality Case Due to Various Poisoning Agents.

OBJECTIVE

- To List out the Common Poisonous Materials.
- To collect the Poison cases from Government District Head Quarters Hospital Tiruppur.
- To collect the Data Mode of Poisoning and Nature of Poisoning.
- To collect the Data about the Common Treatment to Patients.
- To collect the Poison Death Cases from Medical Record Department in Government District Head Quarters Hospital Tiruppur.
- To collect the Data about Nature of Death in Poison Cases.

PLAN OF WORK

The present study was focused to collect the information about Morbidity & Mortality from Government District Headquarters Hospital, Tiruppur. The present study was planned to collected data in Toxicological Emergency Ward and Medical Record Department at Government District Head Quarters Hospital, Tiruppur.

Plan of work:

- Collection of Various Poison Cases.
- Designation of a Toxicological Work Sheet Form (work sheet contains Patient Name, Compound and Quantity of Poison Intake, Mode of Ingestion, Date, Time, Sex, Age, Nature of Poison , Patients Address)
- Evaluation of the collected Data on the basis of Age, Sex, Nature of Poisoning and Mode of Poisoning.
- Collection of the Death Cases Due to Poison.
- Collection of the Data on the basis of Nature of Poison, Mode of Ingestion, Mode of Death.

PATIENTS AND METHODOLOGY

- **STUDY SITE –** Government District Head Quarters Hospital, Tiruppur.
- **DURATION OF STUDY – 7** months
- **STUDY POPULATION -** from the above said studies cases were collected. Cases like Cow Dung, Rodenticides, Snake Bite, Oleander, Alcohol Intoxication, Tablet Poisoning, Organophosphorous compound (OPC), Ant Killer, Cockroach Killer, Alcohol+OPC, Hair dye, Lysol, Machine Oil, Dettol+Kerosene including Insecticides. This study is mainly based on Mode of Poisoning, Nature of Poisoning and Death Due to Poison.
- **FOLLOWING OBSERVATIONS WERE MADE**
  - Name
  - Age
  - Sex
  - Mode of Poisoning (Poisoning due to Accidental or Suicidal significant Morbidity & Mortality)
  - Nature of Poisoning (pattern of Poisoning in a region depends upon various factors such as Availability Cost and access to Toxic Agent, Socio-Economic Status, Cultural and Religious Characteristic of People e.g. Cow Dung, Rodenticides, Pesticides, Oleander, Snake Bites and Alcohol Intoxication etc.
  - Common treatments given to the patients. (Activated Charcoal- it blinds to the Poison and stop it from being further absorbed in the blood)

    Anti-dote – this prevents the Poison from Working or Reverse effects of Poison

    Sedatives – if the person agitated sedative will be given.

    A ventilator (breathing machine) – this may be used if the person stops breathing.

DEATH CASES DUE TO POISON:

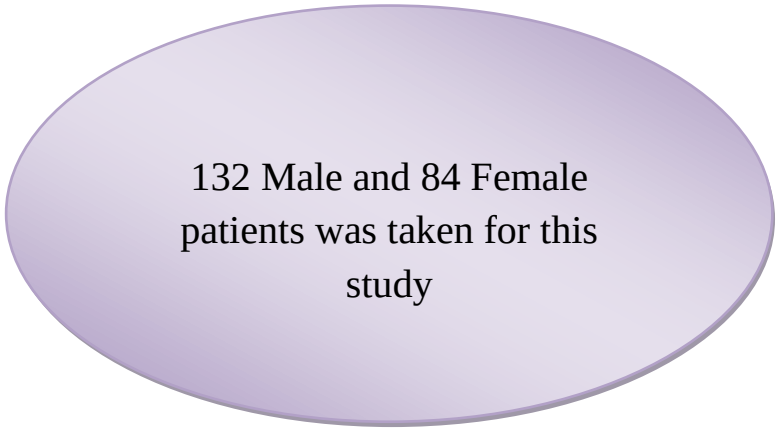
- A Poison Death Case was collected in Medical Record Department (MRD), Government District Head Quarters Hospital, tiruppur.
- Data has been collected on the basis of Nature of Poison, Mode of Poison, Name, Age, Sex, Marital Status and Month Wise.

RESULTS

GENDER WISE DISTRIBUTION

Table: 3 Showing Gender Wise Distribution of Poisoning

SEX	NO OF PATIENTS	PERCENTAGE %
MALE	132	62
FEMALE	84	38
TOTAL	216	100



.

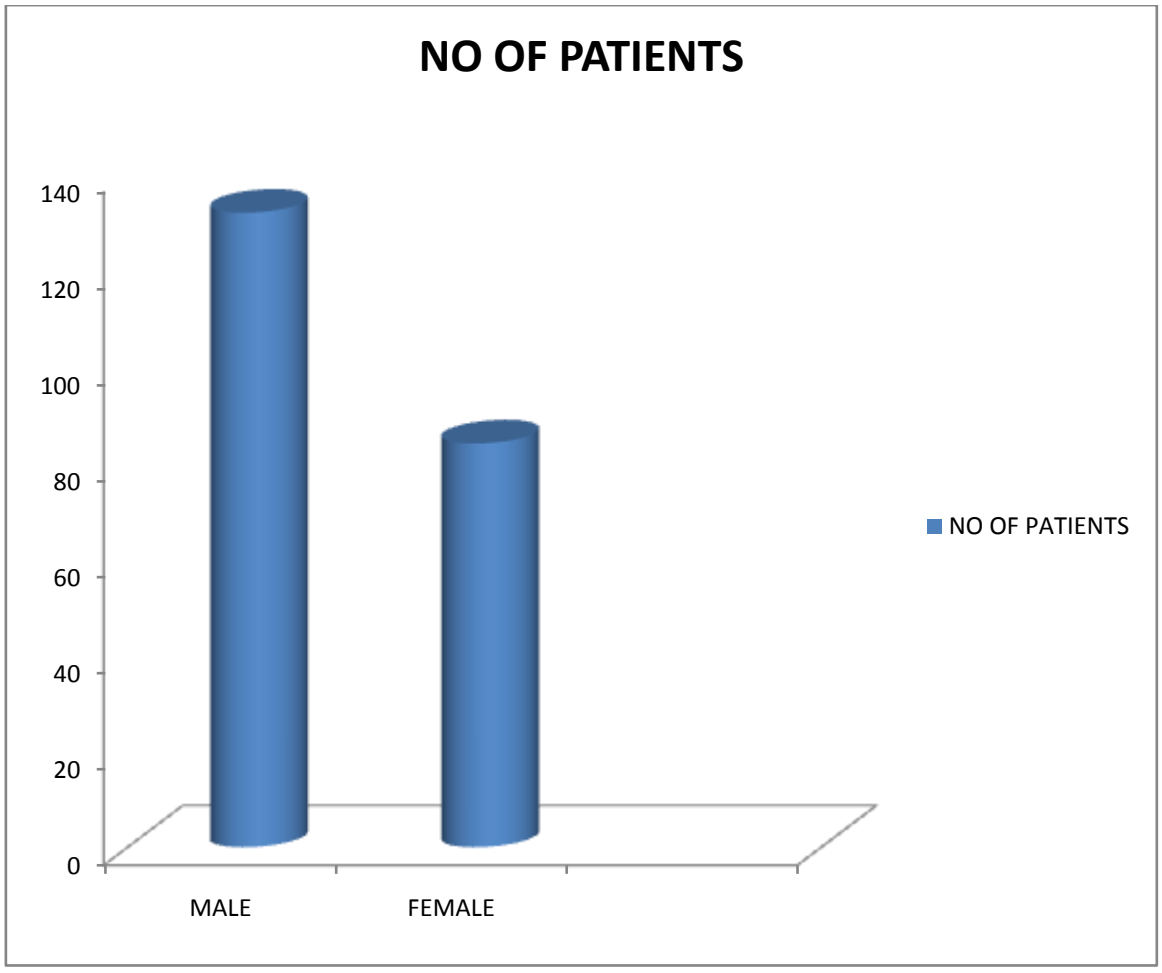


Figure: 11 Illustrates Percentage of Gender Wise Distribution in Poisoning Patients

Out of 216 patients 132 (62%) patients were Male and 84(38%) patients were Female. According to this study Males victims are more than Females.

AGE WISE DISTRIBUTION

Table: 4 Showing Age Wise Distribution of Poisoning Patients

AGE GROUP (YEARS)	NO OF PATIENTS	
	MALE	FEMALE
2-10	3	1
10-20	20	13
20-30	56	30
30-40	22	16
40-50	18	10
50-60	6	5
ABOVE 60	7	7

Maximum number of patients belongs to 20-30yrs. Males were the major victims

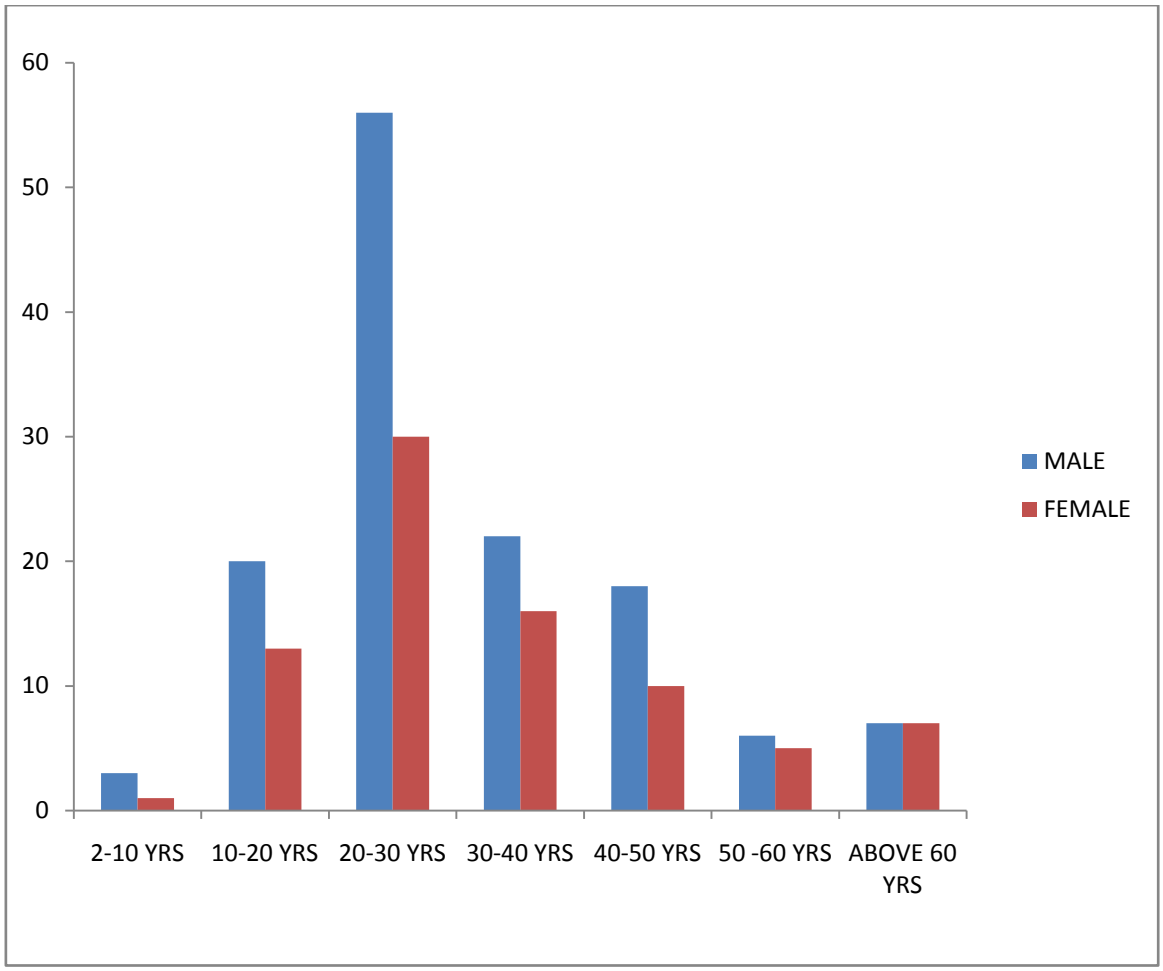


Figure: 12 Illustrates Percentage of Age Wise Distribution of Poisoning Patients

In this study the results show that Maximum Number of Patients belongs to 20-30 years in Male victims 132(62%) followed by Age Group of 30-40years.

NATURE OF POISON

Table: 5 Showing Nature of Poisoning Cases

TYPES OF POIOSNING	NO OF PATIENTS	PERCENTAGE%
Cow dung	68	32
Rat killer	36	17
Snake bite	24	11
Tablet poisoning	16	7
Organophosphorous	14	6
Oleander	15	6.5
Good night liquid	8	4
Insecticide	8	4
Ant killer	12	5
Hair dye	3	1.5
Cockroach killer	4	2
Machine oil	1	0.5
Lysol	3	1.5
Dettol+kerosene	2	1
Alcohol+organophosphorous	2	1
Total	216	100

Major cause of Poison was Cow Dung.

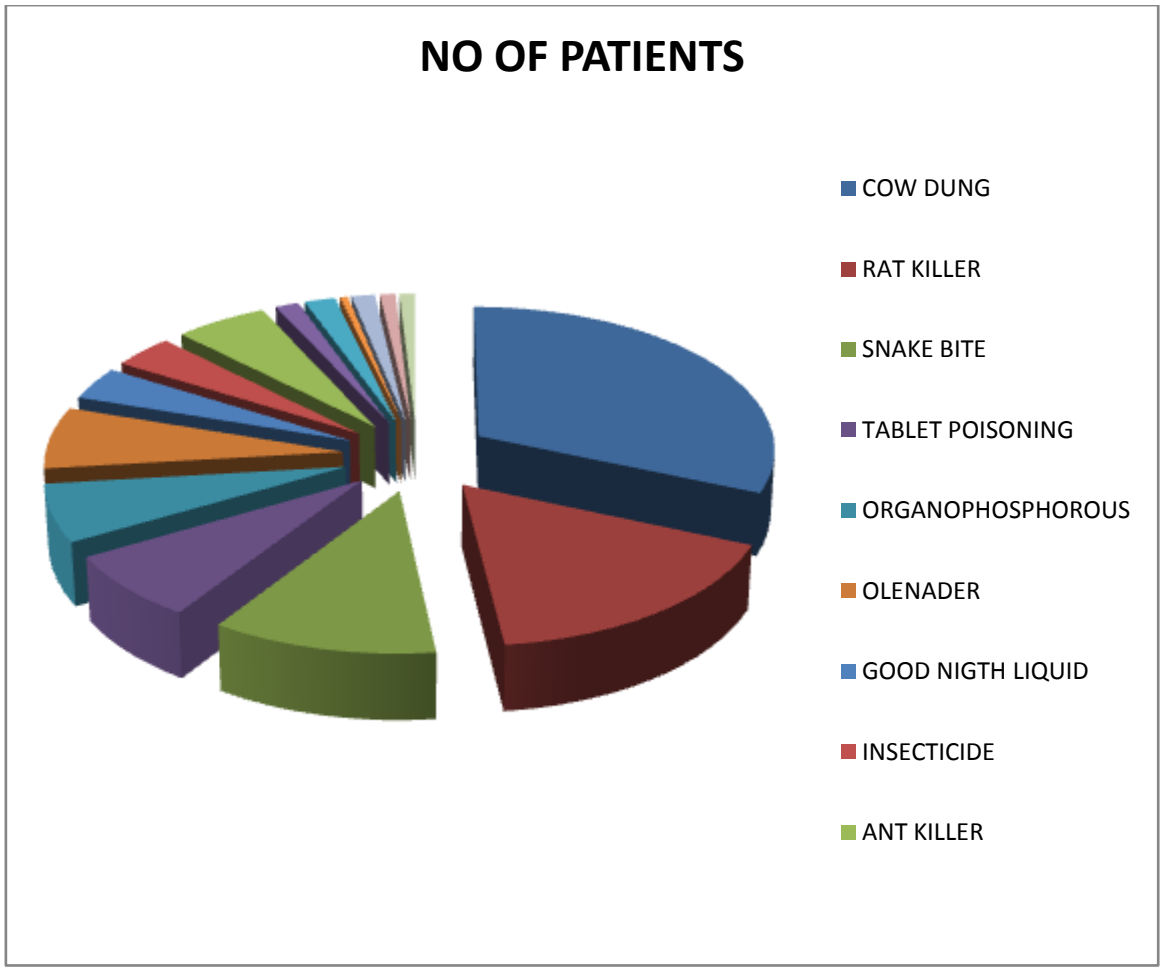


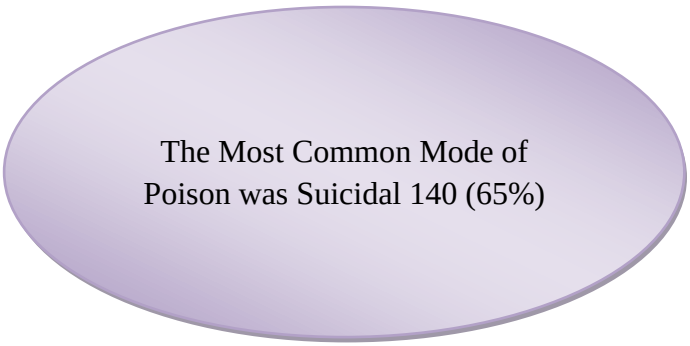
Figure: 13 Illustrates Percentage of Different Types of Poison

The overall Poisoning cases this study shows that Cow Dung (Auramine-O) was the major cause of poison 68 (32%) was followed by Rat Killer poisoning 36 (17).

MODE OF POISON CASES

Table: 6 Showing Mode of Poisoning Cases

MODE OF POISON	NO OF PATIENTS	PERCENTAGE%
Accidental	76	35
Suicidal	140	65
Total	216	100



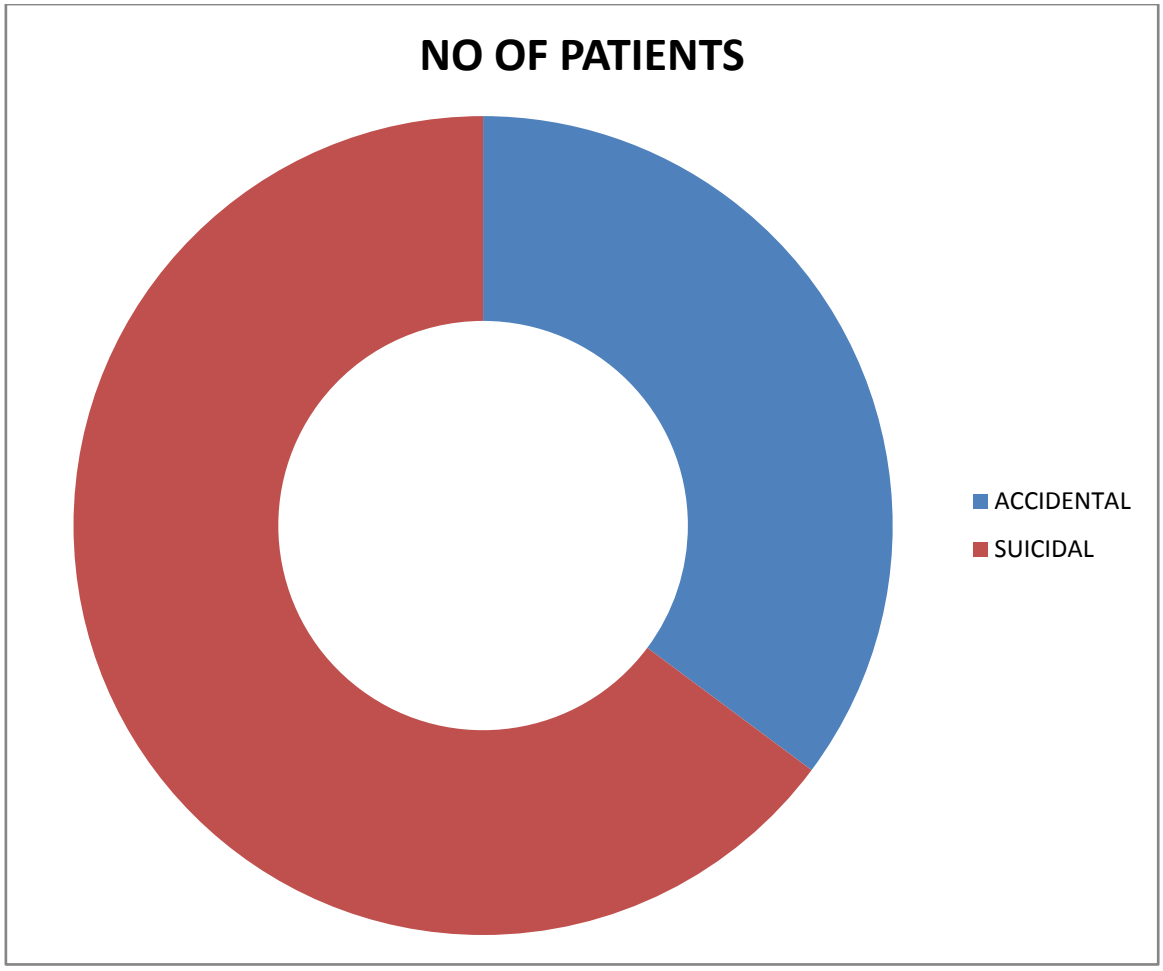


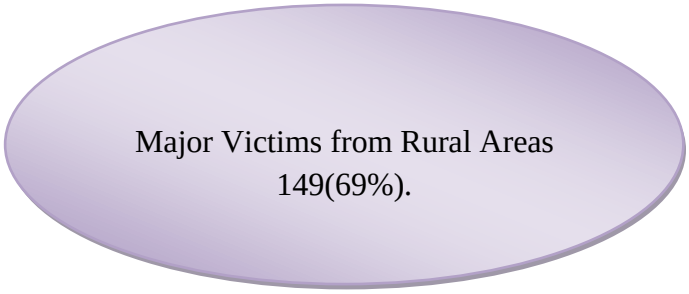
Figure: 14 Illustrates Percentage of Mode of Poisoning Cases

This study shows that the Most Common Mode of Poisoning was Suicidal 140(65%) followed by Accidental 76(35%).

AREA WISE DISTRIBUTION

Table: 7 Showing Area of Poisoning Cases

AREA WITH DISTRIBUTION	NO OF PATIENTS	PERCENTAGE %
Urban	67	31
Rural	149	69
Total	216	100



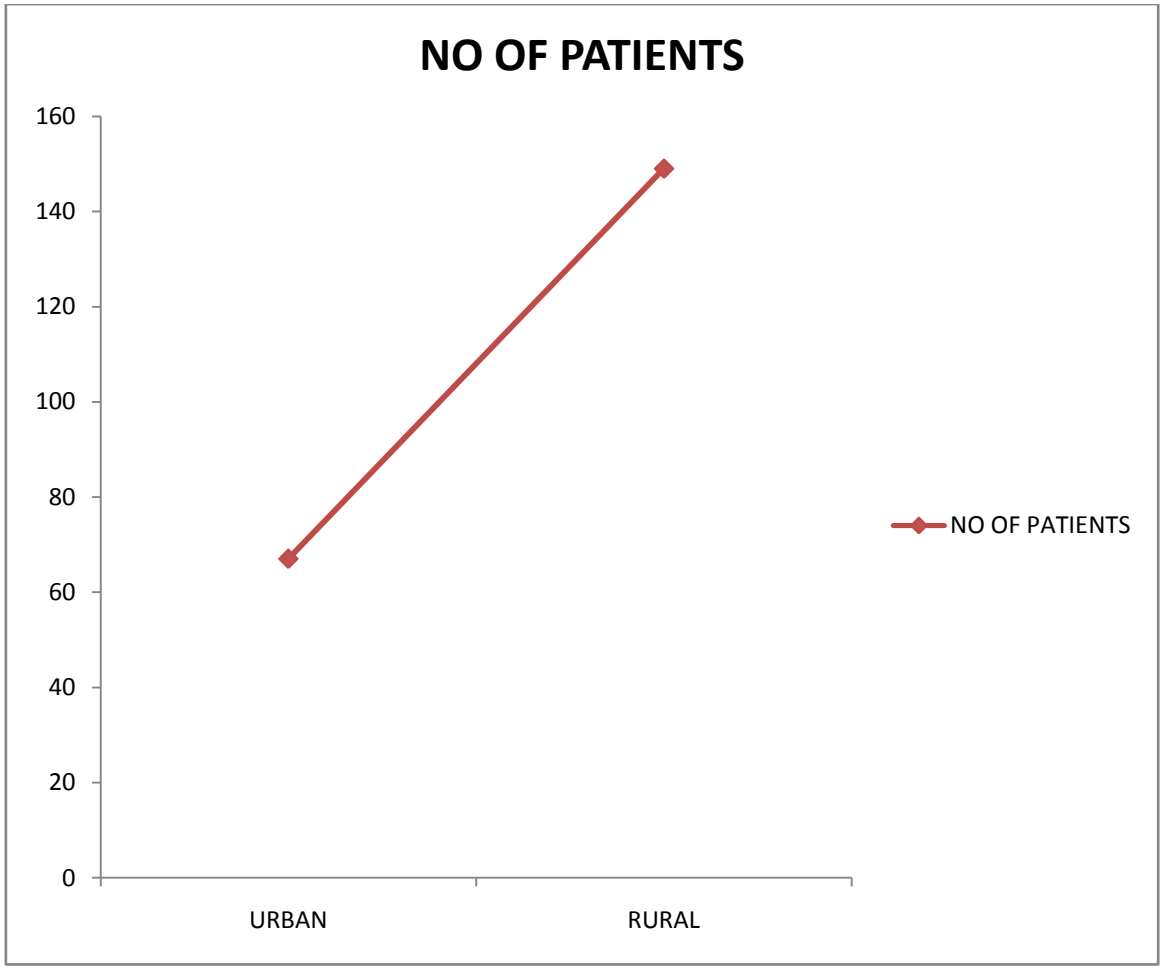


Figure: 15 Illustrate Percentage of Area of Poisoning

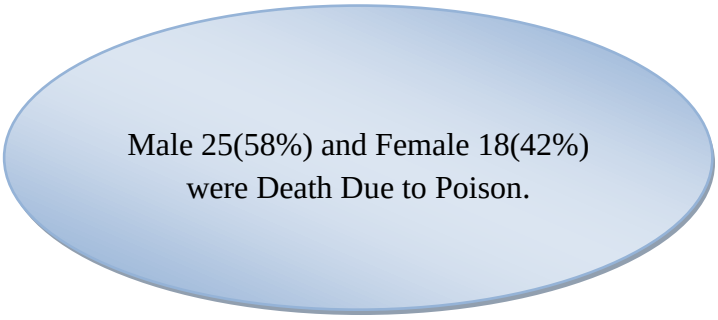
Majority of Cases referred from Primary Health Center or Rural Health Centre. In this study 149(69%) cases from Rural Areas and 67(31%) Patients were from Urban Areas.

POISON DEATH CASES

GENDER WISE DISTRIBUTION

Table: 8 Showing Gender Wise Distribution of Poison Death

SEX	NO OF DEATHS	PERCENTAGE
MALE	25	58
FEMALE	18	42
TOTAL	43	100



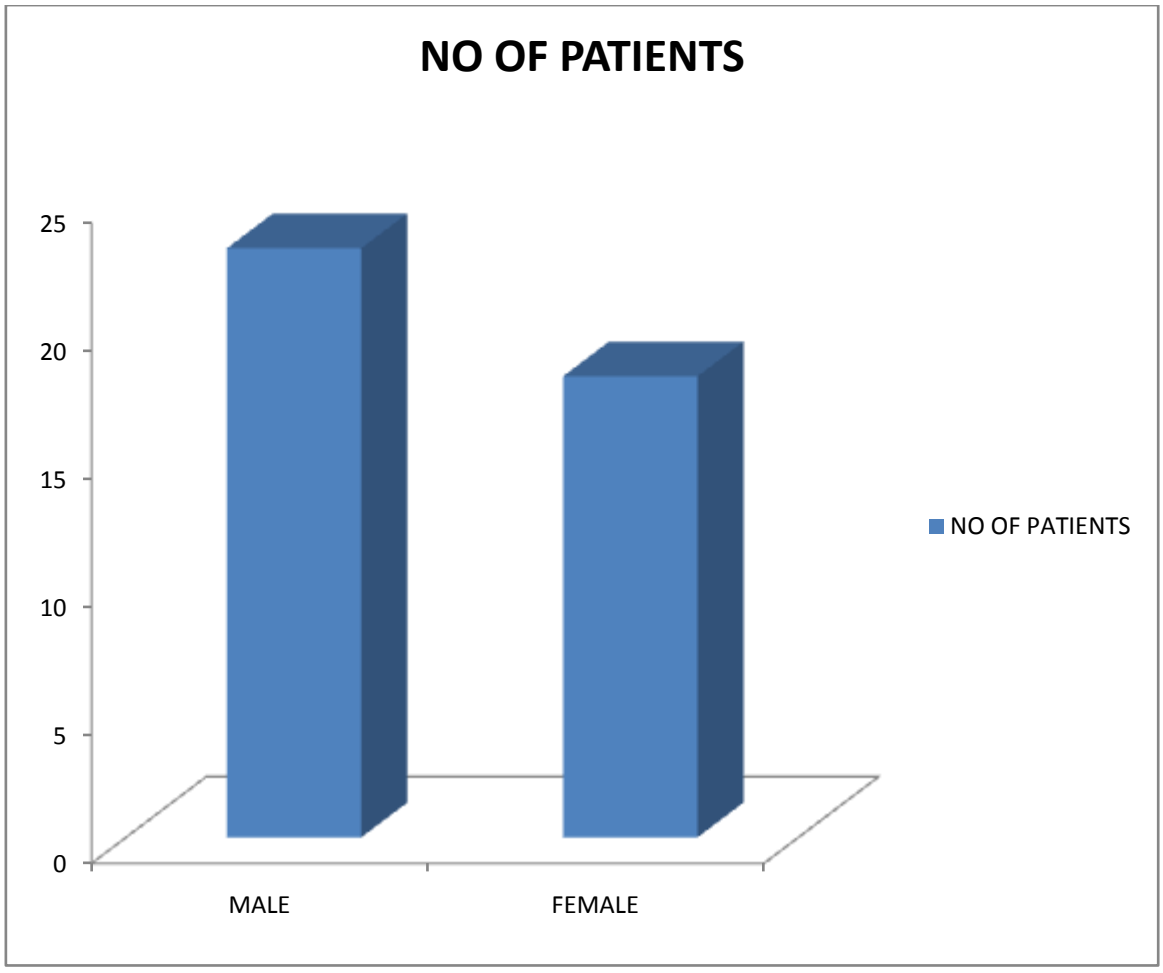


Figure: 16 Illustrate Percentage of Gender Wise Distribution in Poison Death Cases.

Out of 43 cases 25(58%) were Males and 18(42%) Patients were Females. According to this Study Males victims are more than Females.

AGE WISE DISTRIBUTION

Table: 9 Showing Age Wise Distribution of Poison Deaths Cases

AGE GROUPS (YEARS)	NO OF PATIENTS	
	MALE	FEMALE
2-10	-	-
10-20	1	1
20-30	7	7
30-40	3	3
40-50	6	2
50-60	4	4
ABOVE 60	4	4

Maximum Number of Patients are belongs to 20-30 yrs. Male and Female are both equal.

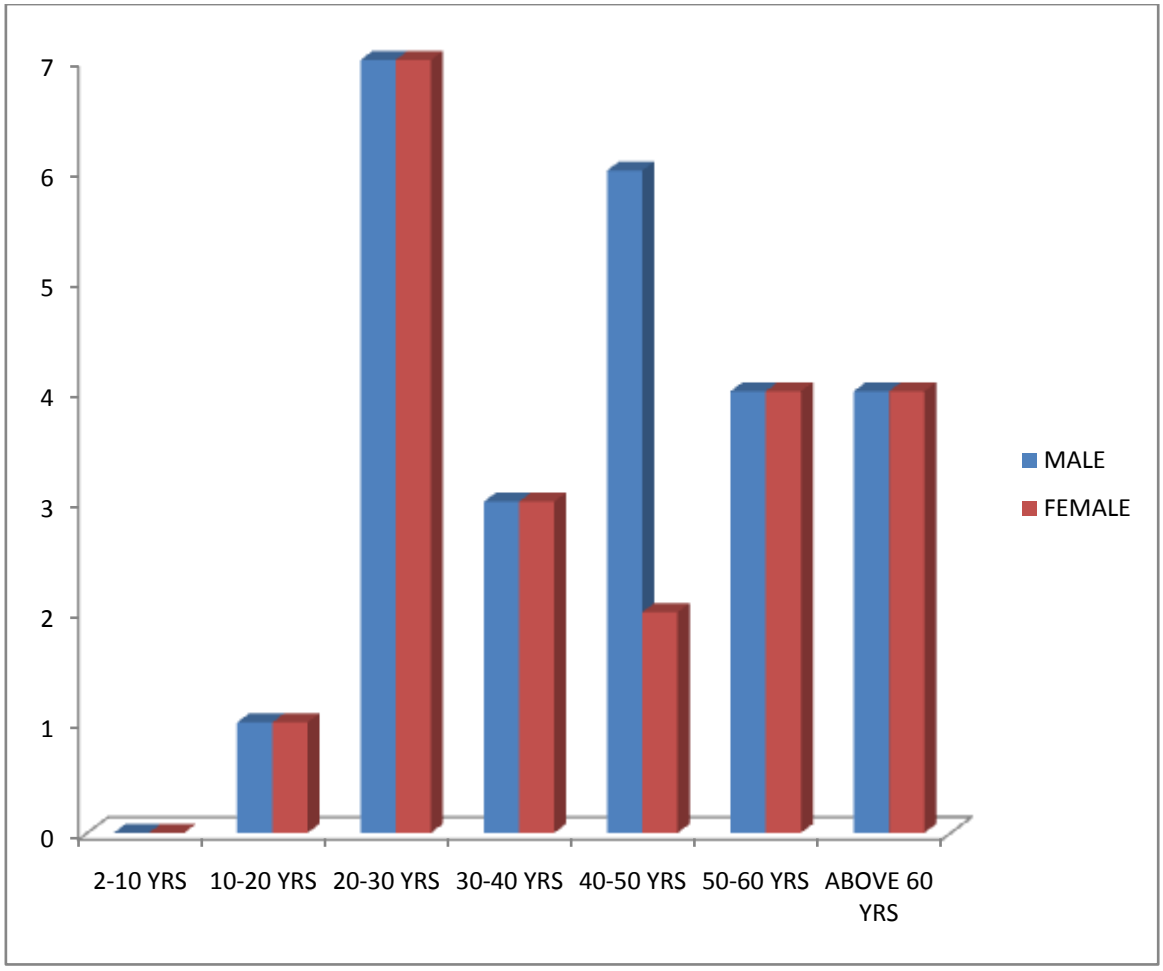


Figure: 17 Illustrate Percentage of Age Wise Distribution of Poison Death Cases.

In this study the results show that Maximum Number of Patients belongs to 20-30 years Male and Female victims followed by age group of 40-50yrs Male.

MONTH WISE DISTRIBUTION

Table: 10 Showing Month Wise Distribution of Poison Death Cases

MONTH	MALE	FEMALE	TOTAL
NOVEMBER-16	1	2	3
DECEMBER-16	4	2	6
JANUARY-17	2	1	3
FEBRUARY-17	9	-	9
MARCH-17	3	6	9
APRIL-17	1	1	2
MAY-17	1	3	4
JUNE-17	3	4	7

Maximum Number of Patients belongs to February and March. Females are Major Victims.

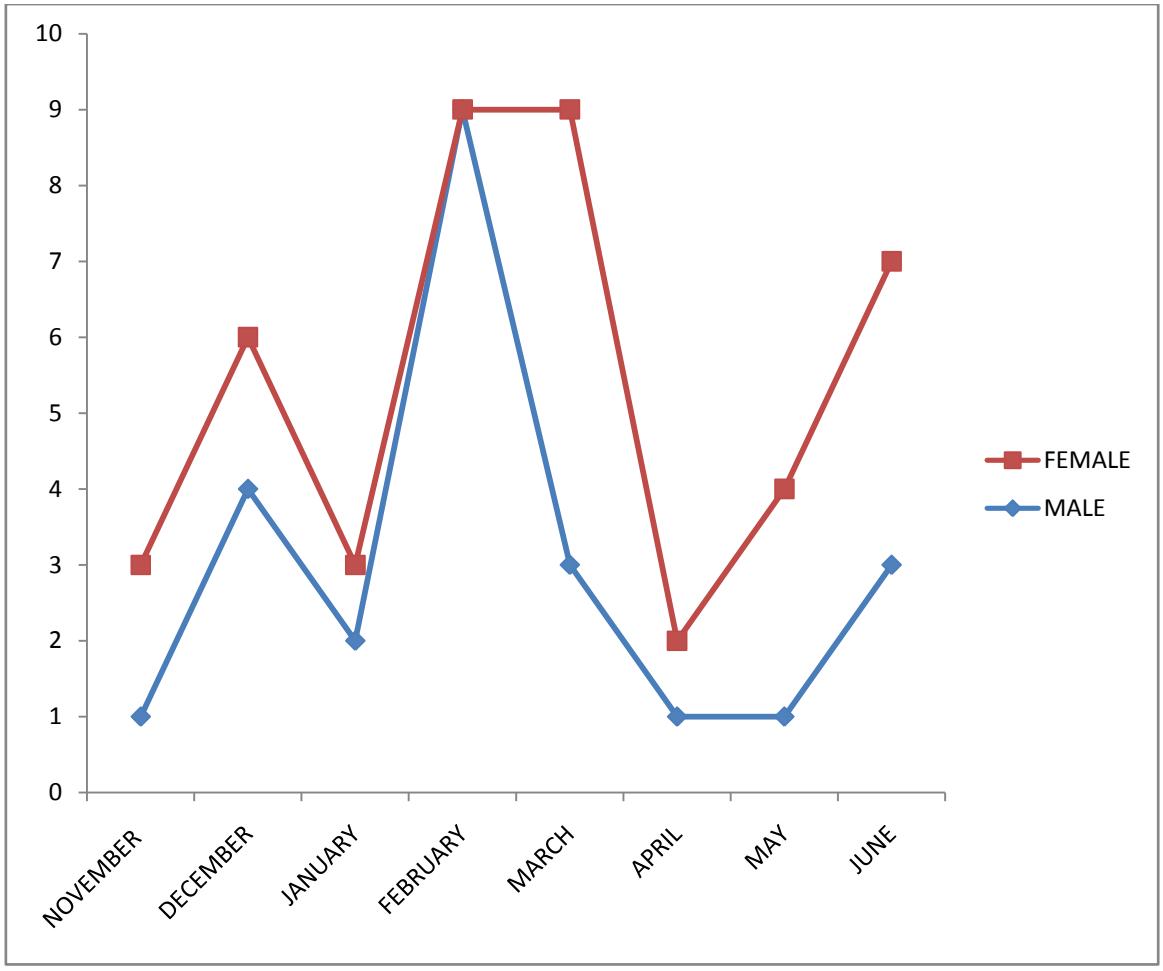


Figure: 18 Illustrate Percentage of Month Wise Distribution of Poison Death

In this study the results show that Maximum Number of Patients belongs to February and March in Months Wise. Female are the Major Victims.

MARITAL STATUS DISTRIBUTION

Table: 11 Showing Marital Status of Poison Death Cases

MONTH	MARRIED		UNMARRIED		DIVORCED		TOTAL
	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE	
November-16	1	2	-	-	-	-	3
December-16	4	1	-	1	-	-	6
January-17	2	1	-	-	-	-	3
February-17	6	-	3	-	-	-	9
March-17	3	5	-	1	-	-	9
April-17	1	-	-	1	-	-	2
May-17	1	2	-	-	-	1	4
June-17	3	2	-	1	-	1	7

Maximum Number of Patients belongs to Married followed by Unmarried.

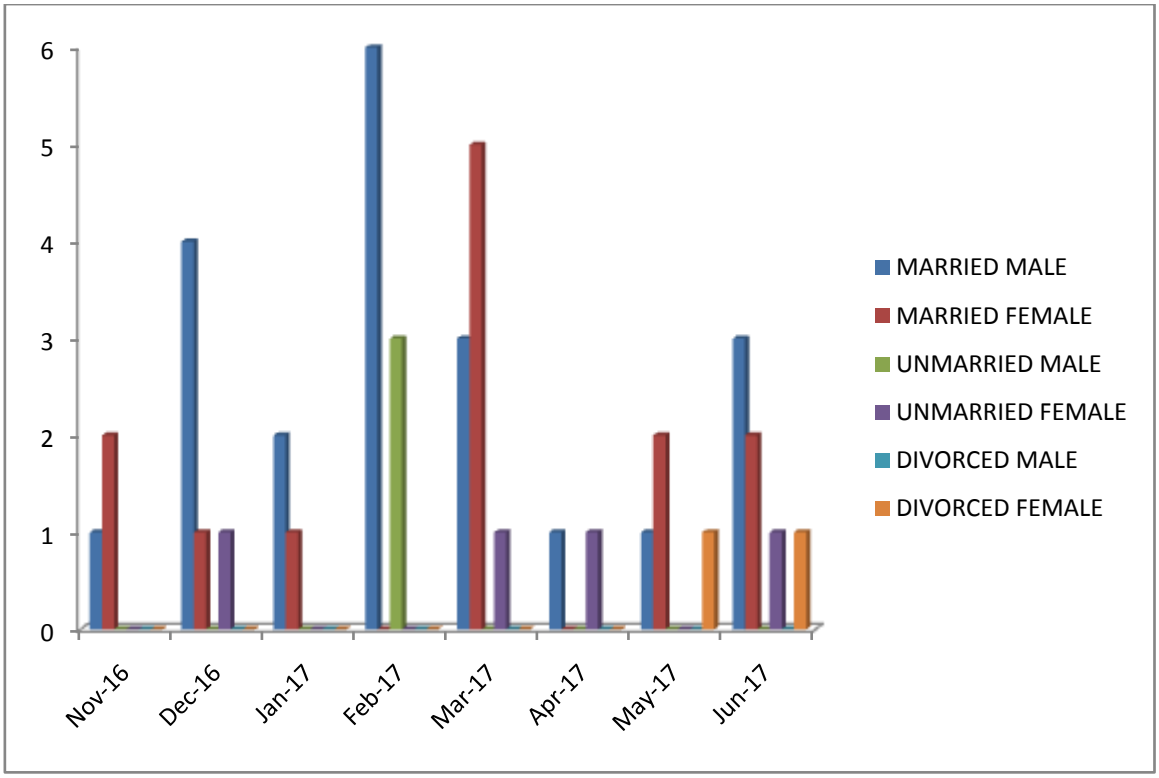


Figure: 19 Illustrate Percentage of Marital Status Distribution of Poison Death

In this study the results show that Maximum Number of Patients belongs to Married followed by Unmarried.

NATURE OF POISON

Table: 12 Showing Deaths Due to Nature of Poisoning Cases

TYPES OF POISONING	NO OF PATIENTS	PERCENTAGE%
Cow dung	14	31
Snake bite	7	16
Organophosphorous	7	16
Rat killer	5	11
Oleander	4	9
Tablet poisoning	3	7
Insecticide	2	4
Ant killer	1	2
Cockroach killer	1	2
Alcohol + OPC	1	2

Major Cause of Death is
Due to Cow Dung Poison.

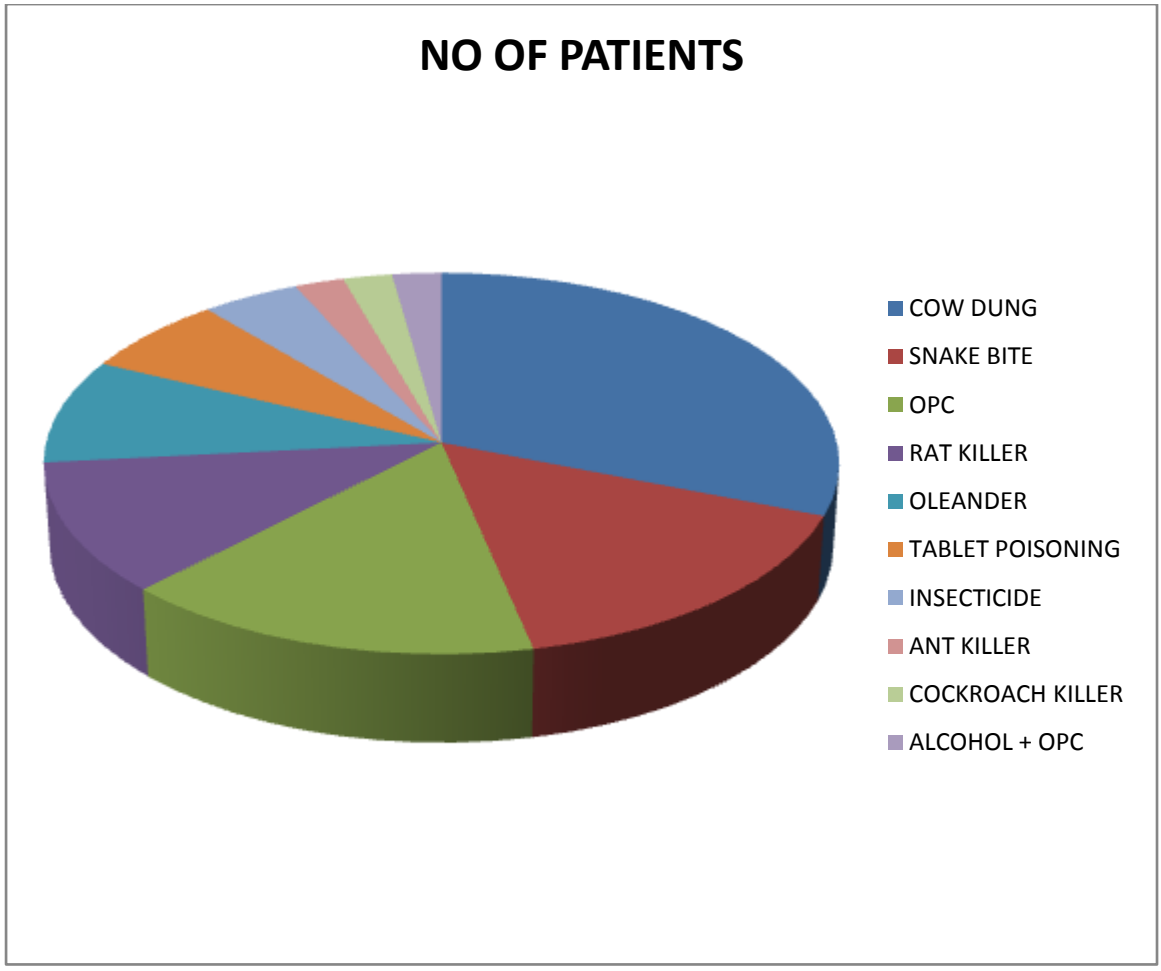


Figure: 20 Illustrate Percentage Death Due to Nature of Poison.

The Overall Death Poisoning Cases this study shows that Cow Dung (Auramine-O) was the Major Cause of Poison 14(31%) was followed by Snake Bite 7(16%) and OPC 7 (16%).

SUMMARY

From our study it has been observed that there is an Alarming Increase in Cases of Poisoning Mainly for Suicidal Purpose. **Cow dung (**Auramine-O) was found to be the Most Common Cause of Poisoning with High Mortality 68 (32%).

Males were the Major Victims and the most Cases were from rural areas. Poisoning is a common reason for presentation to Hospital & most Poisoned patients make a full recovery without specific treatment.

Cow dung powder is the first commonest Acute Poisoning cases for the hospital admission. Cow dung powder is available in two different colours:

i. Yellow powder (Auramine-O)

ii. Green powder (Malachite Green)

It is commonly used in Rural Tamil Nadu (South India) in the districts of Coimbatore, Erode and Tiruppur. Even though the Sale is Legally Banned, the Powder is easily available in grocery shops. It can cause gastrointestinal symptoms and persistent seizures sometimes

Pattern of poisoning in a region depends upon various factors such as availability, cost and access to toxic agents, socio-economic status, cultural and religious characteristics of people.

Cow Dung Powder has been so widely used, that the district authorities banned the sale of this product in 2007. However it is still widely available and there is no trend of a decrease in the incidence of Cow Dung Powder Poisoning during the study period. This underscores the fact that banning such substances, without educating the public or tackling the fundamental cause of deliberate self-harm will not succeed.

DEATH CASES:

From our study it has been observed that there is an alarming increase in Cases of Poisoning leads to Death, mainly the more number of Deaths is due to Cow Dung Poison and it's followed by Snake Bite and Organophosphorous Compound (OPC).

CONCLUSION

From our study it was revealed that

- Cow Dung was found to be most common cause of Poisoning with High Mortality.
- The most common Mode of Poisoning was Suicidal.
- Males were the Major Victims and Most Cases from Rural Areas
- For the diagnosis of Poisons Data will help the future Study of any kind of Poison Substance.
- The Most Death Cases Poisoning is found to Cow Dung Poison.
- Males were the Major Victims and Most Death Due to Poison.

BIBLIOGRAPHY

1. Olson KR, B Anderson, Thomas E. Poisoning and drug over dose.1990; 1: 65.
2. Doull J Klaassen C, Amdur M. (cds) In: Casarett and Doull's Toxicology. The Basic Science of Poisons 1990; 3: 27.
3. http://www.britannica.com/science/poison-biochemistry/types-of-poison.
4. Dr. Indu A. Chadha, Poisoning, Indian Journal Anaesthia 2003; 47(5): 401- 403.
5. Hishan MD, Vijayakumar A, Rajesh N, Sivakumar MN. Auramine-O and Malachite Green Poisoning: rare and fatal. Indian Journal Pharmacy Practice 2013; 6: 72-4.
6. Krishnamoorthy A, Subramanian R, Dhanaselvi P, Prabhu RS, Jayanthi V. clinical presentation of cow dung powder poison-A preliminary communication. Journal Association Physicians India 2001; 49.
7. Gargi J, Tejpal HR. A Retrospective Study of Poisoning in the Northern Region of Punjab. J Punjab Acad Offorensic Med Toxicol 2008; 8: 17-20.
8. El Naggar ARM, El Mahdy NM. Zinc Phosphide Toxicity with a Trial of Tranexamic Acid in its Management. J Adv Res 2011; 2: 149-156.
9. Brent J, Wallace KL, Burkhart KK. Phosphorous In: critical care toxicology – diagnosis and management of the critically poisoned patient. Philadelphia, PA: Elsevier, Mosby; 2005. P. 851-61.
10. Cruz LS, Vargas R, Lopes AA. Snakebite Envenomation and Death in the Developing World. Medical journal 2009; 45-50.
11. Shibendhu G, Prabuddha M, Tanmoy C: Management of Snake Bite in India: Journal of the Association of Physicians of India 2016; 64: 11-15.
12. Jortani, Saeed A, Helm, R Allen, Valdes, Poland, Inhibition of Na,K-ATPase by Oleandrin & Oleandrigenin and their detection by Digoxin Immuno assays clinical chemistry: 1996: 42(10): 1654-1658:

13. Timbrell J.A, Principles of Biochemical Toxicology New York: Informa health care: 349-351.
14. htpp://www.cancer.org/treatment/treatmentsandsideeffects/complementaryandalternativemedicine/herbovitaminandminerals/oleander-leaf.
15. Warfil, Zorob O, Alkatheeri N, Alawadhi A, "A Fatal use of Oleandrin Poisoning", Forensic science international. 179 ;(2-3): 31-36.
16. Nerium Oleander L, IPCS Inchum 2005;10:23.
17. Fagerland, Richard, "Ant chalk could Harm People more than Ants, San Francisco chronicle 2006; 12: 09.
18. Building Maintenance- Structural Integrated Pest Management California Department of general services 2007; 02: 11.
19. Insecticide chalk 2013; 07:10.
20. Steven A, Yawson, Alexander E. Field Caugth Permethrin Resistant Jour Pharm Sci 2008; 4: 11.
21. Doi H , Kikuchi H, Murai H, Kawano Y, Shilgets H. "Motor Neuron Disorder Simulating ALS induced by Chronic Inhalation of Pyrethroid Insecticides Neurology, 67(10): 1894-1895.
22. Bowaman B, Sereda and H.M, Meinhardt H. Simultaneous Presence of DDT and Pyrethroid Residues in Human Breast Milk from a Malaric Endemic Area in South Africa, 144(3); 902-917.
23. Hodgson, Ernest. A Textbook of Modern Toxicology 2010; 5: 108.
24. R Baselt. Deposition of Toxic Drugs and Chemicals in Man 2008; 8: 389-391.
25. Wright N. An Assessment of the Unreliability of the history given by Self-Poisoned patients Clin Toxicology 1980: 16: 381-384.
26. Thomas C Corbridge, Patrick Murray. Toxicology in adults 1998; 99: 1473-1513.

27. Olson KR, Pentel PR, Kelley MT. Physical Assessment and Differential Diagnosis of the Poisoned Patient Medical Toxicology 1978; 2(1): 52-81.
28. Winter SD. Pearson R, Gabow PA. The fall off Serum Anion Gap. Arch Intern Medicinal 1990; 150: 311-13.
29. Oh MS, Carol HJ. Current Concept the Anion Gap, N England J Med 1977; 297: 814.
30. Gabow PA. Disorders associated with an altered Anion gap Kidney Intern 1985; 27: 472-83.
31. Gabow PA. Clay K. Sullivan JB. Organic acids in Ethylene Glycol Intoxication Ann Intern med 1986; 105: 16.
32. Leatherman J, Schmitz PG. Fever Hyper dynamic shock, and Multi-system organ failure A Pseudo-Sepsis Syndrome associated with Chronic Salicylate Intoxication 1991; 110: 1391-96.
33. Glasser L. Sternglaze PD. Combie J. Serum Osmolality and its applicability to Drug Overdose Am J clinical pathology 1973; 60: 695.
34. Winchester JF. Active methods for detoxification In Haddad LM Clinical management of Poisoning and Drug over Dose Philadelphia Saunders. 1990; 2.
35. Kent R Olson, Charles E Baker. Current emergency diagnosis and treatment, Appleton and large publication. Norwalk 1992; 30(4): 730-766.
36. Cope C. The Importance of Oxygen in Treatment of Cyanide Poisoning 1961; 12: 109.
37. Collee GG, Hanson GC. The Management of Acute Poisoning Br J Anaesth 1993; 70: 562-73.
38. Hoffman Rs, Gold frank LR. The Poisoned Patient with altered Consciousness Controversies in the Use of a “Coma Cocktail” 1995; 274: 562.
39. Gold frank LR. The Several Uses of Naloxone ann emerg med 1984; 30:105.

40. Hanson GC. The management of acute Poisoning Br J Aneasth 1993: 70: 563-74.
41. Murat S, Muhammad G. Intensive Care Management of Insecticide Poisoning Critical Care 2001; 5(4): 211-215.
42. Albertson TE. Superiority of Activated Charcoal alone Compared with Ipecac and Activated Charcoal in the Treatment of Acute Toxic Ingestions an Emergency Medicine 1989; 18:56.
43. Gazelle S. Extracorporeal Techniques in the Treatment of Exogenous Intoxication Kidney International 1988; 33: 56.
44. Kulling P, Persson H. The Role of the Intensive care Unit in the Management of Poisoned Patient Medicinal Toxicology 1986; 1: 375-86.
45. M.G.Rajanandh, S Santhosh. Retrospective Assessment of poisoning cases in a Multi Speciality Hospital Journal of Pharmacology and Toxicology 2014; 9(2): 105-109.
46. O Gambhir Singh , A M Singh. Trends of poisoning cases in Melmaruvathur Region of Tamil Nadu A Retrospective study of 3 years International Archives of Integrated Medicine 2014; 1(4): 27- 31.
47. B Maharani, N Vijayakumar. Profile of poisoning Cases in a Tertiary Care Hospital, Tamil Nadu India Journal of Applied Pharmaceutical Science 2013; 3(01): 091-094.
48. Shreya M Shah, Pratik D, Asari, Anand J. Amin. Clinic-Epidemiological Profile of Patients Presenting with Acute Poisoning Int J Cur Res Rev 2016; 8(1): 35-41.
49. A Muruganathan, M Venkatesh. Yellow Cow Dung Powder (Auramine Poisoning) – Study of Clinical Profile and Strategies of Management IOS Journals of Dentals and Medical Sciences 2017; 16(5): 54-59.
50. D K Suneetha, J Inbanathan. Profile of Rat Killer Poisoning Cases in a Tertiary Care Hospital at Mysore International Journal of Scientific Study 2016; 3(12): 264-267.

51. Dr Achinta M, Dr Abhay C. Clinic-Epidemiological Profile of Poisoning in Children in a Rural Based Medical College and Hospital, IOSR Journal of Dental and Medical Sciences 2016; 15(4): 50-55.
52. C Arulmurugan, Sarfaraz A, Mohammed G. A Retrospective Study of Paradigm and Outcome of Acute Poisoning Cases in a Tertiary Care Teaching Hospital in Southern India International Journal of Research in Medical Sciences 2015; 3(10): 2654-2657.
53. K Vivekanandan, Bhavya E. A Study on Poison Cases and Their Management along with Poison awareness Educational Strategies Asian Journal of Pharmaceutical and Clinical Research 2012; 5(2): 71-73.
54. N malangu, G A Ogunbanjo. A Profile of Acute Poisoning at Selected Hospitals in South Africa S A Journal Epidemiology Infect 2009; 24(2): 14-16.
55. Dr Avinash, Dr Navin Puttum. Sani Powder Poison-Legally Banned International Journal of Scientific and Research 2016; 6(9), 101-109.
56. Subash V, Md Fareedullah. Current Review on Organophosphorous Poisoning Archives of Applied Science Research 2010; 2(4): 199-215.
57. Antony J, Celine TM. A Profile of Acute Poisoning Retrospective Study J Sci Soc 2015; 42: 156-60.
58. Brahmankar TR, Sharma SK. A Record Based Study of Frequency and pattern of Medico-Legal Cases Reported at a Tertiary Care Hospital in Miraj Int J Community Med public health 2017; 49(13): 48-52.
59. Jaiprakash H, Sarala, Venkatarathnamma P, Kumar T. Analysis of Different Types of Poison in a Tertiary Care Hospital in Rural South India Food and chemical toxicology 2011; 49(1): 248-250.

60. Linto Mathew, Thomas, Prabhu. A Retrospective Study on Pattern of Poisoning Cases in a Tertiary Care Hospital World Journal of Pharmacy and Pharmaceutical Sciences 2016; 05(22): 1860-1865.
61. Natrajan UM, Natrajan M. A Study of Snake Bite Patients with reference to time of delay in Management J App Med Sci 2016; 01(4): 85-88.
62. Gupta P, Kumar A, Singh SP, Prakash M, Gupta M, Kumar P. Pattern of Cases of Acute Poisoning in Rural Tertiary Care Center in Northern India Jour Community med 2016; 7(4): 307-310.
63. Singh RR, Kumar A, Uraiya D, Dhaon P, Retrospective Analysis of Poisoning Cases admitted in a Tertiary Care Hospital in North Eastern India Intl Jour Med Res 2016; 4(7): 1172-1177.
64. Asari PD, Shah SM, Amin AJ, Patel ND. Drug Utilization Study on Acute Poisoning Cases admitted in a Tertiary Care Hospital in Northern Eastern India Intl J Med Res 2016; 4(7): 1172-1177.
65. Aditya P S, Tushar KM. Socio-Demographic Characteristic of Poisoning Cases in a Tertiary Care level Hospital of West Bengal Indian Journal of Basic and Applied Medical Research 2015; 4(2): 476-482.
66. Prayag A, Ashtagi GS, Mallapur MD. Pattern of poisoning Cases at a Tertiary Health Care Center Belagavi Intl J Med Sci Public Health 2015; 5: 1698-1701.
67. Baraka G, Kaushal K, Pooja R. Retrospective Study of Poisoning Cases at Tertiary Care Hospital in Western Uttar Pradesh J Indian Acad Forensic Med 2016; 38(01): 71-73.
68. Naresh D, Shailendra, Subbartnam Y, Prasuna G, K Rajashekar, Mary R. “Profile of Poisoning Cases in a Tertiary Care Hospital Telangana India” Journal of Evidence Based Medicine and Health Care 2015: 2(20): 3032-3038.

69. Koulapur V, Pujar S, S Honnungar, Jirli P, Patil S. Epidemiological Profile of Pesticide Poisoning Cases in Bijapur, Karnataka in South West India International Journal of Medical Toxicology and Forensic Medicine 2015; 5(4): 180-182.
70. Owasis K, Khan I. Acute poisoning - Ethiological Agents and Demographic Characteristics in Patients Coming to ER of a Tertiary Care Hospital Professional Med J 2015; 22(12): 1591-1594.
71. Mukul J, Divyeshkumar V P. A Study on Clinical Profile of Patients with Acute Poisoning J Med Sci 2015; 4(2): 1-10.
72. Virendra M, Prasad W. Acute Poisoning Cases in Navi Mumbai Journal of Medical Sciences 2015; 2(3): 120-124.
73. Farhana B, Basharata S. Socio-Demographic Poisoning Cases in a Tertiary Care Hospital J Med 2014; 3(6): 1-4.
74. Risal A, Sharma PP, Karkib R. Psychiatric Illnesses Among the Patients Admitted for Self-Poisoning in a Tertiary Care Hospital of Nepal Journal of Advances in Internal Medicine 2013; 02(01): 10-13.
75. Adepu R, Churi S, Jesslin J. Assessment of Prevelance and Mortality Incidences Due to Poisoning in a South Indian Tertiary Care Teaching Hospital Indian Journal of Pharmaceutical Sciences 2010; 72(5): 587-589.

PATIENTS PROFORMA

NAME: AGE:

SEX: IP NO:

ADDRESS:

CHIEF COMPLAINTS:

PAST MEDICAL HISTORY:

PERSONAL HISTORY:

SOCIAL HISTORY:

PHYSICAL EXAMINATIONS:

BP:

TEMP:

PR:

RR:

SYMPTOMS:

NAME & QUANTTITY OF THE COMPOUND:

MODE OF INTAKE:

DATE & TIME OF CONSUMPTION:

INGESTION OF POISON WITH ALCOHOL INTAKE: YES / NO

PAST MEDICATION CHART:

T.NAME	**G.NAME**	**DOSE**	**FREQ**	**DATE**					

ஒப்புமை படிவம்

பங்கேற்பாளர் ஒப்புதல்

என் பங்களிப்பு தன்னார்வமாக இருப்பதை நான் புரிந்துகொள்கிறேன், எந்தவொரு காரணமும் இன்றி இந்த ஆய்விலிருந்து நான் விலகி இருக்கலாம் அல்லது எந்த குறிப்பிட்ட கேள்வியையும் ஆய்வு செய்ய மறுக்கிறேன். எனது அடையாளம் வெளிப்படுத்தப்படவில்லை எனில், என்னுடைய பதில்களை அணுகவும் முடிவுகளை வெளியிடவும் ஆய்ந்துள்ள உறுப்பினர்களை நான் ஒப்புக்கொள்கிறேன். ஆய்வில் பங்கேற்க நான் தானாகவே ஒப்புக்கொள்கிறேன் .

பங்பேற்பளார் கையொப்பம் மற்றும் தேதி