A DISSERTATION ON

A PROSPECTIVE STUDY OF EPIDEMIOLOGICAL, CLINICAL, TOXICITY, OUTCOME OF PATIENTS ADMITTED WITH HISTORY OF ACUTE DRUG POISONING IN A TERTIARY CARE CENTRE.

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M.D. DEGREE BRANCH - I: GENERAL MEDICINE

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CERTIFICATE OF GUIDE

This is to certify that the dissertation titled "

A PROSPECTIVE STUDY OF EPIDEMIOLOGICAL, CLINICAL, TOXICITY, OUTCOME OF PATIENTS ADMITTED WITH HISTORY OF ACUTE DRUG POISONING IN A TERTIARY CARE CENTRE.

submitted by Dr. R.N. MADHURIMA appearing for M.D. GENERAL MEDICINE degree examination in May 2023, is a bonafide record of work done by her, under my guidance and supervision in partial fulfilment of requirements of The Tamil Nadu Dr. M.G.R Medical University, Chennai. I forward this to The Tamil Nadu Dr. M.G.R Medical University, Chennai.

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CERTIFICATE - II

This is to certify that this dissertation work titled "**A PROSPECTIVE STUDY OF EPIDEMIOLOGICAL, CLINICAL, TOXICITY, OUTCOME OF PATIENTS ADMITTED WITH HISTORY OF ACUTE DRUG POISONING IN A TERTIARY CARE CENTRE**"of the candidate DR. R.N .MADHURIMA with registration Number 200120100525 for the award of the degree of M.D. in the branch of Internal Medicine. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 2 percentage of plagiarism in the dissertation.

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DECLARATION

I, **Dr. R.N.MADHURIMA,** certainly declare that this dissertation titled", **A PROSPECTIVE STUDY OF EPIDEMIOLOGICAL, CLINICAL, TOXICITY, OUTCOME OF PATIENTS ADMITTED WITH HISTORY OF ACUTE DRUG POISONING IN A TERTIARY CARE CENTRE"**, represent a genuine work of mine, done at the Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, under the supervision of **Prof.DR.S.USHA LAKSHMI M.D** Chief **and** Professor,**Prof.Dr.RAGHUNANDHANAN M.D** Chief and Professor Madras Medical College and Rajiv Gandhi Government General Hospital.

I, also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other university board, either in India or abroad.

This is submitted to The Tamil Nadu Dr. MGR Medical University, Chennai in partial fulfilment of the rules and regulations for the award of. M.D General Medicine

Dr. R.N.MADHURIMA

Date: 20/12/2022

Place: **Chennai**

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ABSTRACT

Background and objectives:

Our study aimed at

- To know the epidemiological profile of patients admitted in our hospital with history of
- Acute tablet poisoning.
- To study the various clinical presentations,haematological and biochemical , radiological abnormalities associated with the outcome of the patient

Materials and Methods:

After selecting patients as per inclusion and exclusion criteria, detailed history regarding type of drug intake ,quantity of consumption time of consumption,intent suicidal or accidental,demographic data and clinical presentations .

Blood investigations like complete blood picture,renal function tests,liver function tests,serum electrolytes,coagulation profile,chest X-ray, ecg , ABG, urine analysis, toxic drug analysis and their reports were included in this study.outcome of the various drug poisonings were recorded.

RESULTS:

In the present study 80 patients admitted and studied over a period of 6 months.

Majority of participants were between 31 - 40 yrs.

Majority of patients were from lower middle class and studied Above high school.

Among the total study participants, BENZODIAZEPINES are the most common drug poisoning, followed by Non steroidal anti inflammatory drugs and anti hypertensives.

Mean duration of stay in hospital of study participants was 4-7 days.

All the study participants were recovered and discharged.

CONCLUSION:

Patients hospitalised with acute drug poisoning have relatively good short term outcomes.In the long term however ,these patients have a high rate of re admissions due to drug poisonings.

Most of the adolescents hospitalised due to acute self poisoning were likely to be depressive and had repeated self harm.

There is significant association noted between time since consumption, comorbidities, class of drug taken , specific antidote and organ failure.

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1. INTRODUCTION

"**POISONS AND MEDICINES ARE OFTEN TIMES SAME SUBSTANCES GIVEN WITH DIFFERENT INTENTS"** (Peter mare Latham (1789 -1875))

The patients with acute drug poisoning are relatively common in hospitals, usually require relatively simple care and have good short-term outcome. However, some of these patients are at risk of acute morbidity and poor long-term outcome.

In most cases the needed care is symptomatic, the hospital length of stays are less than 3 days and primary outcome is good. The substances used in self poisoning vary depending on area and the culture.

Typical agents used in self poisoning include pharmaceutical products, mostly psychoactive drugs and paracetamol, pesticides, rodenticides, household chemicals, and illegal drugs. Alcohol intake is often associated with self poisonings.

Patients admitted to hospital due to acute self induced drug poisoning present various types of Motivation. Some poisoning accidental, mostly among children or accidents in recreational use and in self medication.

Patients admitted to hospital due to hospital often have previous admissions due to self poisoning.

It has been stated that it is difficult to recognise patients risk for both increased short and long term morbidity and mortality. This is because the reported hospital stays are short, which complicates screening and because of patients unreliable medical history.

It seems that it is essential to prevent deaths due to acute morbidity during hospital stays, as well as later in life, since acute mortality due to acute drug poisonings is low among hospitalized patients without complications.

The present study was undertaken to evaluate factors related to both short term and long term outcomes in order to obtain information for improving care.

The study doesn't cover poisoning due to toxic alcohols, herbal, animal, carbon monoxide, herbicides, rodenticides, pesticides. The study hypothesis is that at risk patients can be identified and that management of these risks may have an impact on the outcome.

2. REVIEW OF LITERATURE

Definitions:

Poisoning is a condition in which a substance that is injurious to physical health or can cause death is taken orally, inhaled, transdermally or parenterally. Normally used substances such as water can be poisonous depending on the amount ingested.

Acute drug poisoning is the most common form of deliberate self harm.

Deliberate self harm is an act where by an individual has the intention to cause physical or psychiatric harm to himself or herself but not necessarily with suicidal intent.

Drug poisoning is a condition where substances that are normally used in medical practice are ingested in an inappropriate manner, usually in deliberate self harm, or recreational use.

Self poisoning is a condition in which an individual takes a poisonous substance, either accidentally or with suicidal or recreational intent.

Intoxication is a condition that may follow ingestion of a poison or an inappropriate use of a drug and can be described as an altered level of consciousness, mental status or physiology.

ACTION OF POISONS :

I) LOCAL: The local action by coming in direct contact with part. (1) Chemical destruction by corrosive.

2) Irritant will produce inflammation and congestion.

3) Effects on motor and sensory nerves, e.g., tingling of skin and

tongue by aconite, dilation of pupils by belladonna or datura.

REMOTE:

Remote action produced either by corrosives causing severe pain followed by shock(reflex)or by poison being first absorbed into the system through the blood, and excreted through tissue will produce organ damage. e.g., cantharides acting on kidneys produces nephritis, nux vomica.

COMBINED :

Drugs like carbolic acid, oxalic acid, phosphorus, etc., have local and remote actions.

3. CAUSES MODIFYING ACTION OF POISONS

I) **QUANTITY**:

More the quantity, more severe are the toxic effects.

A large quantity of poison taken orally may cause excessive vomiting, causing its rapid elimination and decreased toxicity e.g., alcohol, CUSO4.

Action can be modified by varying the dose significantly more amount of arsenic produce death without causing noticeable symptoms by shock.

II) **FORM**:

PHYSICAL STATE: Poisons act most rapidly when gaseous and less when liquid. In case of solids, the action depends on their solubility.

BY CHEMICAL MIXATION:

Based on the mixing ability of the poisonous substances with one another action of a poison varies. This chemical combination of poisons, HCL &AgNO3 when combined together form un dissolvable salts of Agcl2 which is harmless.

Some poisons are not soluble in water, they become soluble in H2O and also mixes with acid in the stomach and being dissolved in to the blood like copper arsenate and lead carbonate.

C) **MECHANICAL COMBINATION:**

When mixing of poison with inactive or inert substance may produce alteration of poisons like using very small amount /increased concentration of acid producing corrosion when same amount mixed with large quantity of water decrease become harmless.

III) MODES OF ADMINISTRATION OF VARYING POISON:

The fastness of the action is in the order described under routes of administration.

As rough guide if the active dose by the mouth is considered as unit, The rectal dose is about one-and half to tow, and the hypodermic dose about one-fourth.

A lethal dose is usually ten or more times the maximum medicinal dose.

The rate of absorption from the alimentary canal is variable. The stomach when empty without food it absorbs more quickly than the filled with substances, also the content of stomach ' nature is in favour of dissolving the poison like when you take phosphorous poison and drink oil following it.

This will enhance the action of phosphorous by increasing its absorption. Gastro enterostomy hastens the entry of poisons into the small bowel. Sleep, narcosis and trauma causing gastrointestinal stasis will retard it.

Retardation during gastrointestinal absorption, dilution and alteration during digestion. The skin is on the whole a bad absorptive organ.

CONDITION OF THE BODY (A) AGE :

Age has a considerable effect upon the dosage of drugs. Underdeveloped enzyme system in children less than two years, more susceptible to the effect of most drugs.

There are some drugs of which children can take more than their proportionate dose, e.g., mercury and belladonna.

There are some of which they cannot take even a proportionate dose e.g. mercury and belladonna. There are some of which they cannot take even a proportionate dose e.g: Morphine.

IDIOSYNCRASY:

It may be defined as the inherent personal hypersensitivity to the agent in question. Certain people are sensitive for certain drugs and even articles of diet e.g., shellfish, eggs and fruit.

The symptoms usually occur in the skin as an urticaria, but may be of more general nature with dyspnea, rigors, fever, diarrhoea, haemorrhage from the bowel and albuminuria.

Fatal cases are comparatively rare, but symptoms may be alarming or dangerous, iodine, bromine, opium, belladonna, cocaine, aspirin, penicillin, and mercury are common examples of drugs to which many people are allergic.

C) HABIT:

Habituation will change the certain drug effects in the body. Tolerance is the organism capability in showing decreased response to a chemical substance in a specified dose when compared to its same action when giving on the same specified dose.

Opium preparations frequently taken, lose much of their effect after a time, and require to be administered in increased doses. Addicts can tolerate quantities of the drug which would endanger life if they had been initial does. Tolerance is seldom a natural phenomenon.

The same effect of habit occurs from the use of tobacco, alcohol, cocaine, morphine and other alkaloids, It is more usually a feature of natural substances, less of synthetic drugs, such as barbiturates, chloral, etc.

D) STATE OF HEALTH : A unhealthy person not tolerates than healthy. General debility, senility, chronic or disabling disease may cause death of a person to a dose that is ordinarily safe, e.g. CO may kill at a blood saturation of only 25 to 30 % .

Some substances can be used in certain diseases /conditions with no harm. But when these substances are used in other conditions, even smaller quantity can be poisonous.

SLEEP AND INTOXICATION: When a person is taking poison along with alcohol or any other substance intoxication or immediately going to sleep after consuming poison ,time of action of the poisoning gets delayed.

F) CUMULATIVE ACTION : Poisons which are eliminated slowly may accumulate in the body when given in repeated doses for a long time and may ultimately produce symptoms of poisoning.

4. EPIDEMIOLOGY

INCIDENCE OF HOSPITALISATIONS:

Not all contacts with the health care system due to acute poisoning lead to hospital admission. The proportion of patients treated by ambulance service or solely on emergency departments varies depending on the area.

AGENTS USED IN ACUTE DRUG POISONING

The most commonly used agents in acute drug poisonings include psychotropic drugs and analgesics, including opioids and non opioid analgesics (paracetamol, NSAIDS). Medical products for neurological or cardiovascular diseases play a minor role In acute poisonings.

TABLE:1:POISONS SYNDROMES (TOXIDROMES) [13]

TOXIDROME	VITAL SIGNS	MENTAL STATUS	PUPILS	SKIN	BOWEL SOUNDS	OTHER	POSSIBLE TOXINS
Sympatho mimetic	Hypertension, tachycardia, Hyperthermia	Agitation, psychosis, delirium, violence	Dilated	Diaphoretic	Normal to increased		Amphetamines, cocaine, PCP, bath salts, ADHD medication
Anti cholinergic	Hypertension, tachycardia, hyperthermia	Agitated, delirium, coma, seizures	Dilated	Dry, hot	Diminished	Ileus ,urinary retention	Antihistamines, tricyclic antidepressants, atropine, jimson weed
Cholinergic	Bradycardia BP and temp typically normal	Confusion, coma, fasciculations	Small	Diaphoretic	Hyperactive	Diarrhea, urination, bronchorrhea, bronchospasm, emesis, lacrimatin, salivation	Organophosphates (insecticides, nerve agents) carbamates (physostigmine, neostigmine, phridostigmine) Alzheimer medications, myasthenia treatments
Opioids	Respiratory depression bradycardia, hypotension, hypothermia	Depression, coma, euphoria	Pinpoint	Normal to decreased	Normal to decreased		Methadone, buprenorphine, morphine, oxycodone, heroin, etc.
Sedative-hypnotics	Respiratory depression, HR normal to	Somnolence, coma	Small or normal	Normal	Normal		Barbiturates, benzodiazepines, ethanol
Serotonin syndrome(similar findings with neuroleptic malignant syndrome)	Hyperthermia, tachycardia, hypertension or hypotension (autonomic instability)	Agitation, confusion, coma	Dilated	Diaphoretic	Increased	Neuromuscular hyper excitability:clonus, hyper reflexia (lower extremities > upper extremities)	SSRIs, lithium, MAOIs, linezolid, tramadol, meperidine, dextromethorphan
Salicylates	Tachypnea, hyperpnea, tachycardia, hyperthermic	Agitation, confusion, coma	Normal	Diaphoretic	Normal	Nausea, vomiting, tinitus, ABG with primary respiratory alkalosis and primary metabolic acidosis; tinitus or difficulty hearing	Aspirin and aspirin-containing products, methyl-salicylate
Withdrawal (sedative-hypnotic)	Tachycardia, tachypnea, Hyperthermia	Agitation, tremor, seizure, hallucinosis, delirium tremens	Dilated	Diaphoretic	Increased		Lack of access to ethanol, benzodiazepines, barbiturates, GHB, or excessive use of flumazenil
Withdrawal (opioid)	Tachycardia	Restlessness, anxiety	Dilated	Diaphoretic	Hyperactive	Nausea, vomiting diarrhea	Lack of access to opioids or excessive use of naloxone

INTAKE MOTIVATION:

Acute drug poisonings can be accidental, overdoses in recreational use, deliberate self harm behaviour or suicide attempts. The most common motivation is usually deliberate self harm, including suicidal attempts, which ranges from 29-85% of the cases depending on the population.

Clinical characteristics:

Patients admitted to hospital due to acute self poisoning suffer the effects and side effects of the drugs that have been ingested. Typical symptoms are sedation, altered consciousness, comatose on admission.

Cardiac dysrhthmias are common, especially after ingestion of tricycling antidepressants. Miosis is typical in opioid intoxication.

Extrapyramidal symptoms may occur following intake of antipsychotics.

TOXIDROMES	SYMPTOMS AND SIGNS	EXAMPLES
α_1 Antagonists	CNS depression, tachycardia, miosis	Chlorpromazine, quetiapine, clozapine, olanzapine, risperidone
α_2 Agonist	CNS depression, bradycardia, hypertension (early), hypotension (late), miosis	Clonidine, oxymetazoline, tetrahydrozoline, tizanidin
Clonus/myoclonus	CNS depression, myoclonic jerks, clonus, hyperreflexia	Carisoprodol, lithium, serotonergic agents, bismuth, organic lead, organic mercury
Sodium channel blockers	CNS toxicity, wide QRS	Cyclic antidepressants and structurally related agent, propoxyphene, quinidine/quinine, amantadine, antihistamines, bupropion, cocaine
Potassium channel blockers	CNS toxicity, long QT	Butyrophenones, methadone, phenothiazines, ziprasidone

Table 3: Toxidromes

Table 2. Typical agents used in acute drug poisonings and clinical manifestations in acute poisoning.

Agent used in drug poisoning	Symptoms, vital signs
Benzodiatsepines	GABA-receptor mediated CNS depression low doses: sedation high doses: coma and respiratory arrest
Tricyclic antidepressants	Symptoms occur within 6 hours of ingestion including CNS depression, anticholinergic symptoms (mydriasis, fever, delirium, tachycardia, ileus, urinary retention), cardiovascular symptoms (prolonged QRS, QTc and PQ-interval)
SSRIs	Sedation, vomiting, cardiovascular symptoms in severe poisonings (prolonged QRS, QTc-interval, ventricular tachycardia)
Antipsychotics	Sedation, rigidity, seizures in severe poisoning NMS (hypethermia, rhabdomyolysis, rigidity)
β-Blockers	Cardiac β2-reseptor mediated bradycardia, reduced inotropia, atrio-ventricular and intra-ventricular conduction blocks
Paracetamole	0–24h from ingestion: nausea, womiting, diaphoresis, malaise 24–48h from ingestion: abdominal pain, elevated liver enzymes (>10 000 IU/l in transaminases) 48–96h from ingestion: liver failure with encephalopathy, coagulopathy, hypoglycemia
Opioids	Opioid-reseptor mediated CNS- and respiratory depression. Bradycardia, hypothermia, non-cardiogenic pulmonary odema
Amphetamines	Periferial release of cateclolamine and re-uptake inhibition of catecolamine and monoamine oxidation inhibition mediated CNS stimulation (Confusion, anxiety, mydriasis, tachycardia, arrhythmias, myocardial ischemia, hyperthermia, rhabdomyolysis)

Following table presents the symptoms of poisonings due to typical agents used in drug poisonings:

5. GENERAL MANAGEMENT OF DRUG POISONED PATIENTS:

Initial life support:

The care of drug poisoned patients consists of four elements: initial life support, decontamination, in cases antidotal therapy and enhanced elimination.

Initial life support consists of airway management and correction of circulatory status. Protecting the airway is essential in order to prevent aspiration and respiratory insufficiency due to lowered consciousness.

Arrhythmias and hemodynamic compromise has to be managed with any patient in critical condition.

The symptoms of the poisoning are unspecific, the medical history of the patient can be unreliable and in some cases such as in severe paracetamol poisoning,
Symptoms are delayed.

Decontamination:

Induced emesis:

Ipecac syrup induced emesis was previously the recommended method for gastric emptying as it may be less Traumatic than gastric lavation and may be performed effectively within one hour from ingestion.

Induced emesis is safe only in patients with normal consciousness. The use of ipecac induced emesis is not beneficial to the outcome.

It doesn't shorten the length of the stay or reduce mortality and probably increases the risk of it is no longer recommended.

GASTRIC LAVAGE:

Gastric lavage is a procedure that aims to empty the stomach using a large Oro gastric tube and lavation with saline. It is useful when a large amount of substance is ingested less than one hour ago.

Whole bowel irrigation:

Whole bowel irrigation is a technique that is used to increase gut motility, through which it decreases absorption and decontaminates the gut from the ingested substance.

A non absorbable poly ethylene glycol solution is administered via a nasogastric tube until the solution has gone through the gut.

Activated charcoal:

Activated charcoal is used in drug poisonings to prevent absorption.

Activated charcoal can adsorb both ingested substances and molecules from the biliary secretion decreasing the toxin concentration in the enterohepatic cycle.Multiple dose Activated charcoal is recommended to use in certain conditions and it can be useful in carbamazepine or theophylline poisonings.

Procedures to enhance elimination:

Alkaline dieresis:

Forced diuresis is used to enhance the elimination of substances excreted renally. Forced alkaline diuresis may be beneficial in salicylate ,methotrexate, or phenobarbital poisonings . Increased urine pH with normal urine output can be safer technique to enhance the elimination of acidous substances.

EXTRACORPOREAL METHODS:

EXTRACORPOREAL methods such as hemodialysis, hemodiafiltration and hemoperfusion can be used to enhance the elimination of toxins.

Hemodialysis or hemodiafiltration can be used to improve clearance of water soluble low protein binding substances.

Haemodiafiltration is better than hemodialysis for clearing higher molecular weight compounds, but the clearance with hemodialysis is usually higher.

Antidotes

Several antidotes are used in acute poisonings such as naloxone in opioid poisoning, flumazenil in benzodiazepine poisonings and digoxin specific antibodies in digitalis poisoning.

A coma cock tail is a diagnostic and sometimes therapeutic tool that includes the administration of naloxone, glucose and thiamine in suspected or known acute drug poisoned patients.

TABLE:4: SPECIFIC ANTIDOTE FOR POISON

POISON	ANTIDOTE	DOSAGE	ROUTE	ADVERSE EFFECTS, WARNINGS, COMMENTS
Acetaminophen	N. Acetyl cysteine (Mucomyst) N. Acetyl cysteine (Acetadote)	140 mg/kg loading, followed by 70 mg.kg q4h 150 mg/kg over 1 hr, followed by 50mg/kg over 4 hr, followed by 100mg/kg over 16 hr	PO IV	Vomiting (patient-tailored regimens are the norm) Anaphylactoid reactions (most commonly seen with loading dose) (Higher doses of the infusion are often recommended depending upon the acetaminophen level and the degree of injury)
Anticholinergic	Physostigmine	0.02 mg/kg over 5 min; may repeat q5- 10min to 2 mg max	IV/IM	Bradycardia, seizures, bronchospasm Note: Do not use if conduction delays on ECG
Benzodiazepines	Flumazenil	0.2 mg/kg over 30 sec; if response is inadequate, repeat q1 min to 1 mg max	IV	Agitation, seizures; do not use for unknown ingestions
Blockers	Glucagon	0.15 mg/kg bolus followed by infusion of 0.05-0.15 mg/kg/hr	IV	Hyperglycemia, vomiting
Calcium channel blockers	Insulin	1 unit/ kg bolus followed by infusion of 0.5-1 unit/kg/hr	IV	Hypoglycemia Follow serum potassium and glucose closely

The acute medical history of drug poisoned patients is unreliable. Therefore, the use of antidotes in cases other than patients with definite diagnosis of certain poisoning may be harmful and cause complications such as seizures in tricyclic anti depressant poisoning in flumazenil administration.

Paracetamol is a typical agent used in drug poisonings. In overdose, It's toxic metabolite n-acetyl p benzoquinonimine production exceeds the elimination, which depends on the amount of glutathione in hepatic cells. N -acetyl cysteine replaces glutathione and enhances the detoxification of NAPQI.

Ethylene glycol, methanol	Fomepizole	15mg/kg load; 10mg/kg q12hx 4 doses; 15mg/kg q12h until EG level is <20 mg/dL	IV	Infuse slowly over 30 min; If fomepizole is not available, can treat with oral ethanol (80 proof)
Iron	Desferoxamine	Infusion of 5-15 mg/kg/hr (max: 6 g/24hr)	IV	Hypotension (minimized by avoiding rapid infusion rates)
Isoniazid (INH)	Pyridoxine	Empirical dosing : 70 mg/kg (max dose = 5g)	IV	May also be used for mushroom ingestions
Lead and other, heavy metals (e.g., arsenic, inorganic mercury)	BAL (dimercaprol)	3.5 mg/kg/dose q4hr, for the 1st day; subsequent dosing depends on the toxin	Deep IM	Local injection site pain and sterile abscess, vomiting, fever, salivation, nephro toxicity Caution: prepared in peanut oil; contraindicated in patients with peanut allergy
	Calcium disodium EDTA	35-50 mg/kg/day x 5 days; may be given as a continuous infusion or 2 divided doses/day	IV	Vomiting, fever, hypertension, arthralgia, allergic reactions, local inflammation, nephro toxicity (maintain adequate hydration, follow UA and renal function)
	Dimercaptosuccinic acid (succimer, DMSA, Chemet)	10 mg/kg/dose q8h x 5 days, then 10mg/kg q12hx 14 days	PO	Vomiting, hepatic transaminase elevation, rash

	Calcium salts	Dose depends on the specific calcium salt	IV	
Carbon Monoxide	Oxygen	100 % FIO2, via non-re breather mask (or ET if intubated)	Inhalation	Some patients may benefit from hyperbaric oxygen (see text)
Cyanide	Cyanide kit: Amyl nitrate	1 crushable ampule; inhale 30 sec of each min	Inhalation	Methemoglobinemia
	Sodium nitrate	0.33 mL/kg of 3% solution if hemoglobin level is not known; otherwise, based on tables with product	IV	Methemoglobinemia Hypotension
	Sodium thiosulfate	1.6 mL/kg of 25% solution; may be repeated q30-60min to max of 50 Ml	IV	If inducing methemoglobinemia is contraindicated; consider only using the thiosulfate component of the kit
	Hydroxocobalamin (Cyanokit)	70 mg/kg (adults: 5 g) given over 15 min	IV	Flushing/ erythema, nausea, rash, heamaturia, hypertension, headache
Digitalis	Digoxin-specific Fab antibodies (Digibind; DigiFab)	1 vial binds 0.6 mg of digitalis glycoside; # vials = digitalis level x weight in kg/100	IV	Allergic reactions (rate), return of condition being treated with digitalis glycoside

6. CLINICAL MANAGEMENT:

Pre hospital care can be classified to basic life support and advanced life support. Basic life support includes for example non invasive airway management using oxygen mask and lateral decubitus position to protect airway and oral or rectal medications. ALS includes invasive airway management, intravenous fluids and medications.

INTENSIVE CARE:

The need for intensive care treatment in acute drug poisoned patients is mainly to stabilize the patients physiology, for special treatment to eliminate the substance that requires the intensive care setting and to manage the complications of poisoning such as pulmonary and cardiovascular complications. The most common organ dysfunction is an altered level of consciousness.

Risk factors:

Depression, low self esteem, and lower social class have been reported as risk factors for deliberate self harm behaviour.

The most common cause of poisoning leading to hospitalisation was acute ethanol poisoning associated with recreational use. Adolescents with possible or evident contributing factors before poisoning presented more typical Deliberate self harm behaviour, including intentional intake of substances, and more frequent poisonings due to pharmaceutical products.
Impulsive behaviour seems to associated with age and the acute contributing factors.

The triggers for self poisoning in the adult population were the need to escape stressful situations and the need for care or attention.

Prevention:

Primary prevention:

The primary prevention of drug poisonings consists of laws regarding product safety, devolopment of safer drugs, enhanced knowledge and suicide prevention.

In primary prevention of suicides, it is important to recognize patients in advantage and provide good mental health care.

Secondary prevention:

Patients admitted to hospital due to acute drug poisoning face high long term risk of death for both natural and unnatural causes of death. Suicides are common among these patients. Patients with acute drug poisonings admitted to hospital have different needs with regards to psychiatric intervention.

1. AIMS AND OBJECTIVES:

- To know the epidemiological profile of patients admitted in our hospital with history of acute drug poisoning.
- To study various clinical presentations, haematological and biochemical, radiological abnormalities associated with outcome of the patients.

To follow up of cases with the help of telecommunication

Inclusion criteria:

1. All the patients admitted above 18 years with history of acute drug poisoning admitted in toxicology ward.
2. With or without comorbidities like hypertension, diabetes mellitus, depressive disorder etc..
3. Patients giving consent to the study.

Exclusion criteria:

1. Patients who had consumed multiple modes of poisoning.
2. Patients who didn't give consent to the study
3. Patients less than 18 years of age.

STUDY CENTRE: Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

DURATION OF STUDY :6 months

STUDY DESIGN: prospective observational study

SAMPLE SIZE : 80 cases

Calculated from the following formula:

Sample size (n) =Z2pq/d2

p= prevalence (26%)

q=1-p

d=Absolute precision (10)

METHOD OF COLLECTION OF DATA:

After selecting patients as per inclusion and exclusion criteria, detailed history will be elicited and clinical examination will be done after stabilisation of airway, breathing and circulation. Details regarding type of drug intake, quantity of consumption ,Time of consumption, intent suicidal or accidental, demographic data and clinical presentations.

Patients selected for the study will undergo blood investigations like complete blood picture, renal function tests, liver function tests, serum electrolytes, coagulation profile, chest X-ray, ecg, echo, ABG, urine analysis, toxic drug analysis and their reports will be included.

Types of poisoning:

1) **Acute or sudden onset of poisoning** can be taking single large bulky dose and also by consuming small quantity multiple times within a short duration.

2) **Chronic method** is consuming minimal quantity for a long time causing progressive worsening of the general condition. The poisons which are commonly used for the purpose of chronic poisoning are arsenic, phosphorus, antimony and opium.

3) **Sub acute poisoning** shows features of both acute and chronic poisoning.

4) **Fulminant poisoning** is produced by a massive dose. In this death

Identification of poison:

1) In the Living: There is no single symptom, and o definite group of symptoms, which are absolutely characteristic of poisoning. The closest resemblance to disease, may be producedinthallium poisoning. A detailed clinical history is of great importance.

The following groups of symptoms are suggestive of poisoning.

- The immediate appearance of pain abdomen, nausea, vomiting, loose stools.
- The unexplained coma with miosis.
- The sudden onset of seizure.
- Delirium with dilated pupils.
- Paralysis, especially of lower motor neurone type. Jaundice and hepatocellular failure.
- Anuria or oliguria with proteinuria and hematuria.
- Persistent cyanosis.
- Rapid onset of neurological or gastrointestinal illness in persons known to be occupationally exposed to chemicals.
- A priority of suspicion is needed to recognise poisoning if history is unavailable.
- swollen/puffiness of face, abnormally cold or warmth in a surrounding of an empty bottle lying around, spilling of chemical substances over the dress & empty cover of tablets or medicine.

By taking into account of the number of tablets or chemical left in

4. COMMON DRUG POISONINGS:

ACETAMINOPHEN:

Acetaminophen toxicity is the second most common cause of liver transplantation worldwide.

Acetaminophen is a non-steroidal anti-inflammatory drug with a mechanism of action different from that of other NSAIDS. It inhibits cyclooxygenase in the brain. It also inhibits prostaglandin synthesis in the central nervous system. It also directly acts on the hypothalamus producing an antipyretic effect

Recommended dose of acetaminophen for adults is 650 mg to 1000mg 4 to 6th hourly not to exceed 4 g/day.

In children the dose is 15mg/kg every 6hrs upto 60mg/kg/day.

Toxicity devolops at 7.5g/day to 10 g/day.

It is metabolised by the liver, where it is conjugated to non-toxic, water soluble metabolite that are excreted in the urine.

Stages of toxicity:

First stage:(30min to 24 hours)

Patient may be asymptomatic or have emesis.

Second stage (18 to 72 hrs)

There may be emesis, right upper quadrant pain, and hypotension.

Third stage:(72 to 96 hrs)

Liver dysfunction is significant with renal failure, coagulopathies, metabolic acidosis and encephalopathy. Gastric symptoms reappear and death is most common at this stage.

Fourth stage:(4 days to 3 weeks)

Marked by recovery.

Treatment:

If the patient presents within 1 hour of ingestion, GI decontamination may be attempted.

If the serum drug levels fall in the toxic range according to rumackMatthew nomogram, an APAP level greater than 10 mcg/ml with an unknown time of ingestion, abnormal labs with ingestion more than 24hrs ago, N-Acetyl cysteine is indicated.

I.v loading regimen:150 mg/kg over 15 min, followed by 50mg/kg over 4hrs, and then 100mg/16 hrs.

BENZODIAZEPINE POISONING:

Benzodiazepines are second only to opiates as the drugs most frequently involved in medication-related deaths.

However, benzodiazepines are rarely fatal when ingested alone, and other respiratory depressant drugs (e.g., opiates) are almost always involved in benzodiazepine-related fatalities (2).

Clinical features:

Because overdoses involving benzodiazepines also involve other drugs, the clinical presentation can vary (according to the drugs ingested). Pure benzodiazepine overdoses produce deep sedation, but rarely result in coma (18).

Respiratory depression (2–12% of cases), bradycardia (1–2% of cases) and hypotension (5–7% of cases) are also uncommon (18).

Benzodiazepine intoxication can also produce an agitated confusional state (with hallucinations) that could be mistaken for alcohol withdrawal (1)

Management:

The management of benzodiazepine overdose involves general supportive care, although an antidote is available.

Flumazenil is a benzodiazepine antagonist that binds to benzodiazepine receptors, but does not exert any agonist actions.

It is effective in reversing benzodiazepine-induced sedation, but is inconsistent in reversing benzodiazepine-induced respiratory depression

CLINICAL USE:

Despite its effectiveness in reversing benzodiazepine-induced sedation, flumazenil is not a popular antidote. This is partly due to concerns about the risk of benzodiazepine withdrawal or seizures, and is partly due to the fact that benzodiazepine overdoses are rarely life-threatening.

Opioid poisoning:

Opiates are implicated in 75% of fatal drug overdoses.

Clinical features:

The classic description of an opiate overdose is a patient who presents with stupor, pinpoint pupils, and slow breathing (bradypnea).

However, these clinical findings are either absent or nonspecific, and it is not possible to identify an opiate overdose based on the clinical presentation or physical examination.

The response to the narcotic antagonist, naloxone, is probably the most reliable method of identifying an opioid over dose.

Naloxone:

Naloxone is a pure opioid antagonist; i.e., it binds to endogenous opioid receptors, but does not elicit any agonist responses.

It is most effective in blocking mu receptors (primarily responsible for analgesia, euphoria, and respiratory depression) and less effective in blocking kappa receptors and delta receptors

Salicylate poisoning:

Clinical features:

Ingestion of 10–30 grams of aspirin (150 mg/kg) can have fatal consequences. Once ingested, acetylsalicylic acid (aspirin) is promptly converted to salicylic acid, which is the active form of the drug. Salicylic acid is readily absorbed from the upper GI tract, and metabolism takes place in the liver. The hallmark of salicylate intoxication is the combination of a respiratory alkalosis and a metabolic acidosis.

Diagnosis:

The plasma salicylate level is used to confirm or exclude the diagnosis of salicylate toxicity. The therapeutic range of salicylates in plasma is 10–30 mg/L (0.7–2.2 mmol/L), and levels above 40 mg/L (2.9 mmol/L) are considered toxic)

Plasma salicylate levels are usually elevated within 4–6 hours after a toxic ingestion.

Management:

Management of salicylate toxicity includes general supportive care (i.e., fluids, vasopressors, and mechanical ventilation), if necessary. Multiple-dose activated charcoal is recommended, if it can be started within 2–3 hours of drug ingestion. The dosing regimen is 25 grams orally every 2 hours for 3 doses.

Antidepressants:

Antidepressants modulate the activity of serotonin and norepinephrine to achieve their effect. This class includes citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, Sertralin.

Clinical manifestations:

Acute overdose:

Acute signs and symptoms include nausea, vomiting, dizziness, blurred vision, and less commonly central nervous system depression and sinus tachycardia.

Seizures and ecg changes including prolongation of the QRS complex and QT interval but rarely occur with most SSRIs even after large overdoses.

Management

Treatment of patients with acute SSRI overdose is largely supportive. Dextrose and thiamine should be considered for patients who present with altered mental status.

After the patient is stabilized, oral activated charcoal (1g/kg) may be useful to adsorb drug remaining in the gastrointestinal tract.

Minimal lethal concentrations of fluoxetine, paroxetine, and sertraline after isolated overdose are 0.63,0.64,1.5 mg/L respectively.

Tricyclic antidepressants:

Tricyclic antidepressants overdose is one of the most common cause of acute poison related icu admissions.

Ingestion of greater than 10mg/kg of TCA is likely to produce significant toxicity while 20-30/kg is considered a potentially lethal dose.

The anticholinergic properties of TCAs produce sinus tachycardia, warm dry skin, brisk reflexes, sedation, seizures, and urinary retention. Pupils are commonly poorly reactive and dilated, but can vary in size.

The high mortality rate associated with TCAs is primarily caused by sodium channel block, which occurs in overdose leads to cardiac conduction abnormalities.

ECG abnormalities includes QRS, QT, PR prolongation and right axis deviation.

Management:

Sodium bicarbonate is the first line agent used to treat TCA related cardiovascular toxicity.

Systemic alkalinization and hypertonic sodium loading both contribute to sodium bicarbonate mechanism of action in treating TCA related toxicity.

All anti arrhythmics should be avoided where possible in patients with TCA poisoning, particularly class Ia anti arrhythmics, which are contraindicated in the treatment of TCA induced cardiac arrhythmias.

AV Blocks resistant to treatment with sodium bicarbonate may require temporary cardiac pacing.

Anti convulsants:

Phenytoin:

Intentional phenytoin overdose rarely leads to death, provided adequate supportive care is administered.

Clinical features of phenytoin toxicity:

Central nervous system:

Dizziness, tremor (intention), visual disturbance, horizontal and vertical nystagmus, diplopia, miosis or mydriasis, ophthalmoplegia, abnormal gait (bradykinesia, truncal ataxia), choreoathetoid movements, irritability, agitation, confusion, hallucinations, fatigue, coma, encephalopathy, dysarthria, meningeal irritation with pleocytosis, seizures.

Dermatologic:

Hirsutism, acne, rashes (including Stevens–Johnson syndrome)

Diagnosis:

Diagnosis is made by history, clinical exam, and serum drug levels. The therapeutic range is 10 to 20 µg/mL and toxicity generally correlates with increasing plasma levels .

Correlation of plasma phenytoin levels and side effects:

Total plasma level(ug/ml)	Toxic effects
< 10	Usually none
10 -20	Occasional Mild nystagmus
20-30	Nystagmus
30-40	Ataxia,slurred speech,nausea, vomiting
40- 50	Lethargy,confusion
>50	Coma,seizures

Management:

- Treat acute oral overdose or severe supratherapeutic toxicity with multidose of oral activated charcoal (1 g/kg) every 4 to 6 hours for the first 24 hours.
- Correct acidosis to decrease free serum phenytoin.
- Treat hypotension from IV administration of phenytoin with IV isotonic crystalloid and discontinuation of the infusion.
- Treat bradydysrhythmias with atropine or cardiac pacing
- Treat seizures with a benzodiazepine or phenobarbital.

Antihypertensives:

Calcium channel blockers:

Calcium channel blockers are used in the treatment of hypertension, vasospasm and rate control of supraventricular tachydysrhythmias.

Clinical features:

Toxicity usually develops within 6 hours of ingestion of an immediate release product. With sustained-release preparations toxicity can be delayed 12 to 24 hours.

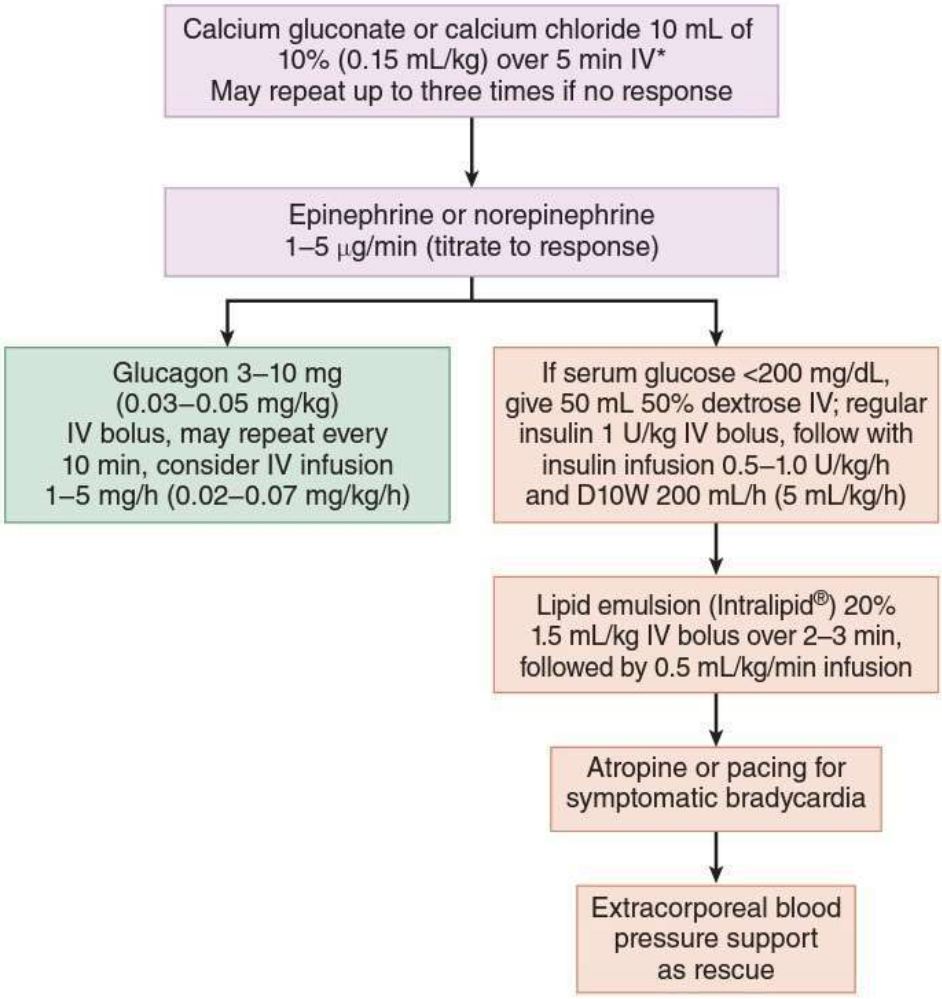
Toxicity primarily affects the cardiovascular system causing sinus bradycardia, atrioventricular block, and hypotension. Verapamil and diltiazem have a proportionally greater effect on the myocardium than the dihydropyridines. Dihydropyridine overdose can result in reflex tachycardia.

With severe toxicity, all classes of calcium channel blockers can cause bradycardia, depressed myocardial contractility, and vasodilatation. Hyperglycemia, lactic acidosis, and noncardiogenic pulmonary edema may occur.

Central nervous system effects are due to hypoperfusion and other etiologies should be sought if the blood pressure is normal.

Management:

Treatment is supportive, with an emphasis on increasing cardiac output and systemic vascular resistance (Fig. 108-2). Establish continuous cardiac monitoring and IV access. Administer IV fluids for hypotension.



Beta blockers:

β-Blockers are used in the management of hypertension, acute coronary syndromes, dysrhythmias, congestive heart failure, thyrotoxicosis, social phobias, migraines, and glaucoma. In overdose, their negative inotropic and chronotropic effects result in progressive bradycardia and hypotension.

Common findings with beta blocker toxicity:

1**.cardiac:**

- Hypotension
- Bradycardia
- Conduction delays and blocks
- Ventricular dysrhythmias.
- Asystole
- Decreased contractility

2**.central nervous system**:

- Depressed mental status
- Coma
- Psychosis
- Seizures

3.**pulmonary**:

Bronchospasm

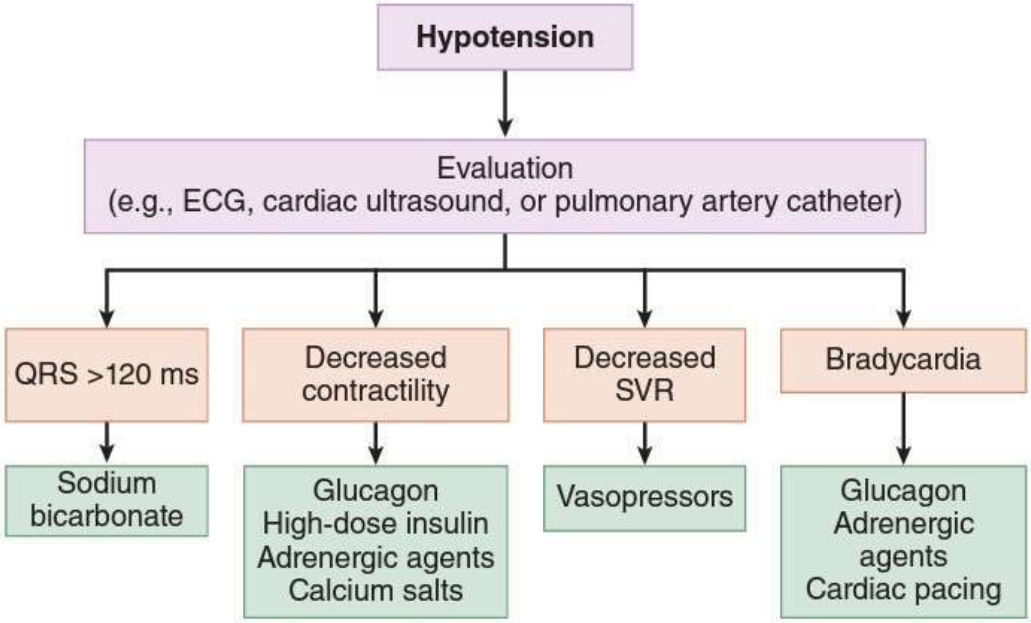
4.**Endocrine**:

Hypoglycemia

Diagnosis:

The diagnosis is made based on clinical findings. An ECG should be obtained in all cases.

Laboratory studies are directed at identifying underlying medical conditions or complications.



Management:

- Administer activated charcoal 1 g/kg within 1 to 2 hours of ingestion if no contraindications exist.
- Glucagon has inotropic and chronotropic effects and is the agent of choice for the treatment of toxicity. Administer as an IV bolus of 3 to 10 mg (0.05 mg/kg). Follow with a continuous infusion of 1 to 5 mg/h. Nausea and vomiting are common side effects. Pretreatment with antiemetics is recommended.
- Use vasopressors, such as norepinephrine 2 to 30 µg/kg/min or epinephrine 1 to 20 µg/kg/min, for refractory bradycardia and hypotension. Significantly higher doses may be necessary.

RESULTS

The results of the prospective study done to study the epidemiological, clinical, toxicity, outcome of 80 patients admitted with history of acute tablet poisoning in a tertiary care centre are as follows:

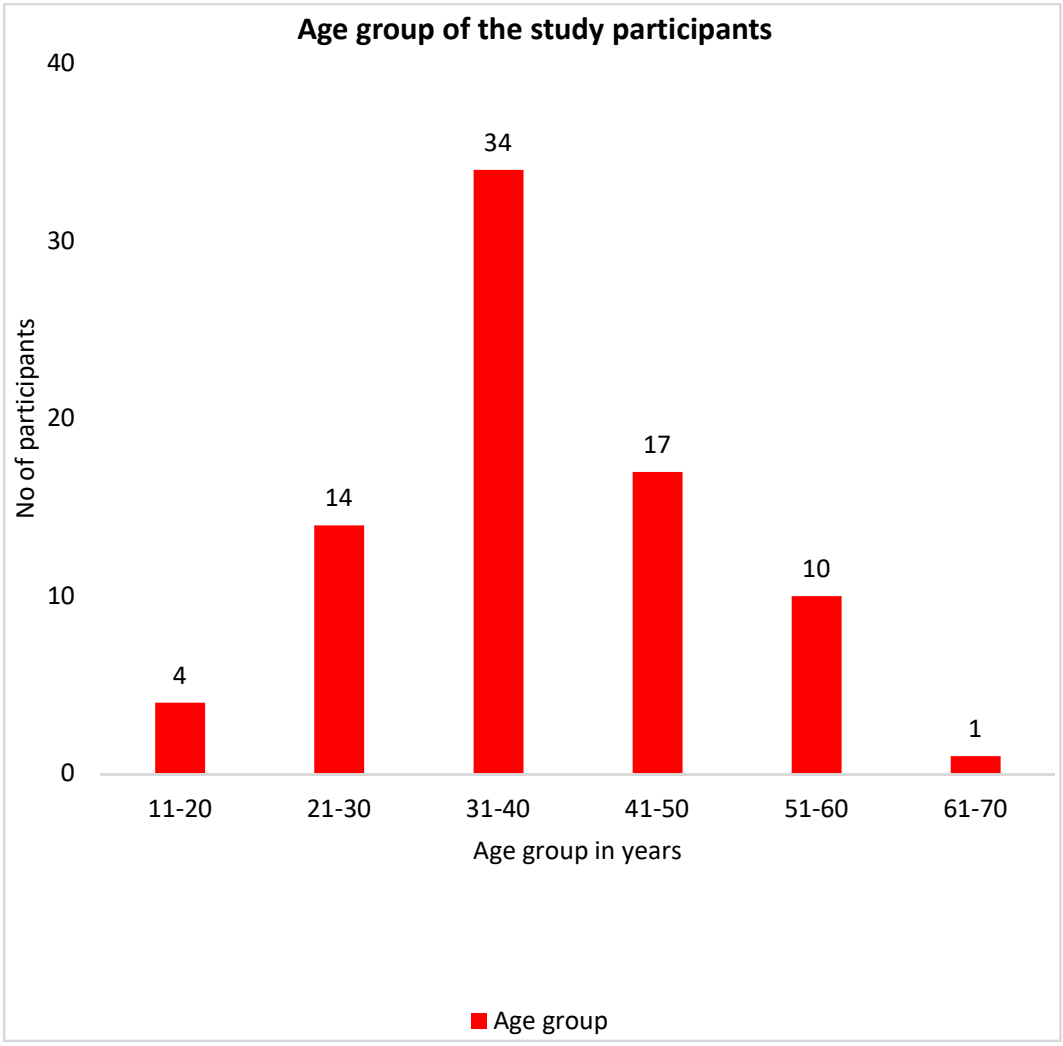
Age group of the study participants:

Table 1. Age group of the study participants

Age group (in years)	Frequency	Percentage
11-20	4	5
21-30	14	17.5
31-40	34	42.5
41-50	17	21.3
51-60	10	12.5
61-70	1	1.3
Total	80	100

The mean age of the study participants was 38.36 ±10 years. Majority of participants were from 31 to 40 years. 17 participants were in 41 to 50 years age group, 14 were from 21 to 30 years age group, 10 were from 51 to 60 years age group, 4 were from 11 to 20 years age group and 1 participant was above 61 years.

Chart 1. Age group of the study participants



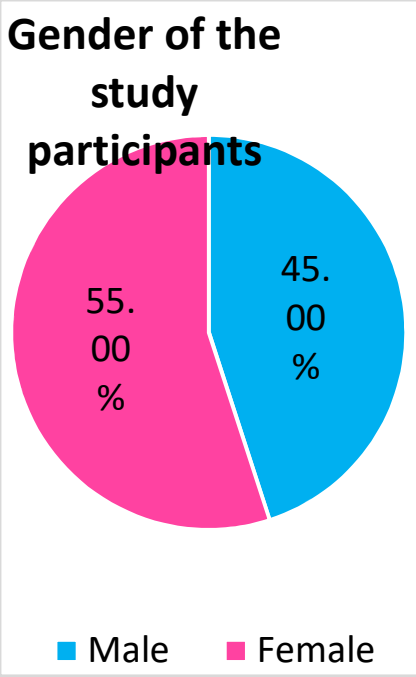
Gender of the study participants:

Table 2. Gender of the study participants

Gender	Frequency	Percentage
Male	36	45
Female	44	55
Total	80	100

Among the study participants, 36 participants were male and 44 participants were females.

Chart 2. Gender of the study participants



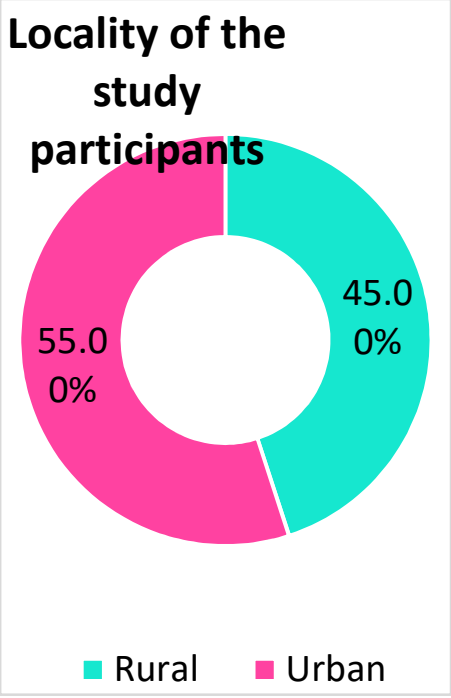
Locality of the study participants:

Table 3. Locality of the study participants

Locality	Frequency	Percentage
Rural	36	45
Urban	44	55
Total	80	100

Among the study participants, 36 participants were from rural area and 44 participants were from urban area.

Chart 3. Locality of the study participants



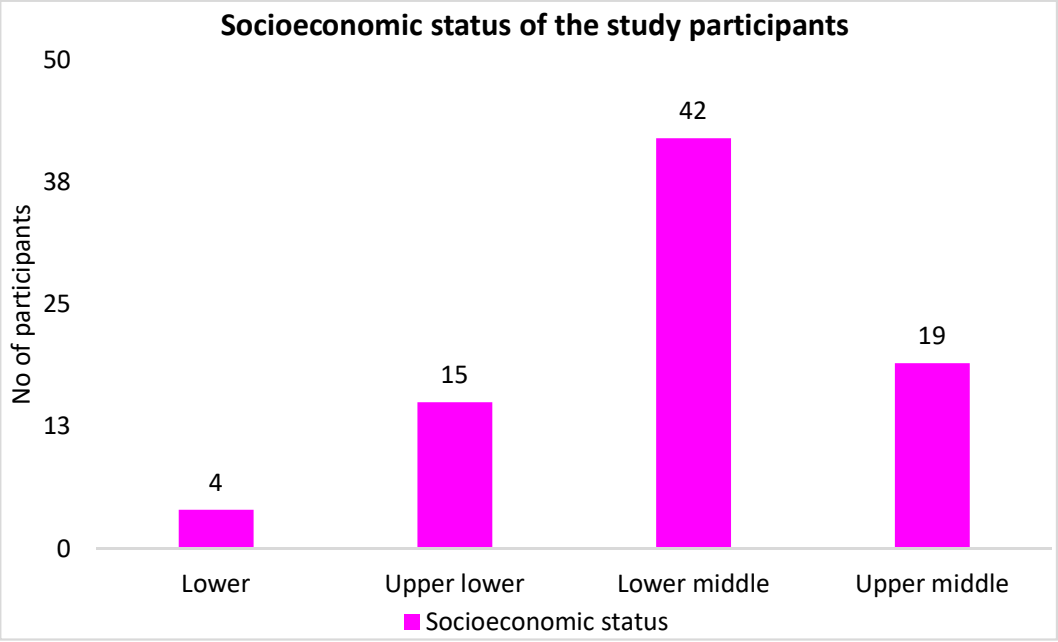
Socioeconomic status of the study participants:

Table 4. Socioeconomic status of the study participants

Socioeconomic status	Frequency	Percentage
Lower	4	5
Upper lower	15	18.8
Lower middle	42	52.5
Upper middle	19	23.8
Total	80	100

Among the socioeconomic status of the study participants, 4 participants were from lower class, 15 participants were from upper lower class, 42 participants were from lower middle class and 19 participants were from upper middle class.

Chart 4. Socioeconomic status of the study participants



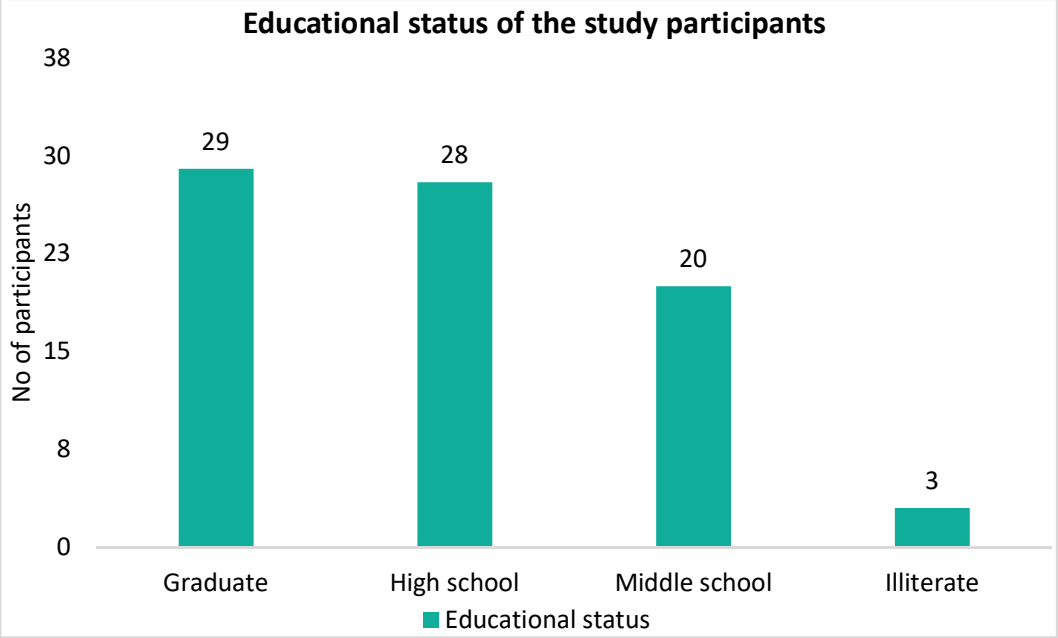
Educational status of the study participants:

Table 5. Educational status of the study participants

Educational status	Frequency	Percentage
Graduate	29	36.3
High school	28	35
Middle school	20	25
Illiterate	3	3.8
Total	80	100

Among the study participants, 29 participants were graduate, 28 participants studied upto high school, 20 participants studied upto middle school and 3 participants were illiterate.

Chart 5. Educational status of the study participants



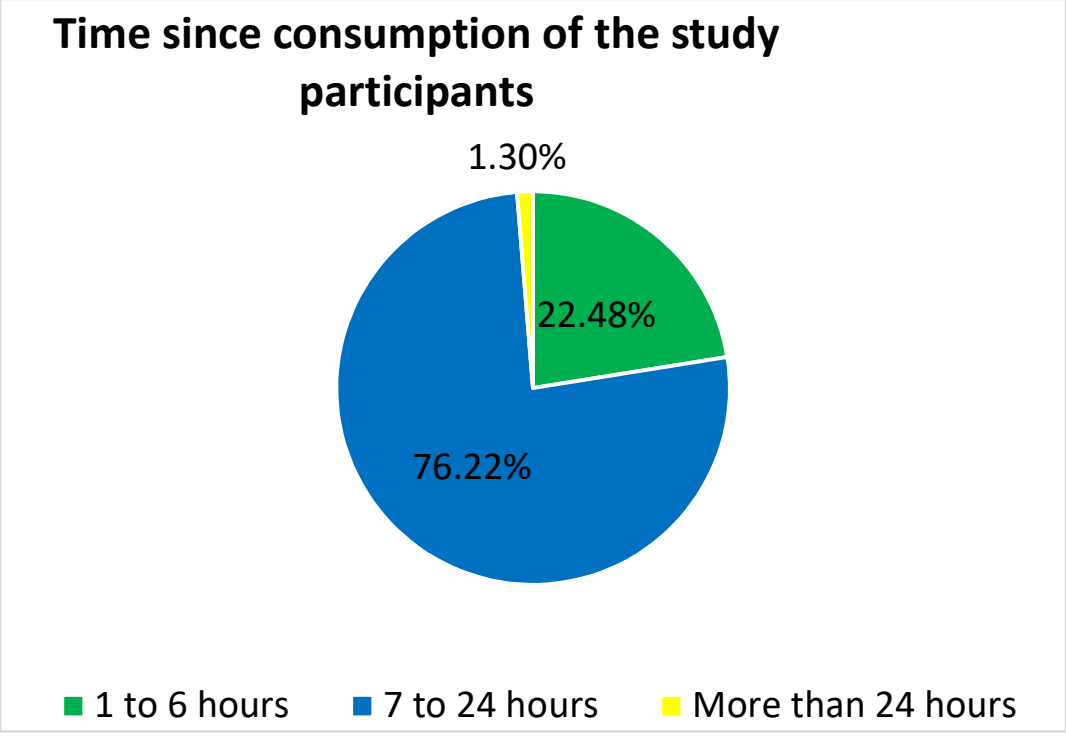
Time since consumption of the study participants:

Table 6. Time since consumption of the study participants

Time since consumption	Frequency	Percentage
1 to 6 hours	18	22.5
7 to 24 hours	61	76.3
More than 24 hours	1	1.3
Total	80	100

The mean time since consumption to arriving to hospital was 12.41 ±5.75 hours. 18 participants arrived from 1 to 6 hours, 61 participants from 7 to 24 hours and 1 participant arrived after more than 24 hours.

Chart 6. Time since consumption of the study participants



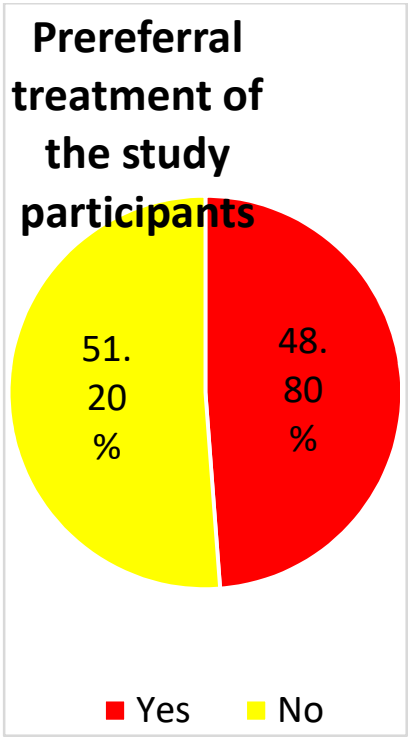
Prereferral treatment of the study participants:

Table 7. Prereferral treatment of the study participants

Prereferral treatment	Frequency	Percentage
Yes	39	48.8
No	41	51.2
Total	80	100

Among the study participants, 39 participants had prereferral treatment and 41 participants did not have prereferral treatment.

Chart 7. Prereferral treatment of the study participants



Type of drug consumed by the study participants:

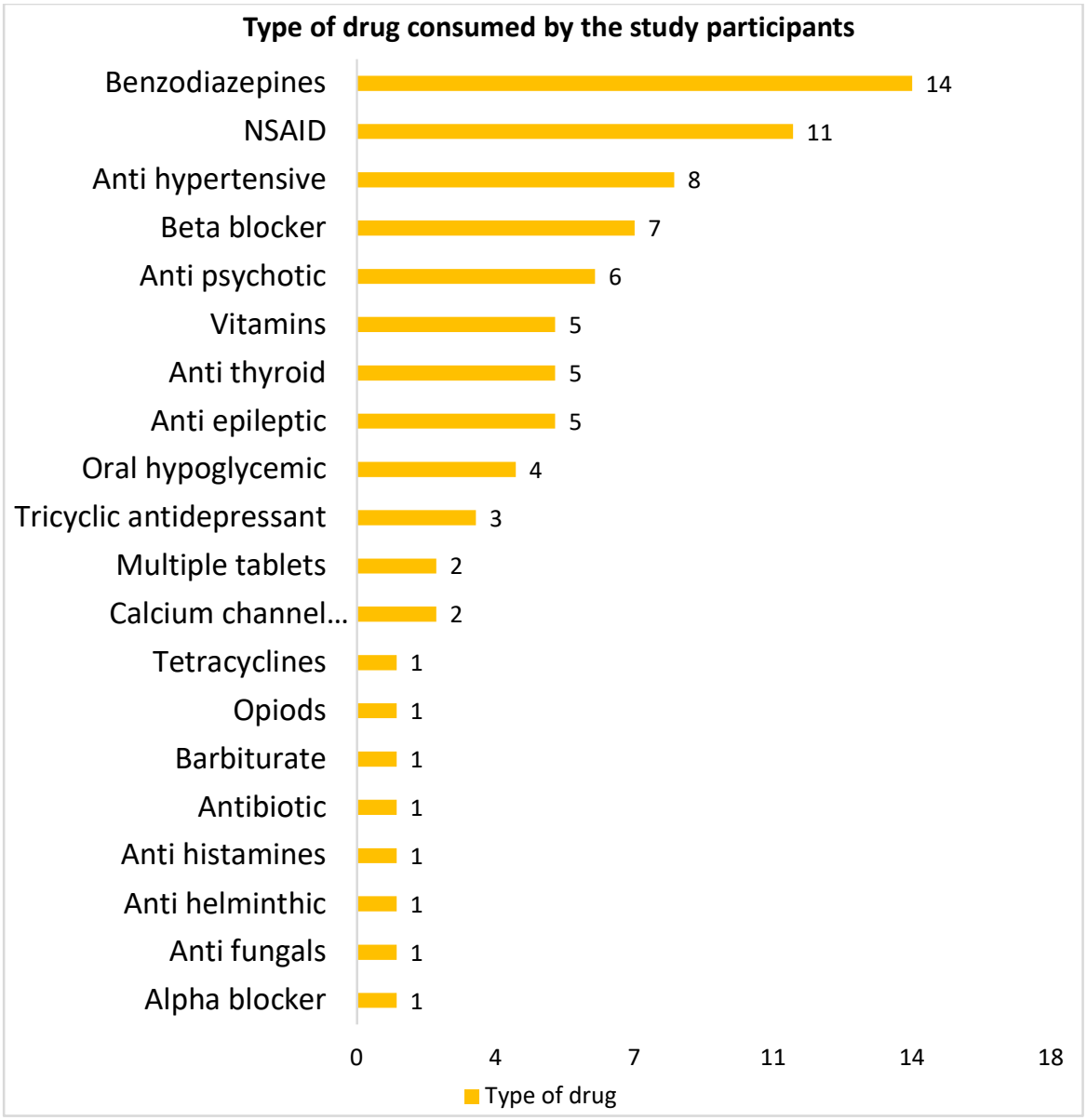
Table 8. Type of drug consumed by the study participants

Type of drug	Frequency	Percentage
Benzodiazepines	14	17.5
NSAID	11	13.8
Anti hypertensive	8	10.0
Beta blocker	7	8.8
Anti psychotic	6	7.5
Anti epileptic	5	6.3
Anti thyroid	5	6.3
Vitamins	5	6.3
Oral hypoglycemic	4	5.0
Tricyclic antidepressant	3	3.8
Calcium channel blocker	2	2.5
Multiple tablets	2	2.5
Alpha blocker	1	1.3
Anti fungals	1	1.3
Anti helminthic	1	1.3
Anti histamines	1	1.3

Antibiotic	1	1.3
Barbiturate	1	1.3
Sedatives	1	1.3
Tetracyclines	1	1.3
Total	80	100

Among the total study participants, 14 participants had consumed Benzodiazepines followed by 11 participants had consumed NSAIDs. 8 participants had Anti-hypertensive, 7 had Beta blocker, 6 had Anti-psychotic, 5 had Anti-epileptic, 5 had Anti thyroid, 5 had Vitamins, 4 had Oral hypoglycemic, 3 had Tricyclic antidepressant, 2 had Calcium channel blocker, 2 had Multiple tablets, 1 had Alpha blocker, 1 had Anti fungals, 1 had Anti helminthic, 1 had Anti histamines, 1 had Antibiotic, 1 had Barbiturate, 1 had opiod and 1 had Tetracyclines.

Chart 8. Type of drug consumed by the study participants



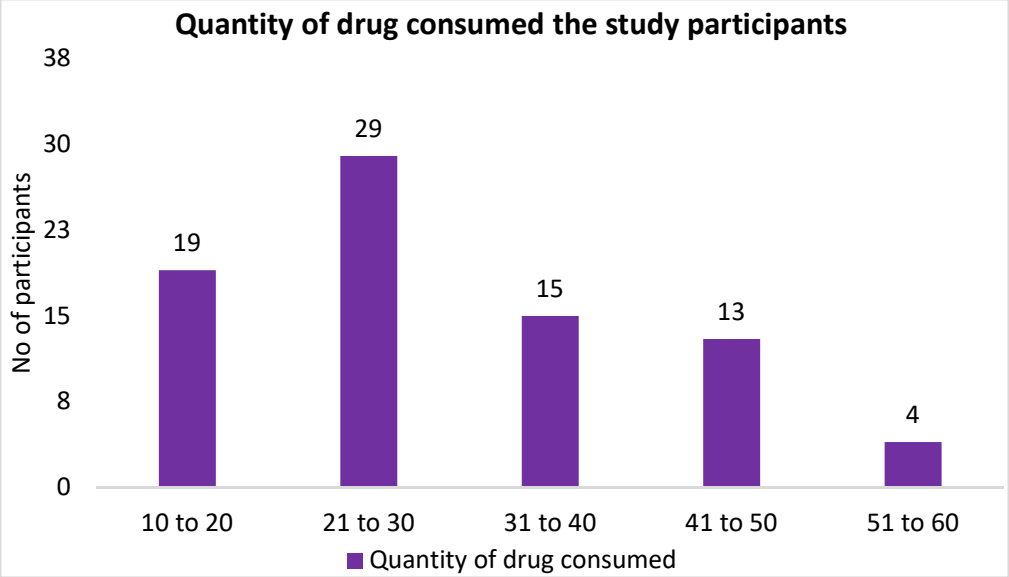
Quantity of drug consumed the study participants:

Table 9. Quantity of drug consumed the study participants

Quantity of drug consumed	Frequency	Percentage
10 to 20	19	23.8
21 to 30	29	36.3
31 to 40	15	18.8
41 to 50	13	16.3
51 to 60	4	5
Total	80	100

The mean quantity of tablets consumed by the participants were 32.26 ±11.93 tablets. 19 participants consumed 10 to 20 tablets, 29 participants consumed 21 to 30 tablets, 15 participants consumed 31 to 40 tablets, 13 participants consumed 41 to 50 tablets and 4 participants consumed 51 to 60 tablets.

Chart 9. Quantity of drug consumed the study participants



Symptoms of the study participants:

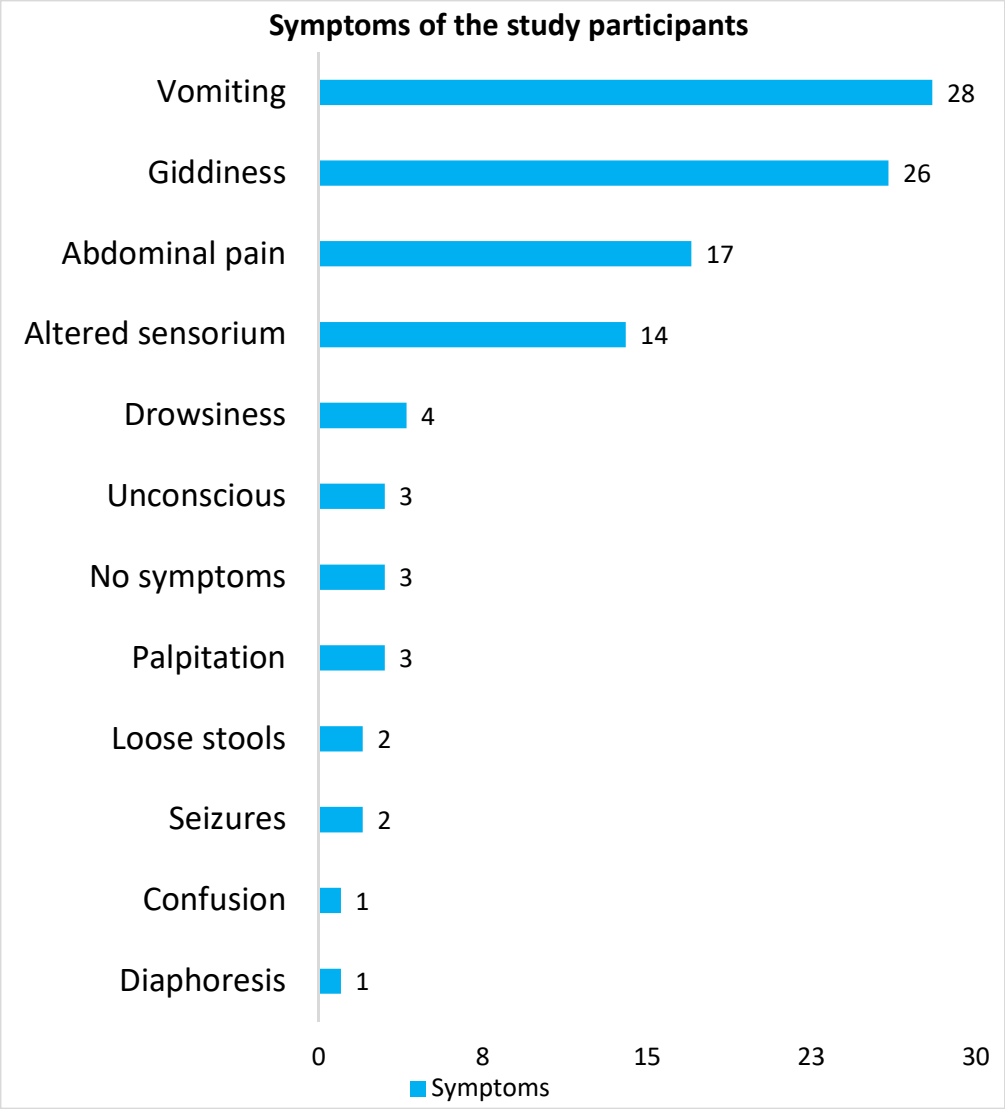
Table 10. Symptoms of the study participants

Symptoms	Frequency	Percentage
Vomiting	28	35
Giddiness	26	32.5
Abdominal pain	17	21.2
Altered sensorium	14	17.5
Drowsiness	4	5
Palpitation	3	3.7
No symptoms	3	3.7
Unconscious	3	3.7
Seizures	2	2.5
Loose stools	2	2.5
Diaphoresis	1	1.2
Confusion	1	1.2

Majority of the study participants had vomiting as the main symptom (28 participants). 26 participants had giddiness, 17 participants had abdominal pain, 14 participants had altered sensorium, 4 participants had drowsiness, 3 participants had palpitations, 3 participants had

no symptoms, 3 were unconscious, 2 had seizures, 2 had loose stools, 1 had diaphoresis and 1 had confusion.

Chart 10. Symptoms of the study participants



Pulse rate of the study participants at time of admission:

The mean pulse rate of the study participants was 85.99 ±15.88 beats per minute.

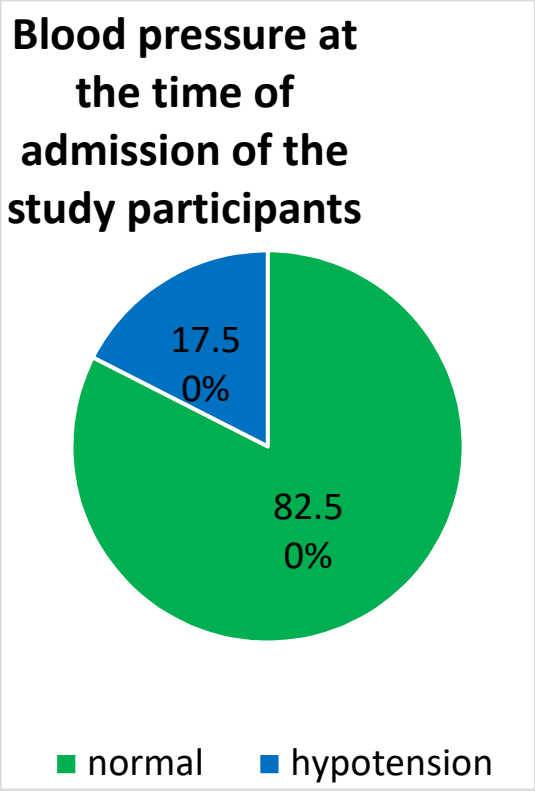
Blood pressure at the time of admission of the study participants:

Table 11. Blood pressure at the time of admission of the study participants

Blood pressure at the time of admission	Frequency	Percentage
Normal	66	82.5
Hypotension	14	17.5
Total	80	100

Among the study participants 66 participants had normal blood pressure during admission and 14 participants had hypotension during admission.

Chart 11. Blood pressure at the time of admission of the study participants



Respiratory rate of the study participants at time of admission:

The mean Respiratory rate of the study participants at time of admission was 18.31 ± 3.01 breaths per minute.

Coagulation profile of the study participants:

The coagulation profile was not altered for all the participants.

Mean urea of the study participants:

The mean blood urea of the study participants was 33.21 ±4.51g/dl.

Mean creatinine of the study participants:

The Mean serum creatinine of the study participants 0.83 ±0.15 g/dl.

Mean sodium levels of the study participants:

The Mean sodium levels of the study participants was 136.99 ±3.99.

Mean potassium levels of the study participants:

The Mean potassium levels of the study participants was 4.15 ±0.73.

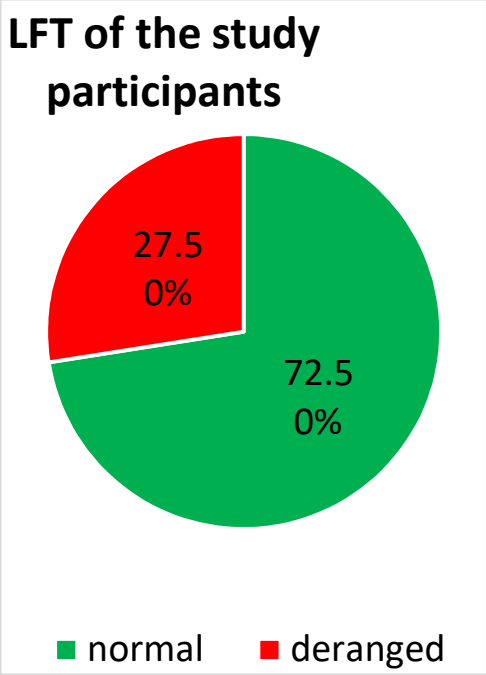
LFT of the study participants:

Table 12. LFT of the study participants

LFT	Frequency	Percentage
Normal	58	72.5
Deranged	22	27.5
Total	80	100

Among the total participants, 58 participants had normal LFT and 22 participants had deranged LFT.

Chart 12. LFT of the study participants



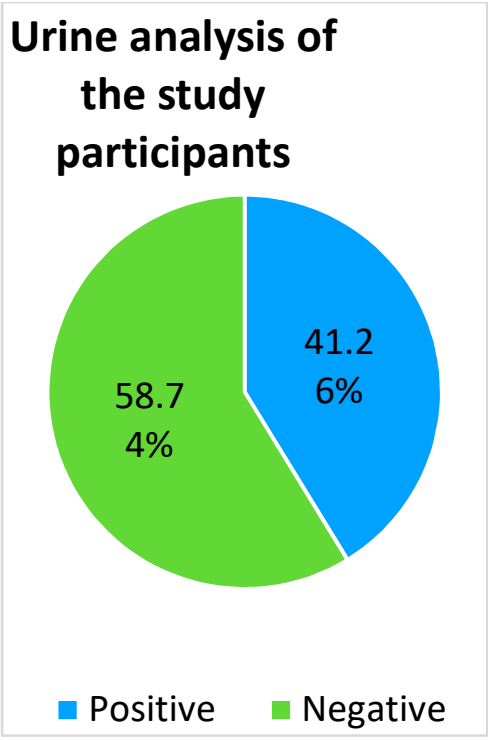
Urine analysis of the study participants:

Table 13. Urine analysis of the study participants

Urinalysis	Frequency	Percentage
Positive	33	41.3
Negative	47	58.8
Total	80	100

Among the total participants, 33 participants had positive urinalysis and 47 participants had negative urinalysis.

Chart 13. Urine analysis of the study participants



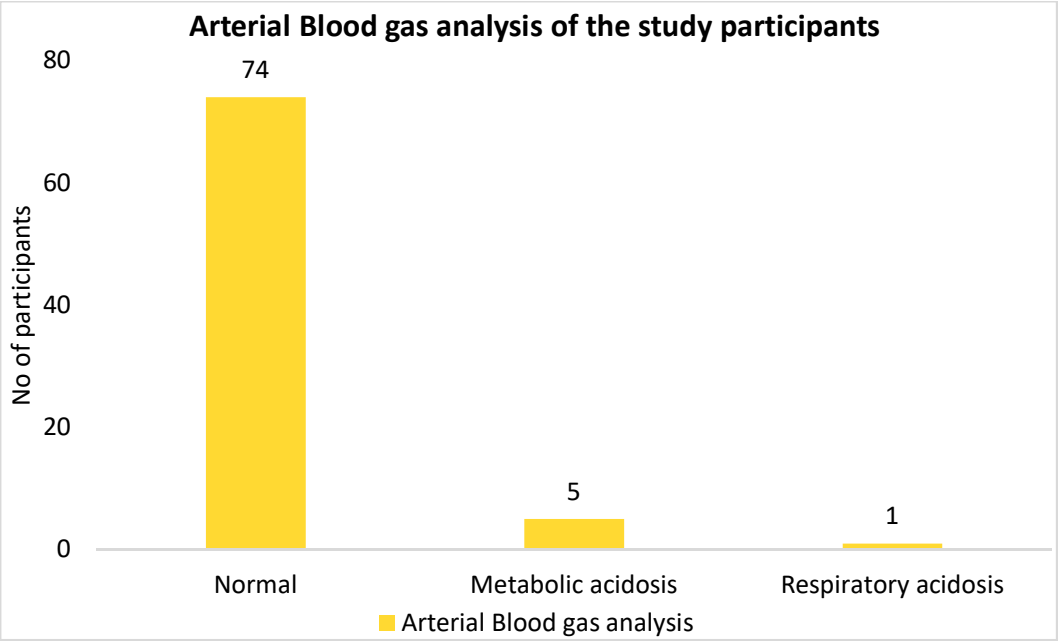
Arterial Blood gas analysis of the study participants:

Table 14. Arterial Blood gas analysis of the study participants

Arterial Blood gas analysis	Frequency	Percentage
Normal	74	92.5
Metabolic acidosis	5	6.3
Respiratory acidosis	1	1.3
Total	80	100

Among the study participants, 74 had normal arterial blood gas analysis, 5 had metabolic acidosis and 1 had respiratory acidosis.

Chart 14. Arterial Blood gas analysis of the study participants



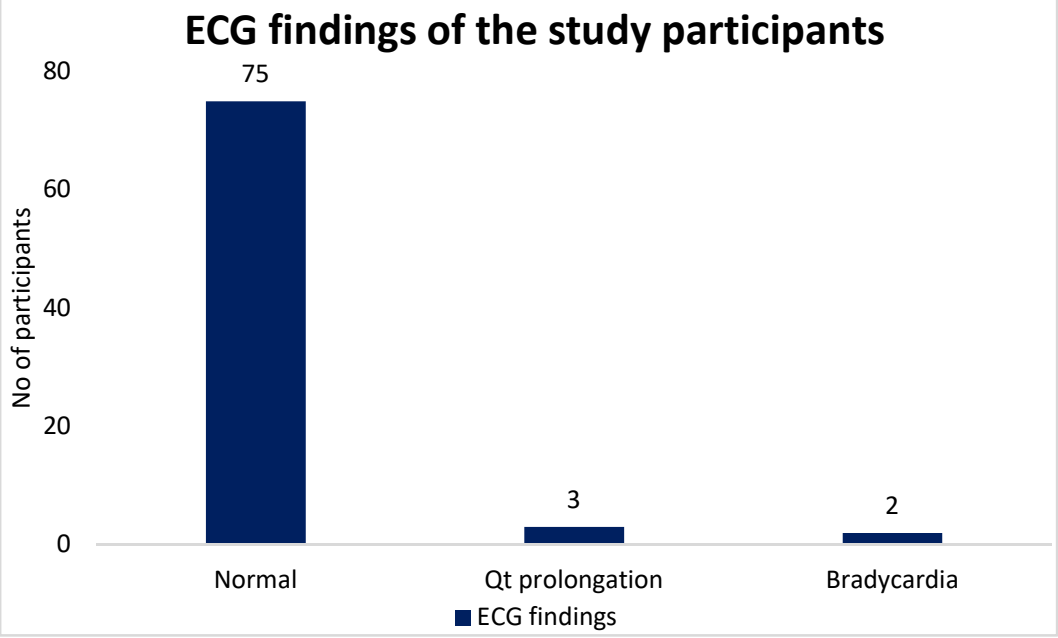
ECG findings of the study participants:

Table 15. ECG findings of the study participants

ECG findings	Frequency	Percentage
Normal	75	93.8
Qt prolongation	3	3.8
Bradycardia	2	2.5
Total	80	100

Among the study participants, 75 had normal ECG findings, 3 had Qt prolongation and 2 had bradycardia.

Chart 15. ECG findings of the study participants



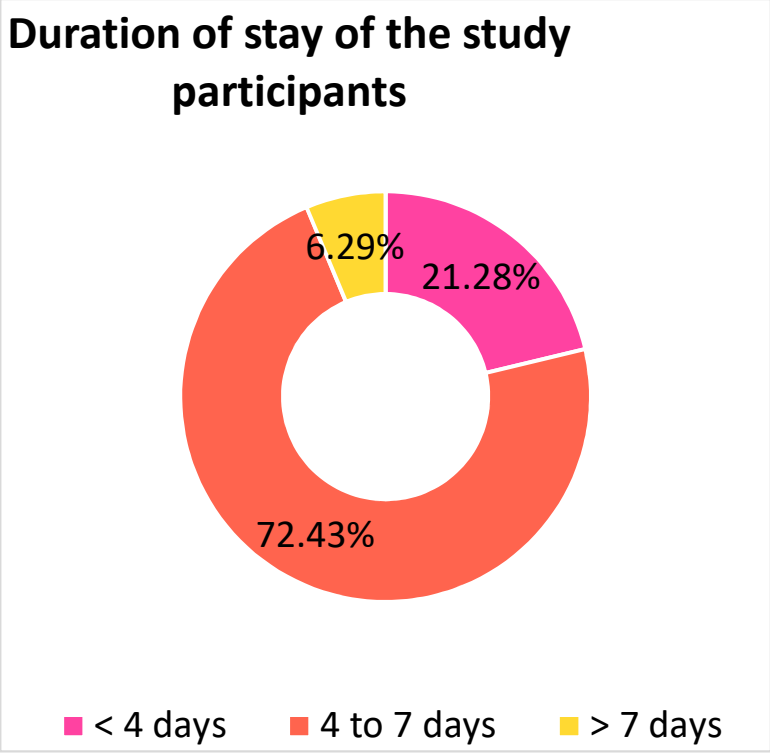
Duration of stay of the study participants:

Table 16. Duration of stay of the study participants

Duration of stay	Frequency	Percentage
< 4 days	17	21.3
4 to 7 days	58	72.5
> 7 days	5	6.3
Total	80	100

The mean duration of stay of the study participants was 4.83 ±1.66 days. 17 participants had duration of stay of less than 4 days, 58 participants had duration of 4 to 7 days and 5 had more than 7 days.

Chart 16. Duration of stay of the study participants



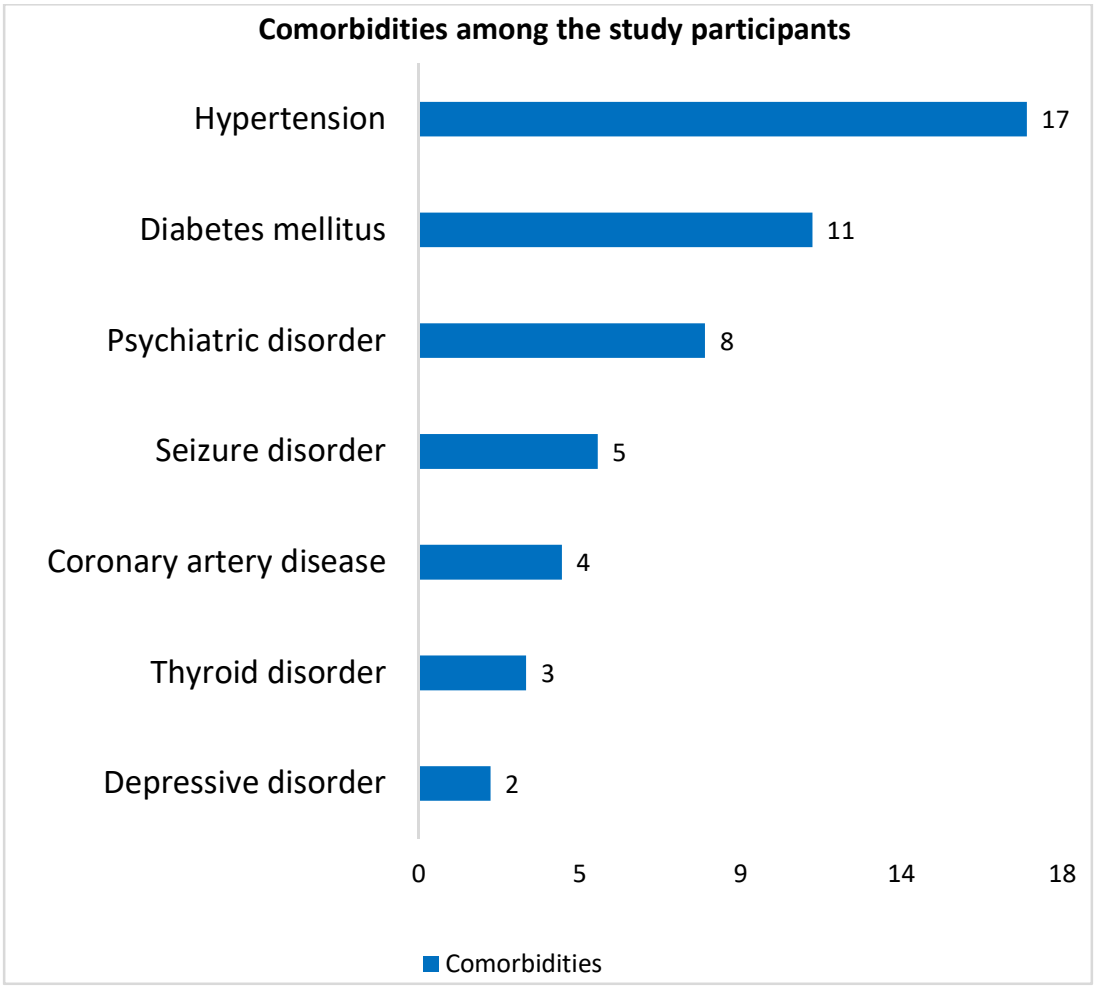
Comorbidities among the study participants:

Table 17. Comorbidities among the study participants

Comorbidities	Frequency	Percentage
Hypertension	17	21.3
Diabetes mellitus	11	13.8
Psychiatric disorder	8	10
Seizure disorder	5	6.3
Coronary artery disease	4	5
Thyroid disorder	3	3.8
Depressive disorder	2	2.5
Nil	44	55

Among the study participants, 17 had hypertension, 11 participants had diabetes mellitus, 8 had psychiatric disorder, 5 had seizure disorder, 4 had coronary artery disease, 3 had thyroid disorder and 2 had depressive disorder. 44 participants did not have any comorbidities.

Chart 17. Comorbidities among the study participants



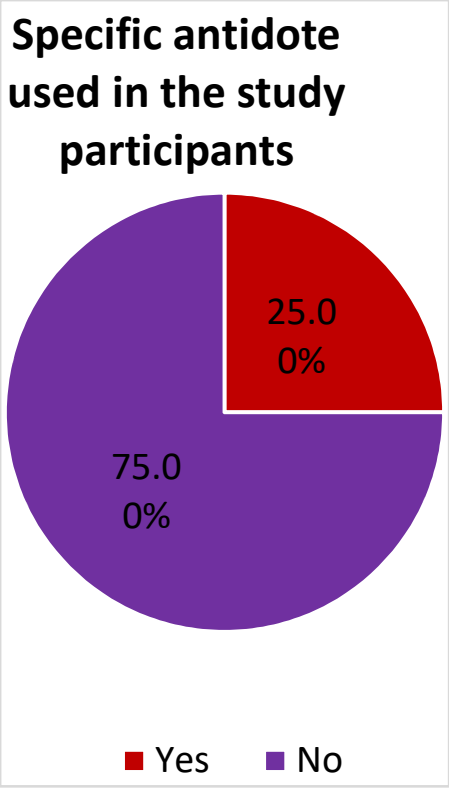
Specific antidote used in the study participants:

Table 18. Specific antidote used in the study participants

Specific antidote used	Frequency	Percentage
Yes	20	25
No	60	75
Total	80	100

Among the study participants, 20 participants were treated with specific antidote and 60 participants were not treated with specific antidote.

Chart 18. Specific antidote used in the study participants



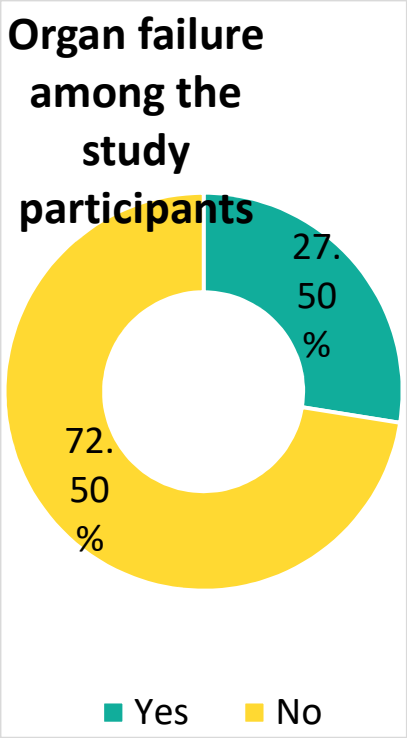
Organ failure among the study participants:

Table 19. Organ failure among the study participants

Organ failure	Frequency	Percentage
Yes	22	27.5
No	58	72.5
Total	80	100

22 participants had organ failure and 58 participants did not have organ failure.

Chart 19. Organ failure among the study participants



Outcome among the study participants:

All the study participants recovered and were discharged.

Association between time since consumption and organ failure among the study participants:

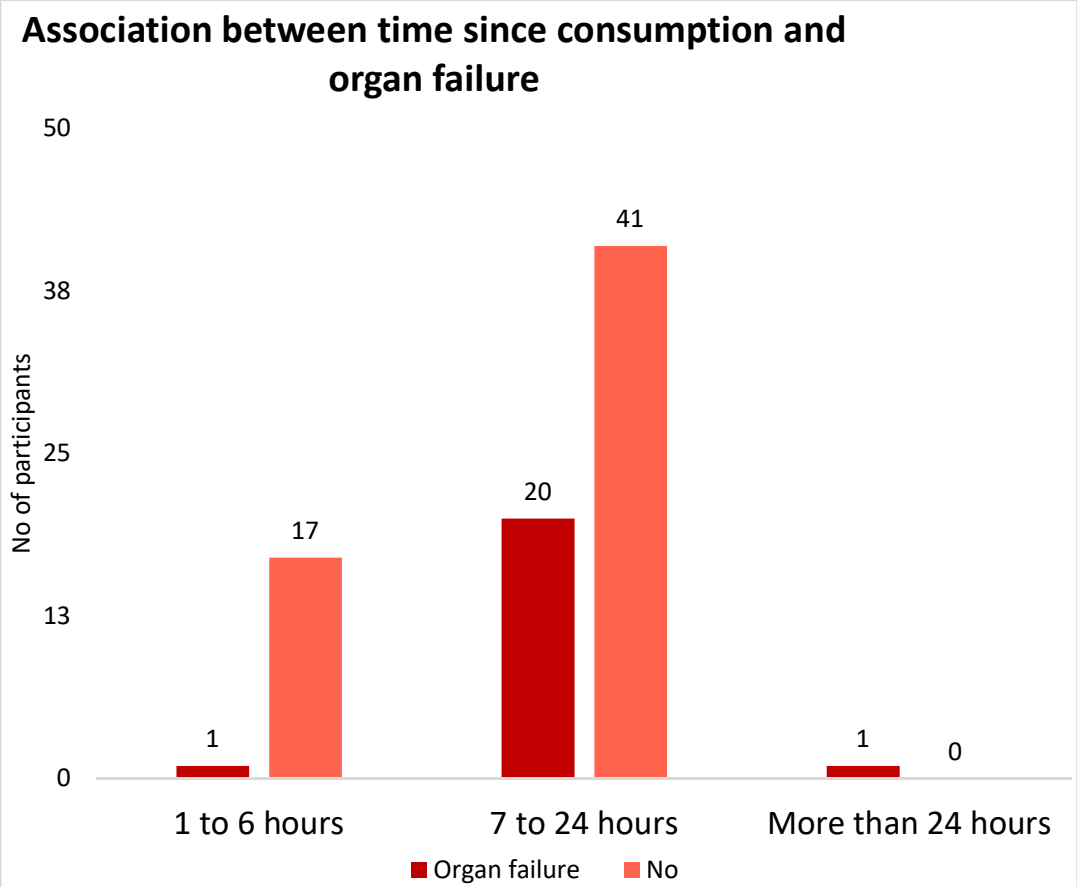
Table 20. Association between time since consumption and organ failure among the study participants

Time since consumption	Organ failure	No	Total	Fischer exact test value	P value
1 to 6 hours	1 5.5%	17 94.5%	18 100%	5.281	**0.02***
7 to 24 hours	20 32.7%	41 67.3%	61 100%		
More than 24 hours	1 100%	0	1 100%		
Total	22 27.5%	58 72.5%	80 100%		

*- statistically significant by Fischer Exact test

In the study participants who had time since consumption as 1 to 6 hours, 5.5% had organ failure and 94.5% did not have organ failure. In the study participants who had time since consumption as 7 to 24 hours, 32.7% had organ failure and 67.3% did not have organ failure. In the study participants who had time since consumption more than 24 hours, 100% had organ failure. This difference was statistically significant by Fischer exact test. ($p < 0.05$)

Chart 20. Association between time since consumption and organ failure among the study participants



Association between quantity of consumption and organ failure among the study participants:

Table 21. Association between quantity of consumption and organ failure among the study participants

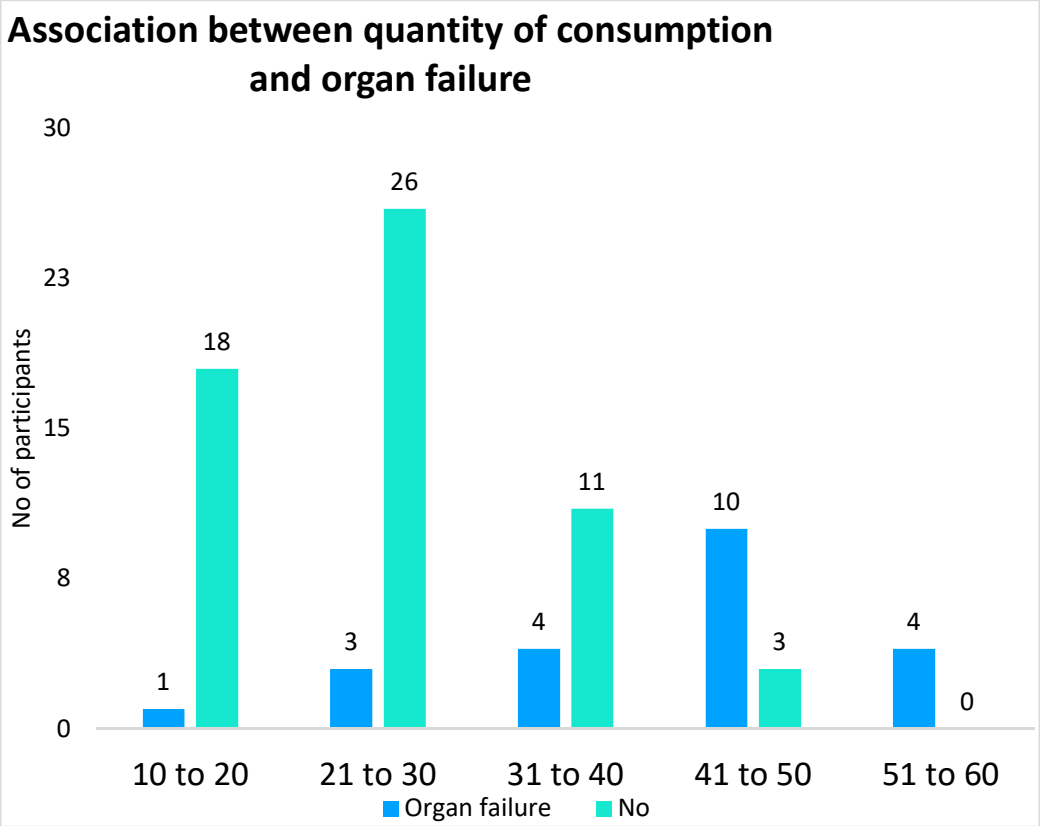
Quantity of consumption	Organ failure	No	Total	Fischer exact test value	P value
10 to 20	1 5.2%	18 94.8%	19 100%	29.89	**< 0.001***
21 to 30	3 10.3%	26 89.7%	29 100%		
31 to 40	4 26.7%	11 73.3%	15 100%		
41 to 50	10 76.9%	3 23.1%	13 100%		
51 to 60	4 100%	0	4 100%		
Total	22 27.5%	58 72.5%	80 100%		

*- statistically significant by Fischer Exact test

In participants who consumed 10 to 20 tablets, 5.2% had organ failure and 94.8% did not have organ failure. In participants who consumed 21 to 30 tablets, 10.3% had organ failure

and 89.7% did not have organ failure. In participants who consumed 31 to 40 tablets, 26.7% had organ failure and 73.3% did not have organ failure. In participants who consumed 41 to 50 tablets, 76.9% had organ failure and 23.1% did not have organ failure. In participants who consumed 51 to 60 tablets, 100% had organ failure. This difference was statistically significant by Fischer exact test. ($p < 0.05$)

Chart 21. Association between quantity of consumption and organ failure among the study participants



Association between age group and organ failure among the study participants:

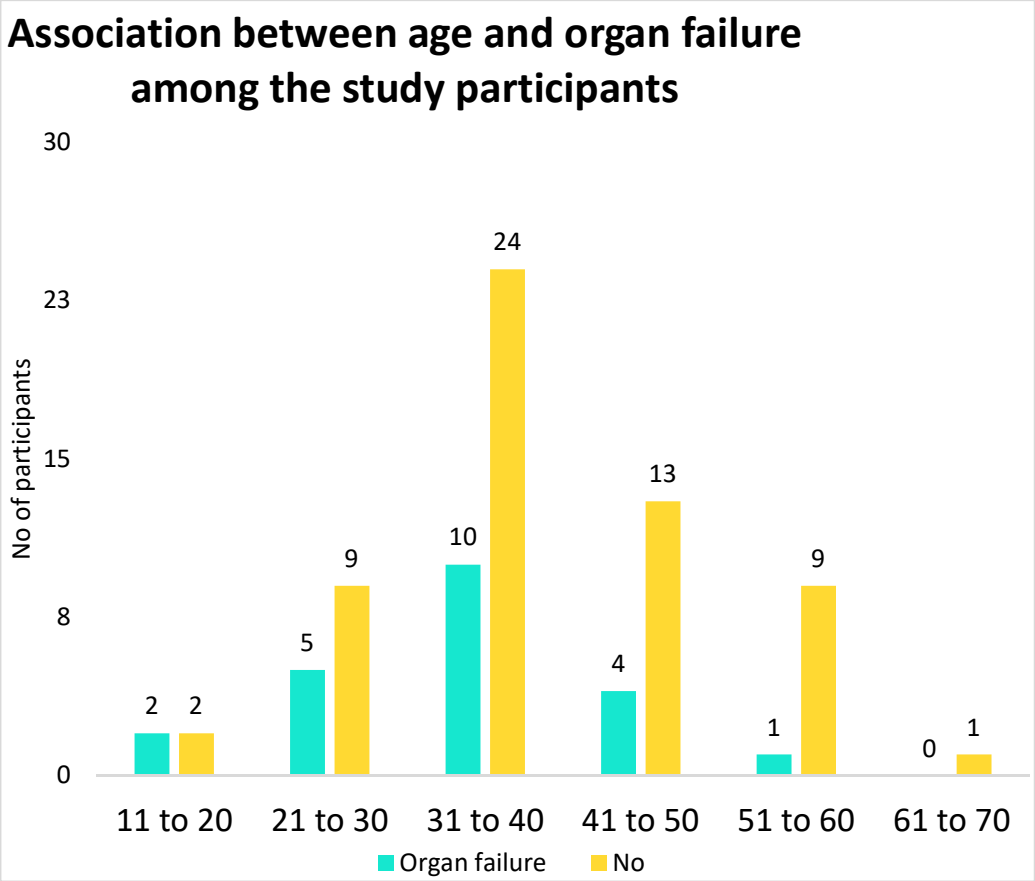
Table 22. Association between age and organ failure among the study participants

Age group	Organ failure	No	Total	Fischer exact test value	P value
11 to 20	2 50%	2 50%	4 100%	3.734	0.621
21 to 30	5 35.7%	9 64.3%	14 100%		
31 to 40	10 29.4%	24 70.6%	34 100%		
41 to 50	4 23.5%	13 76.5%	17 100%		
51 to 60	1 10%	9 90%	10 100%		
61 to 70	0	1 100%	1 100%		
Total	22 27.5%	58 72.5%	80 100%		

In participants with age of 11 to 20 years, 50% had organ failure and 50% did not have organ failure. In participants with age of 21 to 30 years, 35.7% had organ failure and 64.3% did not have organ failure. In participants with age of 31 to 40 years, 29.4% had organ failure

and 70.6% did not have organ failure. In participants with age of 41 to 50 years, 23.5% had organ failure and 76.5% did not have organ failure. In participants with age of 51 to 60 years, 10% had organ failure and 90% did not have organ failure. In participants with age of 61 to 70 years, 100% did not have organ failure. This difference was not statistically significant by Fischer exact test.

Chart 22. Association between age and organ failure among the study participants



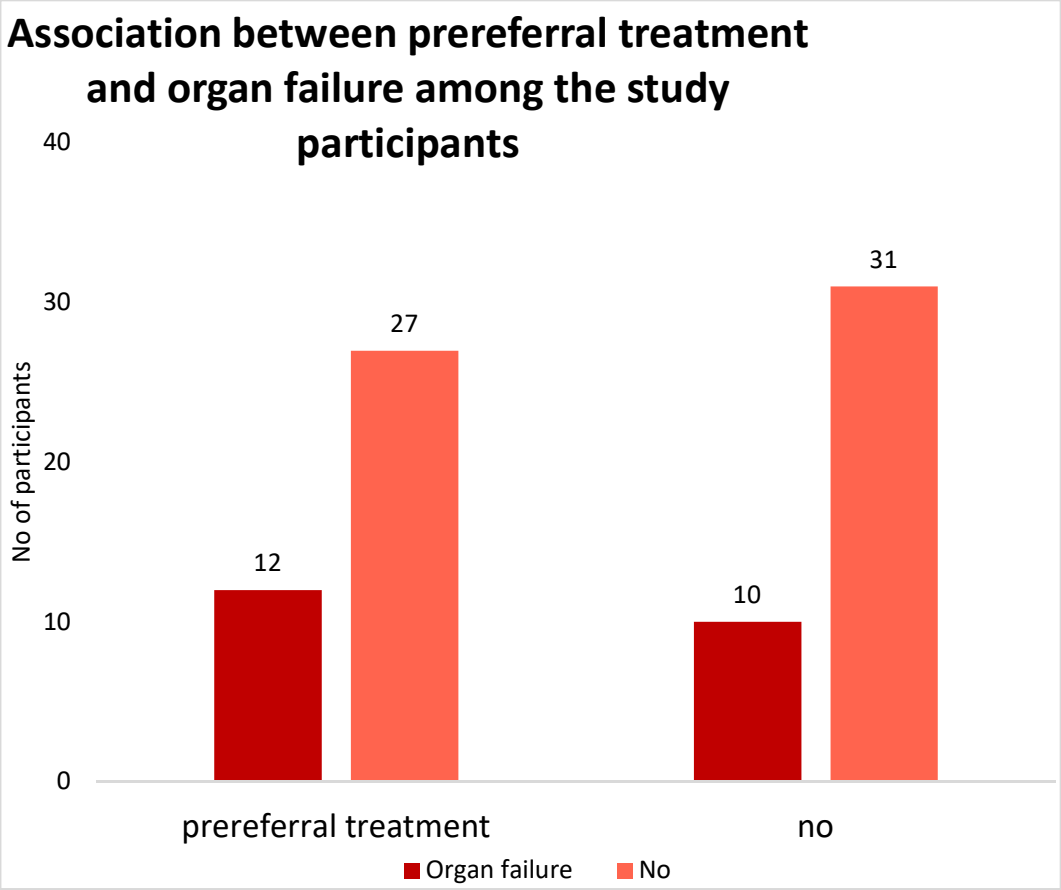
Association between prereferral treatment and organ failure among the study participants:

Table 23. Association between prereferral treatment and organ failure among the study participants

prereferral treatment	Organ failure	No	Total	Fischer exact test value	P value
Yes	12 30.8%	27 69.2%	39 100%	0.408	0.619
No	10 24.4%	31 75.6%	41 100%		
Total	22 27.5%	58 72.5%	80 100%		

In the study participants who had prereferral treatment, 30.8% had organ failure and 69.2% did not have organ failure. In the study participants who did not have prereferral treatment, 24.4% had organ failure and 75.6% did not have organ failure. This difference was not statistically significant by Fischer exact test.

Chart 23. Association between prereferral treatment and organ failure among the study participants



Association between comorbidities and organ failure among the study participants:

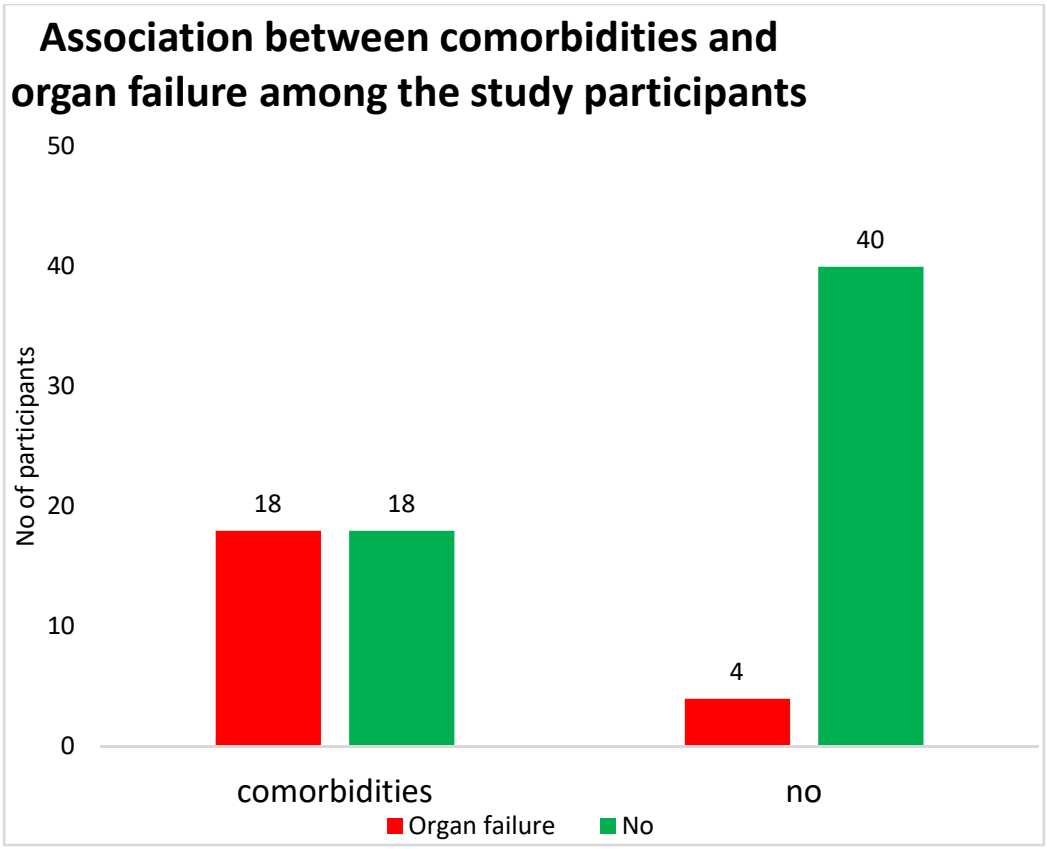
Table 24. Association between comorbidities and organ failure among the study participants

Comorbidities	Organ failure	No	Total	Fischer exact test value	P value
Yes	18 50%	18 50%	36 100%	27.42	**< 0.001***
No	4 9%	40 91%	44 100%		
Total	22 27.5%	58 72.5%	80 100%		

*- statistically significant by Fischer Exact test

In the study participants who had comorbidities, 50% had organ failure and 50% did not have organ failure. In the study participants who did not have comorbidities, 9% had organ failure and 91% did not have organ failure. This difference was statistically significant by Fischer exact test. ($p < 0.05$)

Chart 24. Association between comorbidities and organ failure among the study participants



Association between specific antidote and organ failure among the study participants:

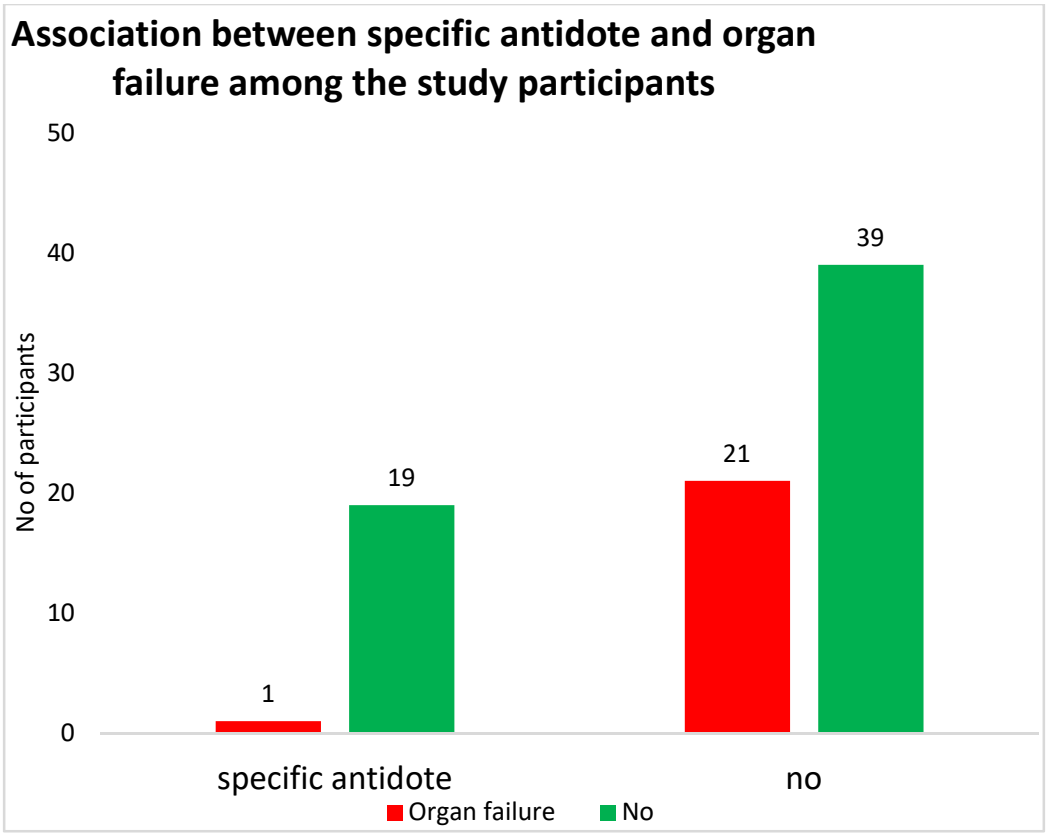
Table 25. Association between specific antidote and organ failure among the study participants

Specific antidote	Organ failure	No	Total	Fischer exact test value	P value
Yes	1 5%	19 95%	20 100%	6.771	**0.02***
No	21 35%	39 65%	60 100%		
Total	22 27.5%	58 72.5%	80 100%		

*- statistically significant by Fischer Exact test

In the study participants who were given specific antidote, 5% had organ failure and 95% did not have organ failure. In the study participants who were not give specific antidotes, 35% had organ failure and 65% did not have organ failure. This difference was statistically significant by Fischer exact test. ($p< 0.05$)

Chart 25. Association between specific antidote and organ failure among the study participants



Association between hypotension at admission and organ failure among the study participants:

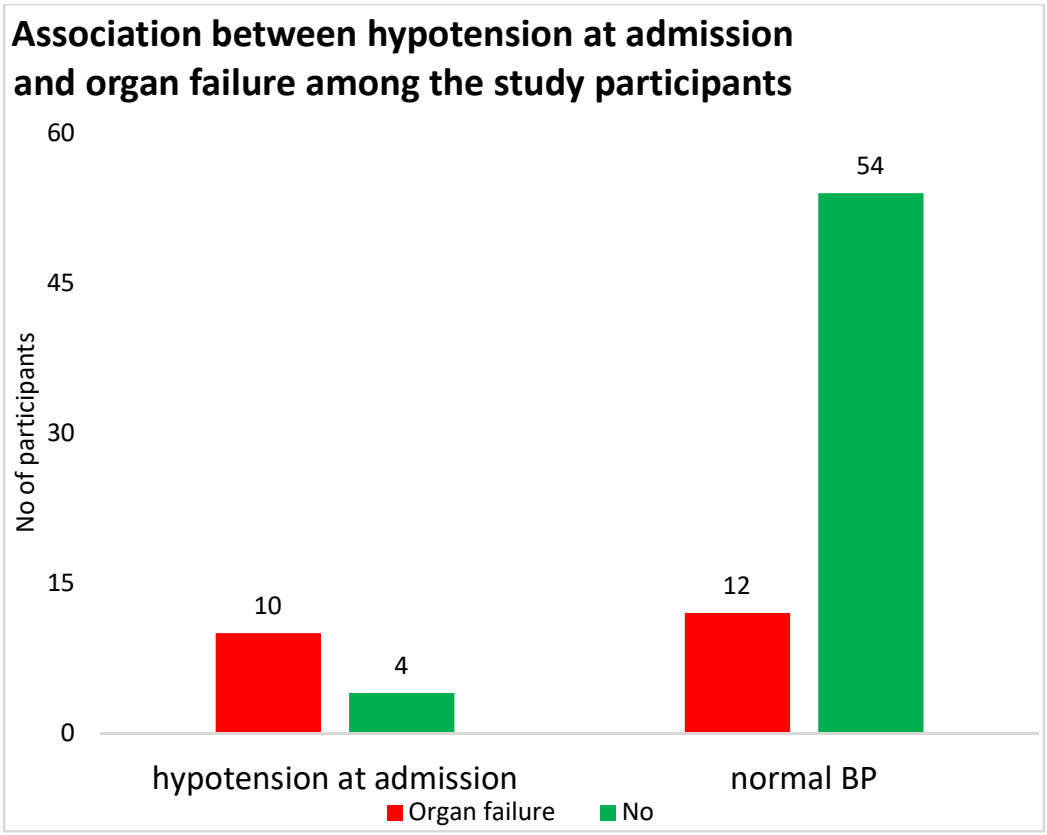
Table 26. Association between hypotension at admission and organ failure among the study participants

hypotension at admission	Organ failure	No	Total	Fischer exact test value	P value
Yes	10 71.4%	4 28.6%	14 100%	16.425	**<0.001***
No	12 18.2%	54 81.8%	66 100%		
Total	22 27.5%	58 72.5%	80 100%		

*- statistically significant by Fischer Exact test

In the study participants who had hypotension at admission, 71.4% had organ failure and 28.6% did not have organ failure. In the study participants who had normal blood pressure during admission, 18.2% had organ failure and 81.8% did not have organ failure. This difference was statistically significant by Fischer exact test. ($p< 0.05$)

Chart 26. Association between hypotension at admission and organ failure among the study participants



Association between class of drugs and organ failure among the study participants:

Table 27. Association between class of drugs and organ failure among the study participants

Class of drugs	Organ failure	No	Total
Alpha blocker	0	1 100%	1 100%
Anti-epileptic	3 60%	2 40%	5 100%
Anti fungals	0	1 100%	1 100%
Anti-helminthic	0	1 100%	1 100%
Anti-histamines	0	1 100%	1 100%
Anti-hypertensive	4 50%	4 50%	8 100%
Anti-psychotic	0	6 100%	6 100%
Anti-thyroid	0	5 100%	5 100%

Antibiotic	0	1 100%	1 100%
Barbiturate	1 100%	0	1 100%
Benzodiazepines	0	14 100%	14 100%
Beta blocker	2 28.6%	5 71.4%	7 100%
Calcium channel blocker	2 100%	0	2 100%
Multiple tablets	1 50%	1 50%	2 100%
NSAID	6 54.5%	5 45.5%	11 100%
Oral hypoglycemic	0	4 100%	4 100%
Opiod	0	1 100%	1 100%
Tetracyclines	0	1 100%	1 100%

Tricyclic antidepressant	3 100%	0	3 100%
Vitamins	0	5 100%	5 100%
Fischer exact test value= 37.190			
P value < **0.001***			

*- statistically significant by Fischer Exact test

The above table shows association between class of drugs and organ failure. In those who consumed antiepileptic, 60% had organ failure. In participants who consumed Anti-hypertensive 50% had organ failure. In those who consumed barbiturate, 100% had organ failure. In participants who consumed Beta blockers, 28.6% had organ failure. In those who consumed Calcium channel blocker, 100% had organ failure. In participants who consumed Multiple tablets, 50% had organ failure. In those who consumed NSAIDs, 54.5% had organ failure. In participants who consumed Tricyclic antidepressants, 100% had organ failure. This difference was statistically significant by Fischer exact test. ($p< 0.05$)

Discussion

In our study We are trying to give the data of acute drug poisoning cases admitted to our hospital over a period of 6 months.

In the present study 80 patients were admitted during the study period .

AGE DISTRIBUTION:

Mean age group of study participants was 38.3 +/- 10 yrs.

Majority of participants were from 31-40yrs.

17 participants were in 41-50 yrs of group, 14 were from 21 to 30 yrs of age group, 10 were from 51-60 yrs of age group,4 were from 11 to 20 yrs age group, and 1 Participant was above 6yrs.

GENDER:

Among the study participants,

36 participants were male .

44 participants were females.

Locality:

Among the socio economic status of the study participants,

42 patients were from lower middle class,

19 patients were from upper middle class,

15 patients were from upper lower class,

4 participants were from lower class.

EDUCATIONAL STATUS:

Among the study participants.

29 patients were graduate,

28 patients studied upto high school,

20 patients studied upto middle school,

3 patients were illiterate.

PRE REFERRAL TREATMENT:

Among the study participants,

39 patients had Pre referral treatment

41 patients did not have Pre referral treatment.

TYPE OF DRUG CONSUMED:

Among the total study participants,

Benzodiazepines are the most common drug poisoning , followed by non steroidal Anti inflammatory drugs, and anti hypertensives.

Among the total study participants, 14 participants had consumed Benzodiazepines followed by 11 participants had consumed NSAIDs.

8 participants had Anti-hypertensive, 7 had Beta blocker, 6 had Anti-psychotic, 5 had Anti-epileptic, 5 had Anti thyroid, 5 had Vitamins,

4 had Oral hypoglycemic, 3 had Tricyclic antidepressant, 2 had Calcium channel blocker, 2 had Multiple tablets, 1 had Alpha blocker, 1 had Anti fungals, 1 had Anti helminthic, 1 had Anti histamines, 1 had Antibiotic, 1 had Barbiturate, 1 had Opiod and 1 had Tetracyclines.

Quantity of drug consumed:

Mean quantity of tablets consumed by the participants were 32.6+/-12 tablets.

19 participants consumed 10 to 20 tablets,

29 patients consumed 21 to 30 tablets,

15 participants consumed 31 to 40 tablets,

13 patients consumed 41 to 50 tablets,

4 patients consumed 51 to 60 tablets.

DURATION OF STAY :

Mean duration of stay of the study participants was 4 -7 days.

17 patients had duration of stay of less than 4 days,

58 patients had duration of 4 to7 days,

5 had more than 7 days.

COMORBIDITIES

Among the study participants,

17 patients had hypertension, 11 patients had diabetes mellitus,

8 had psychiatric disorder, 5 patients had seizure disorder,

4 had CAD, 3 had thyroid disorder, 2 had depressive disorder,

44 did not have comorbidities.

Outcome:

All the study participants recovered and were discharged.

Association between time since consumption and organ failure among the study participants was statically significantly by Fischer exact test.

Assosciation between quantity of consumption and organ failure among the study participants was stastically significant.

Assosciation between comorbidities and organ failure among the study participants was statistically significant.

Assosciation between specific antidote and organ failure among the patients was statistically significant.

There is significant Assosciation between class of drugs and organ failure by Fischer exact test.

In those who had consumed anti epileptics, 60% had organ failure.

In those who had consumed anti hypertensives, 50% had organ failure.

In those who had consumed beta blockers,28.6% had organ failure.

In those who had consumed calcium channel blockers,100% had organ failure.

In those who had consumed multiple tablets,50% had organ failure.

In those who had consumed NSAIDS,54.5% had organ failure.

In those who had consumed tricyclic anti depressants,100% had organ failure.

CONCLUSIONS:

80 cases with acute drug poisoning were studied over a period of 6 months with following results:

In this study:

1. Mean age group of patients are 38-48 yrs.

2. Males constitute 45%, females constitute 45%.

3. Patients from urban constitute 55%, rural constitute 45%.

4. Mean time since consumption to arriving to hospital was 12-16hrs.

5. Pre referral treatment done in 51.2%.

6. Benzodiazepines are the most common drug poisoning in this study group, followed by NSAIDS,followed by ANTI HYPERTENSIVES.

7. Mean quantity of tablets consumed by the study participants were 32.26%.

8. Common symptoms of drug poisoning include vomiting,giddiness,abdominal pain.

9. Mean duration of stay of study participants was 4-6 days.

10. Specific antidote used in 25% of study participants.

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ANNEXURE

INFORMATION SHEET

We are conducting a study on "**A PROSPECTIVE STUDY OF EPIDEMIOLOGICAL, CLINICAL, TOXICITY, OUTCOME OF PATIENTS ADMITTED WITH HISTORY OF ACUTE DRUG POISONING IN A TERTIARY CARE CENTRE**", among patients attending Rajiv GandhiGovernment General Hospital, Chennai and for that your specimen may be valuable to us.

We are selecting certain cases and if you are found eligible, we may be using your blood samples to do certain tests, do certain non invasive radiological investigations.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled

Signature of the Investigator. Signature of the Participant

PATIENT CONSENT FORM

A PROSPECTIVE STUDY OF EPIDEMIOLOGICAL, CLINICAL, TOXICITY, OUTCOME OF PATIENTS ADMITTED WITH HISTORY OF ACUTE DRUG POISONING IN A TERTIARY CARE CENTRE.

Participant Name :

Age:

Sex:

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask the questions and all my questions and doubts have been answered to my satisfaction.

I have been explained about the pitfall in the procedure. I have been explained about the safety , advantage and disadvantage of the technique.

I understand that my participation in the study is voluntary and that I'm free to withdraw at anytime without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and

100

any further research that may be conducted in relation to it, even if I withdraw from the study.

I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.I hereby consent to undergo complete physical examination, pathological and radiological investigation pertaining to the study.

I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.I hereby consent to undergo complete physical examination, pathological and radiological investigation pertaining to the Study.

Signature/ Thumb impressions of participants

CERTIFICATE - II

This is to certify that this dissertation work titled "**A PROSPECTIVE STUDY OF EPIDEMIOLOGICAL, CLINICAL, TOXICITY, OUTCOME OF PATIENTS ADMITTED WITH HISTORY OF ACUTE DRUG POISONING IN A TERTIARY CARE CENTRE**"of the candidate DR. R.N .MADHURIMA with registration Number 200120100525 for the award of the degree of M.D. in the branch of Internal Medicine. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 2 percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

Dr. S. USHALAKSHMI, MD, FMMC
Professor of Medicine
Institute of Internal Medicine
Madras Medical College & RGGGH
Chennai-600 003.

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg. No(CDSCO).ECR/270/Inst./TN/2013/RR-20
EC Reg. No(DHR).EC/NEW/INST/2021/1618
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CERTIFICATE OF APPROVAL

To
Dr.R.N.MADHURIMA,
MD Internal Medicine Post Graduate Student,
Institute of Internal Medicine,
Madras Medical College,
Chennai – 600 003.

Dear Dr. R.N.MADHURIMA,

The Institutional Ethics Committee has considered your request and approved your study titled **"A PROSPECTIVE STUDY OF EPIDEMIOLOGICAL, CLINICAL, TOXICITY, OUTCOME OF PATIENTS ADMITTED WITH HISTORY OF ACUTE DRUG POISONING IN A TERTIARY CARE CENTRE"-NO.30052022** in the meeting held on **04.05.2022** conducted at Madras Medical College, Chennai 3.

1. Prof.P.V.Jayashankar,MS Orth.,D.Orth.,M.Ch Orth (Liverpool) :Chairperson
2. Prof.N.Gopalakrishnan,MD.,DM., FRCP, Director, Inst.of Nephrology,MMC,Ch. : Member Secretary
3. Prof. K.M.Sudha, Prof. Inst. of Pharmacology,MMC,Ch-3 : Member
4. Prof. Alagarsamy Jamila ,MD, Vice Principal, Stanley Medical College, Chennai : Member
5. Prof.Meena Suresh, MD.,DGO.,Prof.of Obst & Gynaec, IOG,Chennai : Member
6. Prof.S.Lakshmi, Prof. of Paediatrics ICH Chennai :Member
7. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
8. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
9. Thiru K.Ranjith, Ch- 91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003.

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S.no	Name	Age	agecat	Sex	Address	Socio economic status	Educational status	Time since consumpion	Prereferral treatment	Type of drug	Class	Intention of poisoning	Quantity	quantityca	Symptoms during presentation		Pulse	Blood pressure	Spo2	Resp.rate	CBC	Coagulation profile
1	Kavipreetha	28	21to30	F	Urban	UM	G	6	No	Amlodipine	Antihypertensive	Suicidal	30	21to30	Vomitings,altered sensorium		102	80/50 mmhg	98 Ra	22	Tc:18k plt: 4lac	Normal
2	Anandhi	55	51to60	F	Urban	LM	HS	12	Yes	Griseofulvin	Anti fungals	Suicidal	10	10to20	Vomitings		86	120/80 mmhg	99Ra	18	Tc:7.8 hb:10	Normal
3	Thangavel	42	11to50	M	Rural	UL	HS	4	No	Alprazolam	Benzodiazepines	Suicidal	10	10to20	Altered sensorium		82	130/80 mmhg	98 Ra	12	Tc:6.4 hb:13.6	Normal
4	Rani	44	11to50	F	Rural	UL	G	10	Yes	10 cetrizine	Anti histamines	Suicidal	20	10to20	Drowsiness		97	120/80	99 ra	16	Tc:11k hb:11.3	Normal
5	Mallga begum	36	31to40	F	Urban	LM	HS	4	No	50 trihexyphenidyl	Tricyclic antidepressants	Suicidal	50	41to50	Unconsciousness		97	90/70 mmhg	97 ETMV	22	Tc:12k hb: 15	Normal
6	Varsha	24	21to30	F	Rural	UM	G	6	No	Eltroxin	Thyroid disorder	Suicidal	20	10to20	Vomitings		84	110/80 mmhg	99 RA	18	Tc:5k hb:14	Normal
7	Vinodha	33	31to40	F	Urban	LM	G	8	No	Paracetamol	Analgesic	Suicidal	30	21to30	Vomitings,abdominal pain		94	110/70mmhg	97RA	22	Tc:6k hb:15	Normal
8	Gowthami	29	21to30	F	Urban	UL	HS	12	Yes	Metoprolol	Betablockers	Suicidal	20	10to20	Giddiness		84	90/60 mm hg	98 Ra	20	Tc:8k hb: 14	Normal
9	Rajeshwari	38	31to40	F	Rural	L	IL	8	No	Imipramine	Tricyclic antidepressant	Suicidal	30	21to30	Drowsiness/vomitings		131	100/60 mmhg	99Ra	20	Tc:9k hb:14	Normal
10	Hemalatha	52	51to60	F	Rural	LM	MS	7	No	B complex + vitc	Vitamins	Suicidal	45	41to50	Abdominal pain vomitings		76	110/70 mmhg	97 Ra	18	Tc :11k hb : 12	Normal
11	Selvi	36	31to40	F	Rural	LM	HS	8	No	Metronidazole	Anti helminthic	Suicidal	20	10to20	Abdominal pain/loose stools/		88	100/70 mmhg	97 Ra	20	Tc:8k hb: 11	Normal
12	Harish	19	11to20	F	Urban	LM	HS	8	No	Phenytoin	Anti epileptic	Suicidal	30	21to30	Drowsiness		121	90/60 mmhg	98ra	18	Tc: 8k hb: 12	Normal
13	Devi	38	31to40	F	Rural	LM	MS	14	Yes	Enalapril	Anti hypertensive	Suicidal	20	10to20	Giddiness, palpitations		105	80/50 mmhg	98 ra	24	Tc:9k hb:12	Normal
14	Karthik	55	51to60	M	Urban	UM	G	18	Yes	Meftol+glipiride	Multiple tablets	Suicidal	40	31to40	Abdominal pain vomitings		104	110/70 mmhg	98 Ra	18	Tc:6k hb:12	Normal
15	Udhaya	36	31to40	F	Rural	LM	HS	8	No	Metformin	Oral antihypoglycemic	Suicidal	20	10to20	Giddiness/palpitations		126	100/70 mmhg	98 ra	20	Tc:10k hb:10	Normal
16	Deepthi	28	21to30	F	Urban	LM	G	16	Yes	Phenobarbitone	Barbiturate	Suicidal	30	21to30	Drowsiness/vomitings		120	90/60 mm hg	94%RA	26	Tc: 10k hb:10	Normal
17	Jai shankar	45	11to50	M	Rural	UM	MS	18	Yes	Aspirin	Salicylates	Suicidal	20	10to20	Vomitings/diaphoresis/confusion		105	100/70 mmhg	98 %RA	20	Tc:7k hb:12	Normal
18	Murali	36	31to40	M	Urban	LM	G	20	Yes	Bisoprolol	Betablockers	Suicidal	25	21to30	Giddiness/vomitings		62	110/70 mmhg	97 %RA	16	Tc:10k hb:12	Normal
19	Manjula	45	11to50	F	Rural	UM	MS	16	Yes	Levetiracetam	Anti epileptic	Suicidal	20	10to20	Drowsiness/vomitings		86	100/70 mmhg	98 %RA	28	Tc:8k hb:14	Normal
20	Velu	32	31to40	M	Rural	LM	MS	20	Yes	Amlodipine	Calcium channel blocker	Suicidal	45	41to50	Giddiness/abdominal pain		76	80/60 mmhg	99 RA	16	Tc:11k hb:10	Normal
21	Nandini	23	21to30	F	Urban	UM	G	16	Yes	Diazepam	Benzodiazepines	Suicidal	25	21to30	Unconsciousness		84	100/70 mmhg	98Ra	18	Tc: 10k hb:10	Normal
22	Prem	45	11to50	M	Rural	LM	HS	20	Yes	Metoprolol	Betablockers	Suicidal	20	10to20	Giddiness		59	90/60 mm hg	97 %RA	16	Tc :11k hb : 12	Normal
23	Preethi	27	21to30	F	Rural	L	HS	8	No	Paracetamol	NSAID	Suicidal	30	21to30	Vomitings,abdominal pain		75	100/60mmhg	98ra	18	Tc:12 hb:14	Normal
24	Balaji	36	31to40	M	Urban	LM	G	6	No	Doxycycline	Tetracyclines	Suicidal	10	10to20	Abdominal pain		86	100/60 mmhg	99 RA	16	Tc :11k hb : 12	Normal
25	Sreedhar	62	61to70	M	Urban	UM	G	18	Yes	Metronidazole	Antibiotic	Suicidal	30	21to30	Loosestools,abdominal pain		85	100/60 mmhg	98 %RA	20	Tc: 10k hb:10	Normal
26	Nagalakshmi	45	11to50	F	Rural	LM	HS	20	Yes	Diazepam	Benzodiazepines	Suicidal	60	51to60	Unconsciousness		78	110/70 mmhg	95 %RA	16	Tc:12 hb:14	Normal
27	Jagadeesh	39	31to40	M	Urban	LM	MS	6	No	Paracetamol	NSAID	Suicidal	36	31to40	Vomitings/abdominal pain		65	110/70 mmhg	98 %RA	18	Tc:11k hb:11.3	Normal
28	Siva	50	11to50	M	Rural	L	HS	24	Yes	Metformin +glimiperide	Oral hypoglycemic	Suicidal	25	21to30	Giddiness/palpitations		76	120/70 mmhg	97 %RA	16	Tc: 8k hb: 12	Normal
29	Geetha	45	11to50	F	Urban	LM	HS	16	Yes	Ferrous sulphate	Vitamins+minerals	Suicidal	20	10to20	Vomitings/abdominal pain		76	110/70 mmhg	98%RA	18	Tc: 8k hb: 12	Normal
30	Varsha	34	31to40	F	Rural	LM	MS	14	Yes	Propranolol	Beta blocker	Suicidal	30	21to30	Giddiness		86	130/80 mmhg	97 %RA	20	Tc:10k hb:12	Normal
31	Chandrasekhar	36	31to40	M	Urban	UM	G	18	Yes	Paracetamol	NSAID	Suicidal	60	51to60	Vomitings		76	110/70mmhg	98 %RA	16	Tc: 8k hb: 12	Normal
32	Saravanan	38	31to40	M	Rural	LM	MS	5	No	Diazepam	Benzodiazepines	Suicidal	30	21to30	Altered sensorium		75	120/70 mmhg	99 RA	18	Tc:11k hb:11.3	Normal
33	Sandhya	47	11to50	F	Urban	UM	MS	10	Yes	Amlodipine	Calcium channelblocker	Suicidal	25	21to30	Vomitings		76	80/60 mm hg	99ra	20	Tc:11k hb:15	Normal
34	Kowsalya	54	51to60	F	Rural	LM	HS	10	No	Aspirin	NSAID	Suicidal	30	21to30	Vomitings		89	100/60 mmhg	98 %RA	16	Tc: 8k hb: 12	Normal
35	Bhavana	46	11to50	F	Urban	LM	G	16	Yes	Tramadol	Sedatives	Suicidal	15	10to20	Giddiness		76	100/70 mmhg	99 RA	18	Tc:11k hb:11.3	Normal
36	Swathi	28	21to30	F	Rural	LM	HS	8	No	Eltroxin	Anti thyroid	Suicidal	30	21to30	Giddiness/abdominal pain		86	130/80 mmhg	98 %RA	16	Tc :11k hb : 12	Normal
37	Reshmi	20	11to20	F	Urban	UM	G	3	No	Diazepam	Benzodiazepines	Suicidal	40	31to40	Altered sensorium		98	110/70 mmhg	99%ra	18	Tc:12 hb:14	Normal
38	Raja	38	31to40	M	Urban	LM	MS	12	No	Phenobarbitone	Benzodiazepines	Suicidal	30	21to30	Altered sensorium		96	100/60mmhg	99 ra	18	Tc:10k hb:10	Normal
39	Savithri	45	11to50	F	Urban	UL	MS	18	Yes	Para+morphine	Multiple tablets	Suicidal	50	41to50	Giddiness		98	110/70 mmhg	96 Ra	20	Tc: 8k hb: 12	Normal
40	Sateesh	30	21to30	M	Urban	LM	G	6	No	Telmisartan	Anti hypertensive	Suicidal	25	21to30	Giddiness		72	120/80	98 %RA	18	Tc :11k hb : 12	Normal
41	Deena	45	11to50	M	Rural	UL	HS	8	No	Calcium ,Bct	Vitamins	Suicidal	40	31to40	No symptoms		86	110/70 mmhg	97ra	16	Tc:10k hb:12	Normal
42	Arul	35	31to40	M	Urban	LM	MS	6	No	Carbamazepine	Anti epileptic	Suicidal	30	21to30	Seizures		98	90/60 mmhg	98 Ra	18	Tc:10k hb:12	Normal
43	Raghu	50	11to50	M	Urban	UL	G	15	Yes	Paracetamol	NSAID	Suicidal	45	41to50	Vomitings		87	100/60 mmhg	97 ra	16	Tc: 8k hb: 12	Normal
44	Yogesh	31	31to40	M	Urban	UM	HS	8	No	Diazepam	Benzodiazepines	Suicidal	50	41to50	Altered sensorium		96	110/60 mmhg	98 %RA	20	Tc:10k hb:10	Normal
45	Anasuya	56	51to60	F	Rural	LM	MS	10	No	Chlorpromazine	Anti psychotic	Suicidal	20	10to20	Vomitings		87	100/70 mmhg	99 RA	18	Tc: 8k hb: 12	Normal
46	Antony	19	11to20	M	Urban	UL	HS	4	No	Alprazolam	Benzodiazepines	Suicidal	50	41to50	Altered sensorium		98	90/60 mm hg	98ra	20	Tc :11k hb : 12	Normal
47	Arumugam	38	31to40	M	Rural	LM	MS	16	Yes	Prazosin	Alpha blocker	Suicidal	30	21to30	Giddiness		99	100/60 mmhg	96 Ra	30	Tc:10k hb:10	Normal
48	Keerthi	26	21to30	F	Urban	UL	G	7	No	Paracetamol	NSAID	Suicidal	60	51to60	Vomitings		121	100/70 mmhg	98ra	22	Tc :11k hb : 13	Normal
49	Suriya	36	31to40	M	Urban	LM	HS	8	No	Diazepam	Benzodiazepines	Suicidal	45	41to50	Altered sensorium		76	110/70 mmhg	97RA	16	Tc:10k hb:14	Normal
50	Raju	45	11to50	M	Urban	UM	G	16	Yes	Bisoprolol	Beta blockers	Suicidal	30	21to30	Giddiness		56	100/70 mmhg	98Ra	18	Tc:10k hb:10	Normal
51	Rangammal	52	51to60	F	Urban	LM	HS	20	No	Eltroxin	Anti thyroid	Suicidal	20	10to20	Vomitings		76	110/70 mmhg	97Ra	16	Tc :11k hb : 12	Normal
52	Danish	52	51to60	M	Urban	UM	G	3	Yes	Metformin	Oral hypoglycemic	Suicidal	35	31to40	Vomitings,giddiness		97	100/70 mmhg	98 %RA	14	Tc: 8k hb: 12	Normal
53	Indrani	36	31to40	F	Urban	LM	HS	14	Yes	Telmisartan	Anti hypertensive	Suicidal	30	21to30	Giddiness		66	110/70 mmhg	97 %RA	18	Tc: 10k hb:10	Normal
54	Thangam	40	31to40	F	Rural	UL	IL	20	Yes	Amlodipine	Anti hypertensive	Suicidal	40	31to40	Giddiness		56	80/60 mmhg	98 %RA	16	Tc :11k hb : 12	Normal
55	Sakthivel	28	21to30	M	Rural	UL	G	16	Yes	Metoprolol	Beta blocker	Suicidal	25	21to30	Giddiness		66	110/70mmhg	99 RA	18	Tc: 10k hb:10	Normal
56	Nagaraj	36	31to40	M	Urban	LM	G	20	Yes	Alprazolam	Benzodiazepines	Suicidal	35	31to40	Altered sensorium		78	100/80 mmhg	97 %RA	20	Tc: 8k hb: 12	Normal
57	Boopalan	42	11to50	M	Rural	UL	HS	18	No	Chlorpromazine	Anti psychotic	Suicidal	45	41to50	Vomitings		98	110/70 mmhg	96 Ra	14	Tc:10k hb:12	Normal
58	Rajesh	36	31to40	M	Urban	LM	G	10	Yes	Riserpine	Anti psychotic	Suicidal	35	31to40	Abdominal pain		99	100/60 mmhg	97 %RA	18	Tc :11k hb : 12	Normal
59	Vishal	19	11to20	M	Urban	LM	HS	16	Yes	Paracetamol	NSAID	Suicidal	15	10to20	Vomitings		96	100/70 mmhg	98 %RA	17	Tc: 10k hb:10	Normal
60	Prem	28	21to30	M	Urban	UM	G	15	Yes	Aspirin	NSAID	Suicidal	20	10to20	Abdominal pain		97	130/80 mmhg	98 %RA	16	Tc :11k hb : 13	Normal
61	Madhuri	31	31to40	F	Rural	LM	MS	20	Yes	Paracetamol	NSAID	Suicidal	45	41to50	Abdominal pain		87	120/70 mmhg	99 RA	18	Tc :11k hb : 13	Normal
62	Devi	42	11to50	F	Urban	LM	HS	6	No	Enalapril	Anti hypertensive	Suicidal	50	41to50	Giddiness		76	120/70 mmhg	96 Ra	20	Tc: 8k hb: 12	Normal
63	Lawrence	34	31to40	M	Rural	LM	MS	8	No	Eltroxin	Anti thyroid	Suicidal	40	31to40	Abdominal pain		87	110/60 mmhg	98 %RA	22	Tc: 10k hb:10	Normal
64	Deepthi	29	21to30	F	Urban	UL	G	9	No	Alprazolam	Benzodiazepines	Suicidal	30	21to30	Altered sensorium		98	100/60 mmhg	98 %RA	18	Tc :11k hb : 13	Normal
65	Moorthy	40	31to40	M	Urban	LM	G	3	No	Phenytoin	Anti epileptic	Suicidal	35	31to40	Seizures		84	110/70 mmhg	96 Ra	19	Tc: 8k hb: 12	Normal
66	Prasad	39	31to40	M	Urban	UM	HS	10	Yes	Levetiracetam	Anti epileptic	Suicidal	40	31to40	Altered sensorium		82	100/70 mmhg	98 %RA	18	Tc :11k hb : 12	Normal
67	Amsaveni	56	51to60	F	Rural	LM	MS	4	No	Calcium ,Bct	Vitamins	Suicidal	25	21to30	No symptoms		77	110/70 mmhg	99 RA	18	Tc :11k hb : 13	Normal
68	Manjula	23	21to30	F	Rural	UL	HS	12	Yes	Amlodipine	Anti hypertensive	Suicidal	30	21to30	Giddiness		67	80/60mmhg	98 %RA	18	Tc: 10k hb:10	Normal
69	Rani	38	31to40	F	Rural	UM	MS	16	No	Eltroxin	Anti thyroid	Suicidal	25	21to30	Vomitings		66	100/60 mmhg	99Ra	16	Tc :11k hb : 12	Normal
70	Selvi	40	31to40	F	Urban	LM	HS	14	No	Alprazolam	Benzodiazepines	Suicidal	30	21to30	Altered sensorium		85	110/70 mmhg	97 %RA	17	Tc: 8k hb: 12	Normal
71	Valarmathi	38	31to40	F	Rural	UM	MS	6	No	Chlorpromazine	Anti psychotic	Suicidal	45	41to50	Abdominal pain		76	100/60mmhg	98 %RA	14	Tc:10k hb:12	Normal
72	Narmatha	23	21to30	F	Rural	UL	HS	10	Yes	Bct calcium	Vitamins	Suicidal	20	10to20	No symptoms		87	110/70 mmhg	97 %RA	16	Tc: 8k hb: 12	Normal
73	Abinaya	37	31to40	F	Urban	LM	G	20	Yes	Trihexyphenidyl	Tricyclic antidepressant	Suicidal	40	31to40	Altered sensorium		98	100/70 mmhg	98 Ra	14	Tc:10k hb:14	Normal
74	Rajeswari	36	31to40	F	Rural	LM	G	20	Yes	Riserpine	Anti psychotic	Suicidal	35	31to40	Vomitings		92	100/60mmhg	98 %RA	18	Tc:11k hb:11.3	Normal
75	Muthu	60	51to60	M	Urban	UM	MS	72	No	Diazepam	Benzodiazepines	Suicidal	45	41to50	Altered sensorium		66	100/70 mmhg	97 %RA	16	Tc:11k hb:11.3	Normal
76	Murali	56	51to60	M	Rural	L	IL	3	No	Amlodipine	Anti hypertensive	Suicidal	60	51to60	Giddiness		57	80/50 mmhg	96 Ra	16	Tc:10k hb:14	Normal
77	Gokul	40	31to40	M	Urban	UM	G	4	No	Metoprolol	Beta blockers	Suicidal	30	21to30	Giddiness		66	90/60 mm hg	97 %RA	26	Tc: 10k hb:10	Normal
78	Shiva	38	31to40	M	Urban	LM	G	7	No	Metformin	Oral hypoglycemic	Suicidal	40	31to40	Vomitings,giddiness		89	100/60 mmhg	98 %RA	17	Tc:10k hb:12	Normal
79	Ragavi	42	11to50	F	Rural	LM	HS	10	Yes	Risperidone	Anti psychotic	Suicidal	35	31to40	Abdominal pain vomitings		101	110/70 mmhg	96 Ra	18	Tc :11k hb : 12	Normal
80	Bhanu	38	31to40	F	Rural	UL	G	12	No	Zolpidem	Benzodiazepines	Suicidal	25	21to30	Giddiness		67	100/60 mmhg	97 %RA	17	Tc: 10k hb:10	Normal

urea	creatinine	LFT	sodium	potassium	Urine analysis	Abg	Ecg	Duration of stay	duration of stay	Specific antidote used	Outcome	organ failure	no comorbids	dm	htn	cad	thyroid dis	psychiatric	depressive	seizure dis
36	0.9	Normal	132	4.9	Negative	Normal	Normal	4	4to7days	Yes	Discharge	Yes	No comorbids							
36	1.1	Normal	132	4.6	Negative	Normal	Normal	3	<4days	No	Discharge	No	No comorbids							
28	0.8	Normal	139	4.7	Positive	Normal	Normal	3	<4days	No	Discharge	No	No comorbids							
30	0.8	Normal	140	3.6	Negative	Normal	Normal	2	<4days	No	Discharge.	No						Psychiatric	D	
30	1.2	Deranged	140	4.5	Positive	Metabolic acidosis	Bradykardia	10	>7days	No	Patient was extubated after 1 week and discharged	Yes	No comorbids							
38	0.8	Normal	141	5	Negative	Normal	Normal	5	4to7days	No	Discharge	No	No comorbids							
40	0.7	Deranged	138	3.6	Positive	Normal	Normal	6	4to7days	Yes	Discharge	Yes	No comorbids							
32	0.8	Normal	140	4.5	Positive	Normal	Normal	4	4to7days	No	Discharge.	Yes	No comorbids							
30	1.2	Normal	132	4.9	Positive	Normal	Prolonged Qt	6	4to7days	Yes	Discharge	Yes						Psychiatric	D	
28	0.8	Normal	142	3.9	Negative	Normal	Normal	2	<4days	No	Discharge	No	No comorbids							
36	1.1	Normal	142	3.8	Negative	Normal	Normal	2	<4days	No	Discharge.	No	No comorbids							
26	0.8	Deranged	139	4.7	Positive	Normal	Normal	3	<4days	No	Discharge	Yes								Seizure di
36	0.6	Normal	138	3.6	Negative	Normal	Normal	3	<4days	No	Discharge	No			HTN					
28	0.8	Normal	132	4.6	Negative	Normal	Normal	3	<4days	No	Discharge	No	No comorbids							
30	0.8	Normal	132	3.8	Negative	Normal	Normal	3	<4days	No	Discharge	No		Dm	HTN					
28	0.8	Deranged	138	4.6	Positive	Normal	Normal	4	4to7days	No	Discharge	Yes	No comorbids							
28	0.9	Normal	142	0.8	Positive	Metabolic acidosis	Normal	3	<4days	No	Discharge	No		Dm		CAD				
38	0.6	Normal	132	3.8	Negative	Normal	Normal	4	4to7days	No	Discharge	No			HTN	CAD				
36	1.1	Deranged	132	4.6	Negative	Normal	Normal	3	<4days	No	Discharge	Yes								Seizure di
32	0.8	Normal	140	3.6	Negative	Normal	Normal	10	>7days	No	Discharge	Yes			HTN					
26	0.8	Deranged	132	3.8	Negative	Normal	Normal	4	4to7days	No	Discharge	No							Depressive	disorder
36	0.6	Normal	132	3.8	Negative	Normal	Normal	3	<4days	No	Discharge	No			HTN					
40	0.7	Deranged	140	4.5	Positive	Normal	Normal	5	4to7days	Yes	Discharge	No	No comorbids							
28	0.8	Normal	140	3.6	Negative	Normal	Normal	5	4to7days	Yes	Discharge	No	No comorbids							
36	0.6	Normal	140	3.6	Negative	Normal	Normal	3	<4days	No	Discharge.	No			HTN	CAD				
28	0.8	Normal	142	3.8	Positive	Normal	Normal	7	4to7days	No	Discharge	No						Psychiatric	D	
28	0.9	Deranged	132	3.8	Positive	Normal	Normal	5	4to7days	Yes	Discharge	Yes			HTN	CAD				
36	0.6	Normal	132	4.6	Negative	Normal	Normal	3	<4days	No	Discharge.	No		Dm	HTN					
36	0.6	Normal	140	4.5	Negative	Normal	Normal	2	<4days	No	Discharge.	No	No comorbids							
28	0.9	Normal	142	0.8	Negative	Normal	Bradykardia	4	4to7days	Yes	Discharge	No	No comorbids							
28	0.9	Deranged	140	3.6	Positive	Normal	Normal	6	4to7days	Yes	Discharge	No		Dm	HTN					
26	0.8	Normal	140	4.5	Positive	Normal	Normal	5	4to7days	No	Discharge	No	No comorbids							
38	0.6	Normal	139	4.7	Positive	Normal	Normal	6	4to7days	Yes	Discharge	Yes	No comorbids							
36	0.6	Normal	132	3.8	Positive	Resp.acidosis	Normal	5	4to7days	No	Discharge	No	No comorbids							
40	0.7	Normal	142	3.9	Positive	Normal	Normal	6	4to7days	Yes	Discharge	No	No comorbids							
36	0.6	Normal	132	3.8	Negative	Normal	Normal	4	4to7days	No	Discharge	No	No comorbids							
28	0.9	Deranged	140	3.6	Positive	Normal	Normal	6	4to7days	No	Discharge	No	No comorbids							
36	1.1	Deranged	132	4.9	Positive	Normal	Normal	6	4to7days	No	Discharge	No	No comorbids							
28	0.8	Deranged	140	3.9	Positive	Normal	Normal	7	4to7days	Yes	Discharge	Yes	No comorbids							
28	0.9	Normal	140	4.5	Negative	Normal	Normal	4	4to7days	No	Discharge	No	No comorbids							
36	1.1	Normal	139	4.7	Negative	Normal	Normal	3	<4days	No	Discharge	No	No comorbids							
30	0.8	Deranged	139	4.7	Negative	Normal	Normal	4	4to7days	No	Discharge	No								Seizure di
28	0.8	Deranged	140	3.9	Positive	Normal	Normal	6	4to7days	Yes	Discharge.	Yes	No comorbids							
38	0.8	Normal	141	5	Positive	Normal	Normal	7	4to7days	No	Discharge	No	No comorbids							
36	0.6	Normal	132	3.8	Positive	Normal	Normal	4	4to7days	No	Discharge	No						Psychiatric	D	
30	1.2	Normal	138	3.6	Positive	Normal	Normal	4	4to7days	No	Discharge	No	No comorbids							
36	0.9	Normal	132	4.6	Negative	Normal	Normal	5	4to7days	No	Discharge	No	No comorbids							
26	0.8	Deranged	141	5	Positive	Metabolic acidosis	Normal	8	>7days	Yes	Discharge	Yes	No comorbids							
24	0.8	Normal	132	3.8	Negative	Normal	Normal	4	4to7days	No	Discharge	No	No comorbids							
32	0.8	Normal	132	4.9	Negative	Normal	Normal	6	4to7days	No	Discharge	No	No comorbids							
40	0.7	Normal	141	5	Negative	Normal	Normal	4	4to7days	No	Discharge	No					Thyroid	disorder		
36	0.9	Normal	132	4.9	Negative	Normal	Normal	5	4to7days	No	Discharge	No		Dm	HTN					
36	0.9	Normal	132	4.6	Negative	Normal	Normal	6	4to7days	No	Discharge	No		Dm	HTN					
36	0.9	Normal	132	4.9	Negative	Normal	Normal	6	4to7days	Yes	Discharge	Yes		Dm	HTN					
28	0.8	Normal	140	3.9	Negative	Normal	Normal	5	4to7days	No	Discharge	No		Dm	HTN					
32	0.8	Deranged	142	3.9	Positive	Normal	Normal	6	4to7days	No	Discharge	No	No comorbids							
38	0.8	Normal	139	4.7	Negative	Normal	Normal	5	4to7days	No	Discharge	No						Psychiatric	D	
45	0.9	Normal	139	4.7	Negative	Normal	Qt prolongation	6	4to7days	No	Discharge	No	No comorbids							
36	0.9	Deranged	132	3.8	Positive	Metabolic acidosis	Normal	7	4to7days	Yes	Discharge	Yes	No comorbids							
36	1.1	Normal	132	4.6	Positive	Normal	Normal	4	4to7days	No	Discharge	No	No comorbids							
30	0.8	Deranged	138	3.6	Positive	Normal	Normal	6	4to7days	Yes	Discharge	Yes	No comorbids							
30	1.2	Normal	139	4.7	Negative	Normal	Normal	4	4to7days	No	Discharge	No		Dm	HTN					
38	0.8	Normal	132	4.6	Negative	Normal	Normal	5	4to7days	No	Discharge	No					Thyroid	disorder		
30	0.8	Deranged	132	4.6	Positive	Normal	Normal	4	4to7days	No	Discharge	No	No comorbids							
36	1.1	Deranged	132	4.6	Positive	Normal	Normal	5	4to7days	No	Discharge	Yes								Seizure di
38	0.8	Normal	138	3.6	Negative	Normal	Normal	4	4to7days	No	Discharge	No								Seizure di
36	0.9	Normal	140	3.6	Negative	Normal	Normal	5	4to7days	No	Discharge	No	No comorbids							
36	0.6	Normal	140	4.5	Negative	Normal	Normal	8	>7days	Yes	Discharge	Yes			HTN					
36	1.1	Normal	132	4.6	Negative	Normal	Normal	5	4to7days	No	Discharge	No					Thyroid	disorder		
36	0.9	Deranged	132	4.9	Positive	Normal	Normal	4	4to7days	No	Discharge	No	No comorbids							
28	0.8	Normal	140	3.6	Negative	Normal	Normal	5	4to7days	No	Discharge	No							Depressive	disorder
36	0.9	Normal	132	4.9	Negative	Normal	Normal	2	<4days	No	Discharge	No	No comorbids							
32	0.8	Deranged	140	3.6	Positive	Metabolic acidosis	Qt prolongation	6	4to7days	Yes	Discharge	Yes						Psychiatric	D	
32	0.8	Normal	140	3.9	Negative	Normal	Normal	5	4to7days	No	Discharge	No						Psychiatric	D	
28	0.9	Normal	140	4.5	Positive	Normal	Normal	4	4to7days	No	Discharge	No	No comorbids							
38	0.8	Normal	142	3.8	Negative	Normal	Normal	8	>7days	Yes	Discharge	Yes		Dm	HTN					
36	0.9	Normal	140	3.6	Negative	Normal	Normal	5	4to7days	Yes	Discharge	Yes		Dm	HTN					
38	0.8	Normal	132	3.8	Negative	Normal	Normal	6	4to7days	No	Discharge	No	No comorbids							
36	0.6	Normal	139	4.7	Negative	Normal	Normal	5	4to7days	No	Discharge	No						Psychiatric	D	
36	0.9	Normal	140	3.6	Negative	Normal	Normal	6	4to7days	No	Discharge	No	No comorbids							