CLINICO-EPIDEMIOLOGICAL PROFILE AND OUTCOME OF PATIENTS WITH ACUTE POISONING PATIENTS PRESENTING TO EMERGENCY OF A TERTIARY CARE CENTRE IN WESTERN RAJASTHAN

THESIS

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DECLARATION

I hereby declare that the thesis titled **"CLINICO-EPIDEMIOLOGICAL PROFILE AND OUTCOME OF PATIENTS WITH ACUTE POISONING PATIENTS PRESENTING TO EMERGENCY OF A TERTIARY CARE CENTRE IN WESTERN RAJASTHAN"** embodies the original work carried out by the undersigned in All India Institute of Medical Sciences, Jodhpur.

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This is to certify that the thesis titled **"CLINICO-EPIDEMIOLOGICAL PROFILE AND OUTCOME OF PATIENTS WITH ACUTE POISONING PATIENTS PRESENTING TO EMERGENCY OF A TERTIARY CARE CENTRE IN WESTERN RAJASTHAN"** is the bonafide work of **Dr. Akhil V George** carried out under our guidance and supervision in the Department of Emergency Medicine, All India Institute of Medical Sciences, Jodhpur.

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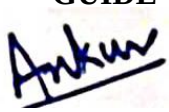

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Dedicated to my family

Acknowledgement

"*Alone we can do so little; together we can do so much*"

-Helen Keller

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Dr. Akhil V George

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LIST OF ABBREVIATION

WHO	**WORLD HEALTH ORGANIZATION**
PCP	**PHENYL CYCLOHEXYL PIPERIDINE**
LSD	**LYSERGIC ACID DIETHYLAMIDE**
CNS	**CENTRAL NERVOUS SYSTEM**
SSRI	**SELECTIVE SEROTONIN REUPTAKE INHIBITORS**
ECG	**ELECTROCARDIOGRAM**
BUN	**BLOOD UREA NITROGEN**
PaCO2	**PARTIAL PRESSURE OF CARBON DIOXIDE**
OPIDN	**ORGANOPHOSPHATE INDUCED DELAYED NEUROPATHY**
SMFA	**SODIUM MONOFLUROACETATE**
NAPQI	**N-ACETYL-P-BENZOQUINONE IMINE**
AST	**ASPARTATE AMINOTRANSFERASE**
ALT	**ALANINE TRANSAMINASE**
PT	**PROTHROMBIN TIME**
INR	**INTERNATIONAL NORMALIZED RATIO**
ICU	**INTENSIVE CARE UNIT**
MDMA	**3,4- METHYLENE DIOXYMETHAMPHETAMINE**
ED	**EMERGENCY DEPARTMENT**
GCS	**GLASGOW COMA SCALE**

IV	**INTRAVENOUS**
AChE	**ACETYLCHOLINESTERASE**
RBC	**RED BLOOD CELL**
PAM	**PRALIDOXIME**
AC	**ACTIVATED CHARCOAL**
PNU	**PROTEIN NITROGEN UNIT**
NAC	**N-ACETYLCYSTEINE**
pH	**POTENTIAL OF HYDROGEN**
CT	**COMPUTED TOMOGRAPHY**
ARDS	**ACUTE RESPIRATORY DISTRESS SYNDROME**
CYP2E1	**CYTOCHROME P450 2E1**
NAPQ1	**N-ACETYL-P-BENZOQUINONE**
BZDs	**BENZODIAZEPINES**
DMPS	**2,3 DIMERCAPTOPROPANE-1-SULFONATE**
DMSA	**DIMERCAPTOSUCCINIC ACID**
qSOFA SCORE	**QUICK SEQUENTIAL ORGAN FAILURE ASSESSMENT**
SD	**STANDARD DEVIATION**
TCAs	**TRICYCLIC ANTIDEPRESSANTS**
ALF	**ACUTE LIVER FAILURE**
SPSS IBM	**STATISTICAL PACKAGE FOR SOCIAL SCIENCES**
IQR	**INTERQUARTILE RANGE**

HTN	**HYPERTENSION**
DM	**DIABETES MELLITUS**
TB	**TUBERCULOSIS**
IHD	**ISCHEMIC HEART DISEASE**
PPD	**PARA-PHENYLENEDIAMINE**
APACHE II	**ACUTE PHYSIOLOGY AND CHRONIC HEALTH EVALUATION II**
LMIC	**LOW MIDDLE-INCOME COUNTRIES**
RBS	**RANDOM BLOOD SUGAR**

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SUMMARY

Background: Acute poisoning is a life-threatening medical emergency that can result in substantial morbidity and mortality. The pattern of poisoning in western Rajasthan is little known. Therefore, it is essential to gather regional clinico-epidemiological data on poisoning. These statistics can be useful for planning the optimal use of available resources for poisoning prevention and treatment. The purpose of this study was to assess epidemiological indicators for acute poisonings and variables influencing the clinical course and outcome of patients admitted to a tertiary care hospital's emergency department in Western Rajasthan.

Material and Methods: This prospective observational cross-sectional study was conducted between 1 January 2021 and 30 June 2022 in the Emergency Department of a tertiary hospital. Patients of either gender or any age attending the emergency department with acute poisoning were included. These cases were analysed for their epidemiological profiles, poisoning characteristics, and various clinical variables influencing patient outcomes.

Results: Out of the 236 patients, 143 were male and 93 were female. The maximum number of patients were from 21-30 age group. Out of the total patients, 190 patients were aged more than 18 years. Most patients were graduates and postgraduates (88 out of 190 patients). One hundred twelve out of 190 patients were married (58.95%). Most of the patients belonged to urban areas. Comorbidities were present in 32 patients. Psychiatric illness was the most common comorbidity. Depression was the most common psychiatric illness, followed by insomnia. The intention of poisoning was suicidal in 185 patients (78.4%) and accidental in 51 patients (21.6%). The oral route was the most common route of ingestion seen in 234 patients (99.1%). Organophosphate was the most common poisoning seen in 55 patients (23.3%), followed by drug ingestion seen in 44 patients (18.6%). The most common symptom at the time of presentation was nausea and vomiting (37.1%), followed by altered sensorium (17.3%). In the present study, 3.4% of patients with poisoning had renal dysfunction, 3.3% had liver dysfunction, 3.1% had coagulopathy, 8.8% needed vasopressor support, and 17.7% required ICU admission. A total of 10 patients died within 28 days. Aluminium phosphide was associated with the highest mortality rate, followed by organophosphate poisoning.

Conclusion: The present study indicates that most poisoning cases involved young people, mainly males. Organophosphorus poisoning is one of the most common poisonings in western Rajasthan, followed by ingestion of drugs. Aluminium phosphide had the highest fatality rate, followed by organophosphorus poisoning.

INTRODUCTION

Accidental and intentional exposure-related acute poisoning is a serious medical emergency that can cause considerable morbidity and fatality (1). The term "poison" refers to a substance that, when ingested, inhaled, or comes into contact with it, causes harm to the body and poses a threat to life(1). Both toxins and venoms are biologically derived poisons, with the latter term being used to refer to an animal's bite or sting (2).

Nearly 800,000 people die by suicide each year, and about 20 times as many individuals try suicide, according to the WHO(3). According to the World Health Organization (WHO), 0.3 million people each year die from acute poisoning, with 200000 of those fatalities attributable to organophosphorus poisoning alone(4). Despite the significance of suicide as a medical and social issue, Indian policymakers pay little consideration(3).

Financial strain, psychological disease, societal norms like the patriarchal system, and other aggravating or predisposing variables associated with poisoning can be found in developing nations like India(2). Women's dependence on males for financial support in the patriarchal society raises the likelihood that they may commit suicide(2). The kind of poisoning is impacted by availability and other elements, including prior awareness of the toxicity through various outlets for information and communication (5). Different geographical areas of the nation have different poisoning trends(2). Young persons are more likely to poison themselves by suicide than older adults, which causes a high economic and physical cost to society(6). Notably, 15% of accidental poisoning-related deaths include children under 5, and many of these incidents go undetected, underreported, or misreported (7). At the national and state levels, specific measures have been made to restrict access to the availability of certain poisons. However, this had little effect and did not change the trend of suicides.

In tropical nations, suicide-related agricultural pesticide poisoning is widespread and associated with significant mortality. Antidepressants & benzodiazepines are widely used in cities and rarely cause fatalities. In Africa and the Pacific, chloroquine, an antimalarial, is frequently used for self-injury. Many nations utilise acetaminophen for self-poisoning. Globally, industrial and domestic chemicals are a common cause of fatalities and permanent disability. Although rare, suicide by plant toxins is frequent in some areas (8).

Depending on the toxins, acute toxicity can manifest in various ways(9). Depression of the central nervous system, miosis, hypothermia, hypotension, respiratory depression, dysrhythmias, and multisystem organ failure are some of the symptoms and

consequences of poisoning(9). Most paediatric poisonings include mildly toxic chemicals, but on rare occasions, some are highly toxic and need prompt medical attention to avoid severe injury or death(10). Patients who show signs of organ failure need to be admitted to the ICU for specialised treatment and organ support(11). High fatality rates can result from a wide range of factors, such as the toxin nature, the timing of presentation, and multi-organ failure (11). Early identification and prompt treatment in the ED and ICU are essential for the poisoned patient to prevent hospital morbidity and death(9).

There have only been a few studies done on the epidemiology and clinical features of poisoning in Rajasthan. The pattern of poisoning in the western part of Rajasthan is poorly understood, though. As a result, there is a need for the generation of regional clinico-epidemiological data on poisoning. These data will be helpful in planning the appropriate use of available resources for the prevention and treatment of poisoning cases. Therefore, this study aimed to evaluate epidemiological indicators for acute poisons and factors impacting the clinical course and outcome of patients who were brought to the emergency room of a tertiary care facility in Western Rajasthan. (11).

REVIEW OF LITERATURE

GENERAL APPROACH TO DRUG POISONING IN ADULTS

OVERVIEW OF APPROACH

Poisoning can present with wide variety of clinical findings and symptoms. Presentation is based on the chemical consumed, the timing of the ingestion, whatever initial prescription drugs the patient might be taking, their health status, and whether they consumed a single drug or multiple drugs. Pattern identification and acute stabilisation are the main goals of the preliminary therapy. Identifying the kind of poisoning needs a comprehensive physical examination and history. The objective of management is to deliver supportive care, avoid poison absorption, and, when needed, administer antidotes and improve excretion methods.

INITIAL EVALUATION AND TREATMENT

All patients should have a rapid initial screening test to determine what has to be done immediately to stabilise and prevent the patient from getting worse. One should examine the airway condition, vital parameters, mental condition, size of the pupils, skin temperature, and moisture. Capillary glucose measurement, ECG, continuous cardiac monitoring, and oxygen saturation are the initial diagnostic tests to be done. Intravenous (IV) access should be acquired in all situations involving significant ingestion. One should maintain in-line cervical immobilisation in individuals with possible concealed trauma. If there is uncertainty regarding the patient's capability to maintain their airway and prevent aspiration, evaluate the airway and perform tracheal intubation. In patients with altered sensorium, we should consider administering naloxone, dextrose and thiamine. In case of suspected opioid addiction and multiple drug poisonings, caution is required due to concern about withdrawal symptoms and symptoms that may be mistaken as symptoms from coingestants. A capillary glucose test should be carried out on individuals with altered consciousness levels. Dextrose should be administered immediately to hypoglycaemic patients. Make sure that one ultimately reveals the patient and keeps a watch for any indications of injury, drug usage (such as needle marks), infection, or swelling of the extremities. In comatose or encephalopathic individuals, measure the core body temperature. Then, if necessary, patient decontamination should be done. To screen for cardiotoxicity associated with the toxin, and

an ECG should be done. One should look for drugs, prescription bottles, or anything associated with drugs, and check clothes, wallets, and pocketbooks.

DIAGNOSIS OF POISONING

History

Even though history is usually the most dependable source of information for determining the aetiology of poisoning, it is frequently inaccurate when given by a patient following deliberate consumption(12). Direct drug effects or psychiatric disorders can make it difficult for the patient to give an accurate history(13). Emergency personnel, police, the patient's employer, relatives, friends, and primary care physicians should be contacted for information when the patient cannot provide a dependable history. Medical records and pharmacists should also be consulted. Since over-the-counter drugs, traditional medicines, and dietary supplements are sometimes not seen as medications by patients and may not be mentioned during the regular inquiry about "drugs," it is crucial to ask about their usage specifically. Finally, only slang or colloquial phrases may be used to describe drugs of abuse ("ecstasy" for MDMA or "bath salts" for synthetic cathinones)(13).

Physical examination

Drug withdrawal episodes, anticholinergic, sympathomimetic, or central hallucinogenic substances cause physiologic excitation (elevated temperature, blood pressure and pulse, respiration rate and depth, central nervous system stimulation).

Ethanol, additional hypnotic-sedative drugs, opiates, cholinergic drugs, sympatholytics, or toxic alcohols (methanol or ethylene glycol) causes depressed mental status, low blood pressure, lower pulse rate, lower respiratory rate and depth, and temperature.

A polydrug overdose or exposure to certain metabolic poisons (hypoglycemic agents, salicylates, or cyanide), membrane-active substances (volatile substances, antiarrhythmic medications, local anaesthetic substances), heavy metals (iron, arsenic, mercury, or lead), or substances with multiple mechanisms of action may result in mixed physiologic effects.

Toxidrome	Vital signs	Mental status	Pupils	Other findings	Examples
Anticholinergic	Hypertension, hyperthermia, tachycardia	Agitation, hallucination	Mydriasis	Dry flush skin, urinary retention	Tricyclic antidepressants, atropine, scopolamine, antihistamine
Cholinergic	Bradycardia, tachycardia, hypertension	Confusion, coma	Miosis	Salivation, lacrimation, urination, diarrhoea, vomiting	Organophosphate, physostigmine
Hallucinogen	Hyperthermia, tachycardia, hypertension	Hallucination, synaesthesia, agitation	Mydriasis	Nystagmus	PCP, LSD
Opioid	Hypothermia, bradycardia, hypotension, bradypnea	Coma, CNS depression	Miosis	Pulmonary edema, hyporeflexia	Opioids
Sedative hypnotic	Hypotension, hypothermia, bradycardia, and bradypnea.	Confusion, CNS depression, coma	Pulmonary edema Miosis	Hyporeflexia	Benzodiazepines, barbiturates, alcohols
Serotonin syndrome	Tachycardia, hyperthermia, hypertension, tachypnea	Confusion, agitation, coma	Mydriasis	Tremor, myoclonus, diaphoresis, hyperreflexia, trismus, rigidity	Miosis, SSRIs, meperidine, dextromethorphan
Sympathomimetic	Hyperthermia, tachycardia, tachypnea	Agitation, hyperalert, paranoia	Mydriasis	Diaphoresis, tremors, hyperreflexia, seizures	Cocaine, amphetamine, pseudoephedrine

Table 1 -: Showing toxidromes of individual poisons

Electrocardiography

Patients with symptoms or those exposed to unknown or possibly cardiotoxic drugs should undergo ECG(14). Cocaine, tricyclic antidepressants, and carbamazepine are medications that block sodium channels, lengthening the QRS interval. Numerous medications block potassium efflux (antipsychotics, sotalol), which causes prolongation of the QT interval. Toxin-induced prolongation of the QRS interval in tricyclic antidepressant overdose demands immediate action.

Radiographic studies

Radiological investigations be helpful in radiopaque toxins, "body packers" or "body stuffers", and to diagnose noncardiogenic pulmonary oedema and acute respiratory distress syndrome due to exposure to specific toxic agents.

Toxicology screens (drug testing)

When people who accidentally ingested toxin are asymptomatic or have clinical signs compatible with their medical background, toxicology screening is rarely required. However, screening for paracetamol and salicylates is strongly advised for individuals with an unclear history of intentional poisoning. Urine screenings can identify opioids, methadone or methamphetamine, benzodiazepines, cocaine metabolites, barbiturates, tetrahydrocannabinol, tricyclic antidepressants, and phencyclidine. Positive and negative screen tests do not definitively confirm or deny a poisoning, and additional testing may be necessary.

Other laboratory studies

Specific agents cause certain laboratory abnormalities, such as electrolyte imbalance, rhabdomyolysis, hepatotoxicity, or methemoglobinemia. Blood gas, co-oximetry, and serum lactate may be necessary for patients with acid-base, cardiovascular, neurological, or respiratory problems. The toxic origin should be ruled out in patients with an elevated serum osmolar gap, acid-base imbalance, or oxygen saturation gap. High anion gap metabolic acidosis may be the first sign of toxic consumption, in salicylates, ethylene glycol, and methanol poisoning. To identify other potential causes of high anion gap acidosis, serum measurements of creatinine, glucose, ketones, and lactate should also be done. Alcoholic

ketoacidosis, diabetic ketoacidosis, or isopropyl alcohol poisoning can all increase blood creatinine levels with normal BUN levels.

Management

The nature of poison, it's severity and the time between exposure and admission all play a part in treatment of poisoned patient.

The sooner decontamination is carried out after initial stabilisation, the more effectively it prevents toxin absorption. The preferred means of decontamination include administering activated charcoal and drenching local exposures with plenty of water or saline irrigation. Other gastrointestinal decontamination techniques may be necessary for specific situations, such as whole-bowel irrigation, dilution, endoscopy, surgery, and cathartics.

Unless the condition is easily reversible, airway protection with tracheal intubation should be done as quickly as possible for a patient with a poor mental status. In salicylate poisoning, mechanical ventilation should be avoided unless essential.

Initial treatment for hypotension should involve boluses of isotonic intravenous (IV) fluids, and vasopressors may be needed if it does not improve. The initial action for hypertension in agitated patients should be done with non-specific sedatives like benzodiazepines(15).

First-line treatment for ventricular tachycardias caused by intoxication with a substance that has sodium channel-blocking properties is sodium bicarbonate. Antiarrhythmic drugs IA (such as procainamide), IC, and III are not indicated and can be harmful since they may worsen cardiac conduction. Patients with drug-induced torsades de pointes and prolonged QT intervals on an ECG may benefit from overdrive pacing with isoproterenol or a temporary pacemaker. Patients with serious tachyarrhythmias or bradyarrhythmias who have taken digoxin should receive specific antigen-binding (Fab) fragment therapy (Digi bind). Bradyarrhythmia combined with hypotension should be handled similarly to other cases, using atropine and temporary pacing. However, the administration of calcium, high-dose insulin, glucagon, vasopressors, isoproterenol, or other therapy may eliminate the need for a temporary pacemaker in patients with calcium channel blockers or beta blocker overdose.

Benzodiazepines generally treat drug-related agitation and seizures. It is appropriate to use specialised medications (eg, physostigmine for the anticholinergic syndrome) to treat the agitation caused by certain toxidromes(16). Severe drug-induced hyperthermia (serotonin

syndrome, sympathomimetic overdose, or neuroleptic malignant syndrome) may require urgent treatment, including immersion in cold water(17).

DISPOSITION

Patients with mild toxicity can be observed in the ED until they are asymptomatic (four to six hours). Patients at risk or with moderate toxicity should be admitted to a suitable observation unit or intermediate-care floor for close monitoring and care. Prior to discharge, all patients with deliberate overdose must undergo psychiatric evaluation.

Any 1 of the eight clinical indicators indicated that a challenging hospital course should be handled in an intensive care unit.

- PaCO2 >45 mmHg
- Need for tracheal intubation
- Seizures
- Low GCS
- Non-sinus cardiac rhythm
- Second- or third-degree atrioventricular block
- Systolic blood pressure <80 mmHg
- QRS duration ≥0.12 seconds

CARBAMATE AND ORGANOPHOSPHATE POISONING

Carbamates and organophosphates are potent cholinesterase inhibitors that can result in severe cholinergic toxicity when ingested, inhaled, or applied topically.

For over 50 years, organophosphates have been utilised as pesticides worldwide. Because of toxicities, the usage of these substances has decreased over the past 10 to 20 years(18). The use of organophosphates and carbamates in healthcare includes the treatment of glaucoma, Alzheimer's disease, and myasthenia gravis, as well as the reversal of neuromuscular blockade (with drugs like edrophonium, echothiopate, pyridostigmine, tacrine, donepezil and neostigmine). Each year, approximately 3,000,000 people worldwide are exposed to carbamate or organophosphate agents, with up to 300,000 deaths(19). Carbamate (methomyl and aldicarb) and organophosphate (parathion, fenthion, malathion and diazinon) pesticides have been specifically associated with human poisoning.

MECHANISM OF ACTION

Acetylcholinesterase(AChE), the enzyme that breaks down acetylcholine, is bound by organophosphorus substances and becomes inactive(20). A conformational modification known as "ageing" occurs in the acetylcholinesterase-organophosphorus complex over time, rendering the enzyme permanently resistant to being reactivated by an antidotal oxime(21). Carbamates are transient cholinesterase inhibitors, in contrast to organophosphates, therefore spontaneously hydrolyse from the enzymatic site of cholinesterase in 48 hours.

CLINICAL FEATURES

Clinical symptoms of organophosphorus poisoning usually appear three hours after oral or respiratory exposures, although symptoms of dermal absorption-related toxicity may take up to 12 hours to appear.

The primary systems involved are neuromuscular junction, autonomic nervous system, and central nervous system (22). Bradycardia, lacrimation, miosis, salivation, bronchospasm, bronchorrhea, urinary incontinence, vomiting, and diarrhoea are the main clinical signs of acute cholinergic toxicity. Fasciculations, muscle weakness, and paralysis are nicotinic side effects. Nicotinic and muscarinic receptors may be responsible for lethargy, central respiratory depression, convulsions, and coma.

In cases of organophosphorus agent poisoning, cardiac irregularities including heart block and QTc prolongation may be seen.

In acute organophosphorus poisoning, mortality can occur due to respiratory failure and depression of the CNS respiratory centre. Ten to 40% of people show cranial nerve abnormalities, neck flexion weakness, proximal muscle weakness, diminished deep tendon reflexes, and respiratory insufficiency 24 to 96 hours following exposure to organophosphorus compounds(23). Specific organophosphorus compounds, such as chlorpyrifos, can cause delayed neuropathy (OPIDN), which generally manifests one to three weeks after consumption(24).

DIAGNOSIS

Organophosphorus substances have a distinctive aroma similar to that of petroleum or garlic, which may aid in making the correct diagnosis.

The severity of toxicity is directly measured by RBC acetylcholinesterase (RBC AChE) activity. The efficiency of oxime therapy can be assessed through sequential measurement of RBC AChE activity.

MANAGEMENT

Oxygen support and prompt endotracheal intubation are required for patients with altered mental status.

Atropine and oxime are used for carbamate or organophosphate poisoning. Atropine inhibits cholinergic activation by competing with acetylcholine at muscarinic receptors. 1 to 2 mg bolus, double dosage every 3 to 5 minutes. Atropine is ineffective in treating neuromuscular impairment, as it does not bind to nicotinic receptor sites. Both muscarinic and nicotinic symptoms can be successfully treated with pralidoxime (2-PAM) and other oximes (HI-6 and obidoxime)(25). To prevent oxime-induced inhibition of acetylcholinesterase, pralidoxime should be added with atropine(26). The current WHO recommendation for IV bolus treatment with pralidoxime is at least 30 mg/kg in adults and 25 to 50 mg/kg in children.

Benzodiazepines should be used to treat seizures induced by organophosphorus agents. When dermal absorption is possible due to topical exposure, the patient should be completely stripped down and the affected regions should undergo a thorough cleaning procedure that includes vigorous irrigation(27). When a patient presents less than an hour after ingesting an organophosphorus chemical, certain physicians may decide to do stomach lavage, which should only be done after endotracheal intubation and initiation of therapy with atropine and an oxime. It is advised to give activated charcoal (AC) (standard dose- 1 g/kg) to patients who arrive within an hour of ingesting an organophosphorus substance or carbamate(28).

RODENTICIDE POISONING

Aluminium Phosphide was identified as the most prevalent suicidal poison in a study of unnatural deaths through an autopsy in Northwest India, accounting for 68.4% of all poisoning deaths between 1992 and 2002.

The signs and symptoms of rodenticide poisoning are

I. Cardiac arrest, cardiac arrhythmias, or refractory shock-

- Early: White (yellow) phosphorus, barium carbonate, or zinc or aluminium phosphide

- Late: sodium monofluoroacetate (SMFA), thallium, arsenic, or fluoroacetamide

II. Seizures- SMFA, fluoroacetamide, tetramine, or arsenic

III. Muscle rigidity, opisthotonus, trismus, and facial grimacing (risus sardonicus) – Strychnine

IV. Coma, lethargy, or cranial neuropathy- Vacor, alpha-chloralose, bromethalin, thallium, arsenic, and (pyriminil, N-3-pyridylmethyl-N-p-nitrophenylurea, PNU)

V. Bleeding or bruising- Anticoagulants (warfarin compounds)

ZINC AND ALUMINUM PHOSPHIDE

Metallic phosphides are applied to preserve grains from rats and other pests during transportation and storage worldwide (30). The most basic way of exposure is the inhalation of phosphine gas, which is created when aluminium or zinc phosphide is exposed to moisture in stored grain. In northern India, aluminium phosphide exposures through intentional suicide consumption are frequent(30).

It can cause hypotension, acidosis, hypoxia, global left ventricular hypokinesis, which can be fatal(31). Gastric secretions transform phosphides into phosphine gas after consumption.

Hematemesis, coughing, nausea, emesis, and retrosternal chest and abdominal pain are the common presentations. The most frequent complications are cardiogenic shock, hemorrhagic pulmonary edema, and cardiac arrhythmias (including bradycardia, atrial fibrillation, atrial flutter, supraventricular tachycardia, and ventricular arrhythmias)(32).

100% oxygen and prompt endotracheal intubation are required for patients with respiratory compromise. Provide fluid resuscitation for hypovolemia and correction of hypoglycemia, hypokalemia and hypomagnesemia. Treat cardiogenic shock with vasoactive medications. If hypomagnesemia is recognised, trials suggest intravenous magnesium delivery can reduce mortality(33). There are several ways to stop the absorption of phosphine, such as lavage with coconut oil to stop the generation of phosphine gas, oral or gastric tube delivery of sodium bicarbonate, lavage with potassium permanganate, or utilisation of activated charcoal(32). Case studies discuss the application of N-acetylcysteine (NAC) as an antioxidant(34).

ACUTE HYDROCARBON EXPOSURE

Over 28,000 cases of poisoning from exposure to hydrocarbons are reported to regional poison control centres each year in the United States, and they are a significant cause of poisoning globally(35).

The method, type of chemical, and amount of exposure all affect the severity of hydrocarbon poisoning(36).

Major effects of consuming hydrocarbons include pulmonary aspiration and chemical pneumonitis. When inhaled or ingested, highly lipid-soluble hydrocarbons quickly diffuse all around the body and into the central nervous system. Hydrocarbons make catecholamines more sensitive to the heart, which can result in arrhythmias.

Other harmful consequences of acute hydrocarbon exposure include hemolysis, hepatotoxicity, gastrointestinal discomfort, and skin and mucosal irritation(36),(37),(38).

EVALUATION

The physician should identify the dose and exposure route. When a history of exposure or ingestion is uncertain, distinctive smells can occasionally be used to detect the existence of hydrocarbon exposure(39).

Soon after consuming a hydrocarbon, fever commonly appears and may develop into tachypnea, tachycardia, and hypoxemia. Although their onset may be delayed for 12 to 24 hours, pulmonary signs owing to aspiration often emerge within 30 minutes of the aspiration(39). Clinical signs of pulmonary toxicity that may appear over the first 24 to 48 hours include persistent hypoxemia, necrotising chemical pneumonitis, lipoid pneumonia, pneumothorax, subcutaneous emphysema , pleural effusion, and hemorrhagic pulmonary oedema that can quickly progress to shock and respiratory arrest(40). Direct effects on the central nervous system (CNS) from ingesting or inhaling hydrocarbons include somnolence, headache, ataxia, disorientation, fuzziness, lethargy, exhaustion, stupor, seizures, and coma. Ingestion of hydrocarbons results in mucosal ulceration and contact irritation of the pharynx, oesophagus, stomach, and small intestine(36). The diagnosis of chemical pneumonitis brought on by hydrocarbon aspiration is made by chest radiography. Initial radiographic signs include several tiny, patchy densities with ill-defined borders.

MANAGEMENT

Emergency stabilisation of the airway, breathing, and circulation may be necessary for patients who have consumed substantial amounts, had asphyxial inhalation or were exposed to aromatic or halogenated hydrocarbons.

Endotracheal intubation should be done for patients with severe respiratory distress, or who have considerable lethargy that prevents them from maintaining their airways. Continuous cardiac monitoring and a 12-lead electrocardiogram should be done for ventricular arrhythmias. Benzodiazepines should be administered to patients having seizures. The patient's contaminated clothes should be removed to stop further exposure. Due to the possibility of provoking vomiting and subsequent pulmonary aspiration, patients with an isolated aliphatic or terpene hydrocarbon intake should not get activated charcoal (36).

CAUSTIC INGESTION

With an estimated 5,000–15,000 caustic ingestions per year in the US, the caustic injury still represents a significant public health issue(41).

Alkali ingestion cause liquefactive necrosis, penetrating transmural injury may result in mediastinitis, oesophagal perforation, and death(42).

Usually, acid consumption causes coagulation necrosis that thromboses the connective tissue and blood vessels underneath the mucosa, generating a protective eschar(43).

Factors affecting severity,

- Corrosive characteristics (pH) of the drug consumed.
- Concentration and quantity consumed.
- Physical state (solid or liquid)
- Duration of time in contact with the mucosa

The frequent complaints include oropharyngeal, retrosternal, or epigastric discomfort, dysphagia/odynophagia, hypersalivation, vomiting, and hematemesis. High-grade injury patients are more likely to develop complications such as haemorrhage, fistulisation, strictures, and oesophagal squamous cell carcinoma.

In order to fully understand the degree of necrosis in all patients, a computed tomography (CT) scan of the chest and abdomen should be done to diagnose the severity of necrosis(44).

MANAGEMENT

Patients who, have oral burns, or are known to have consumed a drug with a greater risk of esophageal damage should be admitted to a medical facility for further testing and supportive treatment. It includes pain management, fluid therapy, and respiratory support. Nasogastric intubation should be avoided as this may provoke retching and vomiting, potentially resulting in the stomach or oesophagal peforation. Broad-spectrum antibiotics for individuals with suspected perforation and proton pump inhibitors to avoid stress ulcers should be prescribed(44). Corticosteroids, neutralising agents, and emetics have no benefit(45).

Emergency surgery is indicated if there are any clinical symptoms of perforation (such as mediastinitis or peritonitis) or if there is evidence of transmural oesophagal or gastric necrosis(46).

In individuals who have consumed caustic, upper endoscopy may predict the likelihood of stricture development. To assess the severity of the damage, upper gastrointestinal endoscopy should be done the first 24 hours after intake(47). Patients with gastrointestinal perforation should not undergo an upper endoscopy. Before the upper endoscopy, the patient should be intubated to preserve the airway if there is respiratory distress, oropharyngeal or glottic oedema, and necrosis.

Endoscopy grade 1 or 2A or CT grade 1 patient need supportive treatment and pain management. High-grade injury patients need to be monitored for at least one week to look for clinical signs of perforation. Most clinicians try dilatation three to six weeks after the damage to reduce the danger of esophageal perforation (42). Patients who have undergone several unsuccessful endoscopic dilatations have to be assessed for surgical reconstruction (48).

ALCOHOL INTOXICATION IN ADULTS

Acute ethanol intoxication can cause a variety of signs and symptoms, such as slurred speech, nystagmus, incoordination, unsteady gait, memory impairment, stupor, or coma. Ethanol-induced peripheral vasodilation, hypotension and tachycardia may occur(49).

The most precise way to determine a patient's ethanol content is through serum measurements. Alternative techniques, such as breath analysis, give findings more quickly but frequently produce ethanol amounts that are lower than those found in venous blood(50).

The mainstay of treatment is supportive care for isolated acute ethanol intoxication. Rapid bedside glucose testing should be performed on all drunk patients. With monitoring of the

airway and breathing, vigorous supportive treatment must be given to all patients with severe ethanol intoxication.

ACUTE OPIOID INTOXICATION

Opioid misuse is a global issue, and overdose deaths are frequent and on the rise(51). Identification of the precise medication, dosage, formulation, co-exposures and past opioid usage should be searches in patients with suspected opioid intoxication.
Signs of opioid poisoning altered mental status, decreased rate of breathing (most notable vital sign), lower tidal volume, reduction in bowel movements, pinpoint pupils.

Opioid toxicity is not entirely excluded by a routine pupil examination (52). Buprenorphine can cause withdrawal symptoms. Dextromethorphan causes pulmonary and CNS symptoms as well as miosis. Acute amnestic syndrome and chest wall stiffness associated with fentanyl(53). Meperidine causes serotonin toxicity and seizures.

In opioid poisoning there should be a focus on supporting the patient's airway and breathing. The best way to administer naloxone is intravenously (it can be given intramuscularly, subcutaneously, or nasally), with higher first doses for apneic individuals.

MISCELLANEOUS

CANNABIS INTOXICATION

It is the most frequently grown, transported, and misused illegal drug in the world(54). Children are more likely to experience neurological abnormalities such as ataxia, hyperkinesis, seizures, lethargy, and extended coma that may be life-threatening(55). Tachycardia, hypertension or hypotension, tachypnea, red eyes, dry mouth, increased hunger, nystagmus, ataxia, and slurred speech are physiological symptoms of cannabis intoxication in adults(56).

Management is supportive, emphasising airway, breathing, circulation, and hypoglycemia. Benzodiazepines are frequently helpful if symptoms of severe anxiety or agitation appear.

MDMA (ECSTASY) INTOXICATION

MDMA causes emotions of heightened physical and mental abilities, ecstasy, and greater alertness while decreasing tiredness. Poisoning can result in arrhythmia, myocardial infarction, hypertensive crises, and aortic dissection. Increased fluid consumption and ongoing antidiuretic hormone release lead to hypervolemic hyponatremia. In addition to CNS symptoms, including agitation, hyperactivity, anxiety, and even delirium, serotonin syndrome is a potentially fatal illness marked by the triad of autonomic dysfunction, aberrant neuromuscular activity, and altered mental status(57). Investigations include measurement of blood sugar, levels of aspirin and salicylates, electrocardiogram (ECG), serum electrolytes, creatine kinase, and urine myoglobin, creatinine, and aminotransferase, coagulation studies.

Altered sensorium caused by MDMA-associated hyponatremia usually requires endotracheal intubation(57). In order to treat the central component, benzodiazepines are used. A single dosage of activated charcoal at 1 g/kg can be used if patient is presenting in an hour. Benzodiazepines are the therapy of choice for serotonin syndrome, either combined with cyproheptadine or not.

CAMPHOR POISONING

Annually, 11,000 paediatric camphor exposures are reported to poison control centres in the United States. The presentation will include a potent medicinal odour, oropharyngeal irritation, nausea, emesis, and stomach pain. Seizures might be the first indication of exposure, with agitation, confusion, myoclonus, hyperreflexia, lethargy, or coma(58).

PARACETAMOL OVERDOSE

In the United States and many other nations, paracetamol is still a leading contributor to overdoses, liver failure, and fatal overdoses(59).

Risk factors for liver damage from acetaminophen consumption are, excessive acetaminophen consumption (most important), delaying the use of N-acetylcysteine (NAC) treatment after taking acetaminophen, unusually high cytochrome P450 activity, reduced ability to glucuronidate or sulphate, decrease in glutathione levels

Clinical Manifestations

Four sequential stages are used to describe the clinical course of poisoning.

- Stage I (30mins to 24 hours)- Patients frequently experience nausea, vomiting, diaphoresis, pallor, lethargy, and malaise within the first 24 hours after an overdose. Laboratory tests are frequently normal, and some individuals continue to be asymptomatic.
- Stage II (24 to 72 hours)- Stage I symptoms initially disappear, and patients seem to get better clinically, but with subclinical elevations of liver aminotransferases.
- Stage III (72 to 96 hours)- In addition to jaundice, hepatic encephalopathy, a significant rise in liver enzymes, hyperammonemia, and a bleeding diathesis, can occur. Plasma ALT and AST levels frequently surpass 10,000 IU/L, a prolonged PT/INR, lactic acidosis, hypoglycemia, and a total bilirubin concentration exceeding 4.0 mg/dL or 68 micromol/L are all indicators of severe hepatotoxicity.
- Stage IV (four days to two weeks)- Patients that make it through stage III begin their recovery, which typically starts on day four and lasts for seven days.

The frequency of renal impairment is correlated with the amount of acetaminophen was consumed. Measurements of serum paracetamol concentration should be taken four hours following the alleged time of intake. To decide whether N-acetylcysteine (NAC) treatment is necessary, the concentration must be assessed using the modified Rumack-Matthew nomogram. If sustained-release acetaminophen is used, the four- and eight-hour blood paracetamol concentrations should be tested. If either value is over the nomogram's line, therapy with NAC should be started(60).

Patients who consumed more than 7.5 to 10 g of acetaminophen exhibit the following symptoms like (61), nausea, vomiting, jaundice, sick appearance, and liver or abdominal discomfort, supratherapeutic serum acetaminophen levels, elevated ALT or AST levels upon presentation (>50 U/L). All patients with clinical indicators of toxicity (such as liver tenderness), elevated aminotransferases (ALT or AST 50 U/L), supratherapeutic serum acetaminophen concentrations are advised to receive treatment with NAC.

The 20-hour IV dosage regimen that has been approved is done as follows:

- 150 mg/kg IV of a first loading dose should be given over 15 to 60 minutes.
- Then administer 50 mg/kg over 4 hours and dose of 100 mg/kg over 16 hours to finish.

About 33% of patients using oral N-acetylcysteine experience nausea and vomiting. When the serum acetaminophen concentration is undetectable, the ALT is declining or within the normal range, and the INR is less than two, treatment may be discontinued.

BENZODIAZEPINE POISONING

Sedation, ataxia, and respiratory depression are the clinical signs of overdose. The patient's airway, breathing, and circulation are immediately evaluated and managed. Giving activated charcoal has no additional advantage but can make managing the patient's airway more difficult and enhance the risk of aspiration if they become drowsier. There are no efficient methods to increase BZD clearance. Dosage of 0.2 mg IV flumazenil can be given as antidote of benzodiazepines.

MERCURY TOXICITY

Elemental, inorganic, and organic forms of mercury all have the potential to be harmful(62). Exposure sources include dentistry, gold mining, thermometers, barometers, and fluorescent light bulbs.

Coughing, dyspnea, chest discomfort, stomatitis, conjunctivitis, dermatitis, nausea, vomiting, severe diarrhoea, gum inflammation, and excessive salivation are signs of acute mercury intoxication.

Tremors, sleeplessness, personality changes, anxiety, irritability, phobias, memory loss, , weakness, and sleepiness are all symptoms of chronic exposure of mercury toxicity(63). Small infants exposed to elemental mercury, inorganic salts, and organic phenylmercury compounds develop acrodynia. Subtle changes in kidney tubular function have been linked to occupational exposure to elemental mercury vapour and inorganic mercury. Measuring mercury in the blood and 24-hour urine confirms the diagnosis. Symptomatic mercury toxicity with elevated urine mercury levels has shown improvement after chelation therapy, including dimercaprol, penicillamine, unithiol (2,3 dimercaptopropane-1-sulfonate[DMPS] (63).

LITERATURE ON CLINICAL-EPIDEMIOLOGICAL PROFILES AND OUTCOMES OF PATIENTS WITH ACUTE POISONING PATIENTS

Shah et al. (2013-2014) conducted a study by recruiting 340 cases (216 males) of acute poisoning, with 38.82% belonging to 21-30 years. Out of these cases 62.06% of the cases being accidental poisoning. Poisonous bite (25.88%), followed by organophosphate

(19.41%), was the most common cause. Vomiting, local bite-related symptoms, impaired sensorium, giddiness, and breathing problems were most commonly noted, with an average hospital stay of 5.39 days. In 66.47% of these cases, patients had a full recovery, while 16.47% had fatal outcomes.

Patel et al. (2011- 2013) evaluated 135 patients with acute poisoning. The 11–20 age range was the most prevalent age group with poisoning. Seventy seven percent were suicidal, and most of them took Aluminium Phosphide (31.6%) and Organophosphates (20.4%)(3).

Ahmed et al. (2019-2020) studied acute poisoning in 223 children aged between 2-5 years. The most common cause of unintentional poisoning was kerosene (33%), followed by insecticides/pesticides (26.5%), medications (17%), and home chemicals (17%)(7).

Rajbanshi et al. (2016-2018) studied 85 patients with acute poisoning, most of them caused by suicide attempts. Organophosphorus compounds (43.5%) were responsible for most of the poisoning and required a prolonged stay in the ICU. Age, length of hospital stays, GCS at admission, SOFA score, the requirement for mechanical ventilation, length of ICU admission, coagulopathy, liver failure, and need for vasoactive drugs all impacted the patients' survival rates. (4).

Qiang Dai et al. researched acute poisoning in children that established its high frequency in children, especially in early childhood (34.6%) and preschool (37.3%), more frequently in rural regions (67.9%). The prevalence was higher in boys (53.2%). Drugs (47.2%), chemicals (10.5%), and alcohol (9.6%) were the leading causes of poisoning in urban areas. Pharmaceuticals (17.9%) and pesticides (52.1%) were the primary causes in rural regions. Pesticides (37.5%) and drugs (27.3%) predominated, while the majority of incidents of poisoning (92.3%) were unintentional(64).

Lee et al. (2011-2015) took 5-year poisoning records of 590 children (52.3% boys). The mean age group was 5.07 years. Pesticides were the most often consumed non-pharmaceutical toxin (9.5%), whereas pharmaceutical ingestion (41.4%) was the primary aetiology of poisoning. The average length of time spent in the ED was 5.45 hours. One hundred one patients (17.2%) were admitted to the hospital, 21 of which (3.6%) required acute care(10).

Banerjee et al. (2008-2010) studied 4,432 individuals with a history of poisoning. There were more female patients than male ones. Suicide-related poisoning occurred more frequently (81.08%) than accidental (18.92%). The average time to present an emergency after poison

intake was 6.4 hours. A frequent cause of poisoning was a snakebite (31.9%), followed by organophosphorus (21.84%), rat poison (16.49%), alcohol (13.80%), chemicals (9.04%), and medicines (2.3%). At presentation, the poisoned individuals had a mean GCS score of 6.85 ± 1.62. Three thousand seven hundred twelve patients (83.76%) trial participants survived(65).

Bjornaas et al. (2003- 2004) during their study period found the death of 103 individuals above the age of 16 from acute poisoning. The mean age was 44, with a 2:1 male-to-female ratio. The death occurred outside the hospital in 92 patients (89%). Opioids have been the main hazardous agents (65%), followed by alcohol (9%), TCAs (4%), benzodiazepines (4%), and zopiclone (4%). Seventy one deaths (69%) were classified as unintentional deaths, while 32 (31%) were suicidal. (66).

Mittal et al. 2010-2020) performed a meta-analysis of poisoning studies in which miscellaneous agents were the primary cause of poisoning in children, with an incidence of 45.0%, whereas pesticides were the primary cause of adult toxicity, with an incidence of 63%. Miscellaneous agents (18%) were the second most frequent cause of poisoning, followed by medicines (10%), venoms (6%) and corrosives (2%)(2).

Hock Heck Tan et al. performed a thorough analysis containing case reports, case series, and abstracts from toxicology meetings that were found by searching MEDLINE (1950 – 2007), EMBASE (1974-2007), and other databases. to identify the cardiovascular consequences observed following an overdose of aripiprazole, olanzapine, quetiapine, risperidone, and ziprasidone. There were reports of prolonged QTc intervals and hypotension among 22 adolescents (7–16 years) and 185 adult patients. However, there were only three occurrences of ventricular dysrhythmia and three fatalities from direct cardiovascular damage(67).

Larson et al. (1998-2003) conducted research in which 275 (42%) of the 662 individuals who met the criteria for ALF throughout six years were found to have acetaminophen-related liver injury. The median amount consumed was 24 g. One hundred and thirty-one (48%) incidents of overdoses were unintentional, 122 (44%) were suicidal. Overall, 178 individuals (65%) lived, 74 (27%) passed away before receiving a liver transplant, and 23 (8%) received a liver transplant(68).

Andersson et al. (2013- 2015) studied acute benzodiazepine toxicity. Seven hundred and eighty-seven (76%) of the 1037 patients were male, with a median age of 36. With 575 cases (55%), clonazepam was the most prevalent drug, followed by diazepam (15%), alprazolam

(12%), and oxazepam (9%). Only 25 (2%) and 11 (1%) cases of zolpidem and zopiclone, respectively. In 936 (90%) cases, benzodiazepines were taken with other intoxicants, most often heroin 484 (47%), ethanol 321(31%) and amphetamine 199(19%)(69).

Bagherian et al. performed a metanalysis, with 21 studies comprising 3432 patients of aluminium phosphide poisoning. Male and female patients had mortality rates of 62.3% (95%) and 37.7% (95%), respectively. The survivors' average age was noticeably younger than that of non-survivors(70).

David C et al. (1987-1993) conducted a study on 49 patients who had liver toxicity from acetaminophen (aspartate aminotransferase >1000 U/L). Twenty-one patients (43%) consumed acetaminophen for medical purposes. All hepatotoxic patients consumed more than 4 g/day(71).

Gorp et al. (2009) conducted a study on escitalopram overdose. The median dose of ingestion was 140mg. Only 1 out of 33 individuals who also took other drugs experienced serotonin toxicity, compared to 7 out of 46 people who consumed escitalopram alone (or 15%). Hyperreflexia and inducible clonus were common characteristics. Escitalopram overdoses alone were less likely to result in CNS depression and ICU hospitalisation than overdoses, including sedative co-ingestants. Bradycardia did occur in 11 cases (14%) and QT prolongation in 11 (14%)(72).

Hashmi et al. studied clinico-epidemiological features of corrosive ingestion in 206 patients. They were aged between 2 and 42 years (mean years-23.44 ±7.19), with 135 female patients (65.5%) and 71 male patients (34.5%). Rural populations have a somewhat strong tendency for corrosive intake. One hundred ten patients were unmarried (53.4%), compared to 90 married (43.7%). In patients who were illiterate or less educated and belonged to socioeconomically weaker groups, the prevalence of corrosive intake was much higher. Ninety-five % of the 197 patients who consumed corrosive chemicals did so on purpose to harm themselves. The two most often utilised corrosive substances were laundry bleaches and bathroom cleansers. The hospital stays ranged from one day to 60 days. The most often affected organs by corrosive damage were the oesophagus and oropharynx(73).

Razwiedani et al. studied 207 organophosphate cases, ranging in age between 10 months and 59 years. Most cases were men (58.9%) and suicidal (51%). The whole group's mortality rate was 3.4 %.

Tenenbaum et al. (1992-2012) studied 172 children with hydrocarbon ingestion. The hospitalisation was more common among boys than among girls. At presentation, fever and dyspnea were the most frequent symptoms, followed by vomiting, altered sensorium, and coughing. Typically, the clinical course was mild to moderate and resolved independently. Some children received oxygen, fluids, and antibiotics. Chemical pneumonitis was the primary symptom of hydrocarbon poisoning, and many patients showed abnormal chest x-rays. Only one case led to a fatal outcome(75).

Draanen et al. (2000-2021) did a systematic review of 38 records selected from screening 6512 cases. In this study, 37 of the 38 studies found an association between opioid overdose and mental illness. Evidence also supports the notion that mental illness and overdose are related(76).

Jody L. Green et al. did a systematic review and meta-analysis on oral vs intravenous acetylcysteine for acetaminophen toxicity therapy by screening 4,416 abstracts; 5,164 patients are mentioned in 16 articles. Hepatotoxicity rates were 12.6% and 13.2% for oral and intravenous methods, respectively. Hepatotoxicity rates are increased when treatment is delayed(77).

Manzar et al. researched the aetiology and demographics of children's acute household accidental poisoning. Kerosene (50%) was the most common household agent in our sample, followed by medications (38%), insecticides (7%) and toilet cleansers (5%), with the ratio of men to women 1.2:1. 70% of the kids came within three hours of consuming. The most frequent symptom noted was dyspnea(78).

METHODOLOGY

AIM

To study the clinico-epidemiological profile of patients with acute poisoning presented to the emergency department of a tertiary care centre in western Rajasthan.

OBJECTIVES

- **PRIMARY-** To assess the clinico-epidemological profile of patients with acute poisoning presenting to the emergency department of a tertiary care centre in western Rajasthan.
- **SECONDARY -** Outcome of patients with acute poisoning presenting the emergency department of a tertiary care centre in western Rajasthan.

STUDY SETTING

The study was conducted in the Department of Emergency Medicine, AIIMS Jodhpur, after getting approval from Institutional Ethical Committee (AIIMS/IEC/2021/3369).

STUDY DESIGN - Prospective cross-sectional study

STUDY PARTICIPANTS

- **INCLUSION CRITERIA**

  Patients of either sex belonging to any age group with acute poisoning attended the emergency department from 1 Jan 2021 to 30 June 2022.

- **EXCLUSION CRITERIA**
  - Patients with idiosyncratic or adverse reactions to prescribed drug
  - Food poisoning
  - Snake /unknown bites
  - Patients or relatives refused to participate in the study

DATA COLLECTION

- Patients attended the emergency department exposed to poisons were initially evaluated and resuscitated to maintain their airway, breathing, and circulation.
- Informed consent was obtained from eligible patients /legally authorised representatives (for unconscious patients)
- Data regarding the age, sex, residence, marital status, literacy, comorbidities, psychiatric illness, poisonous agent, intention and route of exposure were collected according to the history given by the patient or their relatives.
- The nature of the poison involved was determined from the circumstantial evidence, reliable history, remaining stuff/container from which poison had been consumed and suggested clinical features.
- A complete history, general examination and systemic examination were carried out in each case.
- Glasgow Coma Scale (GCS) and quick Sequential Organ Failure Assessment (qSOFA) score were calculated at the time of admission.
- The smell from mouth/vomitus/clothes was also assessed.
- The following investigations were carried out in all patients;
    - Stomach wash sample (except corrosive poisoning or presenting late after ingestion)
    - Random blood sugar
    - Blood gas analysis
    - Renal function tests with serum electrolytes
    - Liver function tests
    - Electrocardiogram
    - Complete hemogram
    - Radiographic studies were done in specific conditions
- Treatment was explicitly given to the cases, managed with antidotes if indicated
- If necessary, patient decontamination was carried out after initial patient stabilisation.
- After an initial assessment, care, and a brief observation period, the patient's disposition was determined by the level of toxicity that had been observed and was anticipated to occur.
- Need for vasopressor, acute renal failure, hepatic failure, coagulopathy, length of hospital stay, need of ICU admission and mechanical ventilation were also assessed.

- Discharge/ admission to ward/ICU was also noted.
- Patients were followed up till discharge from the hospital or death.

- **SAMPLING AND SAMPLE SIZE**
    - The study was time-bound. The duration of the study was 18 months, from 1 January 2021 to 30 June 2022.

- **STATISTICAL ANALYSIS**
    - Data was entered and analysed using SPSS IBM software version 22 (IBM SPSS Advanced Statistics, Chicago, IL, USA)
    - Nominal data was described using frequency and percentages and compared using the chi-square or Fischer Exact test.
    - Ordinal Data was described using the median and interquartile Range (IQR) and compared using the Mann-Whitney U test.
    - Continuous data was described using mean ± SD and compared using an unpaired t-test.
    - A p-value of <0.05 was considered statistically significant
    - The association between two variables (ordinal or continuous) was measured by the Spearman rank-order correlation coefficient.

FLOW DIAGRAM OF PARTICIPANTS IN THE STUDY

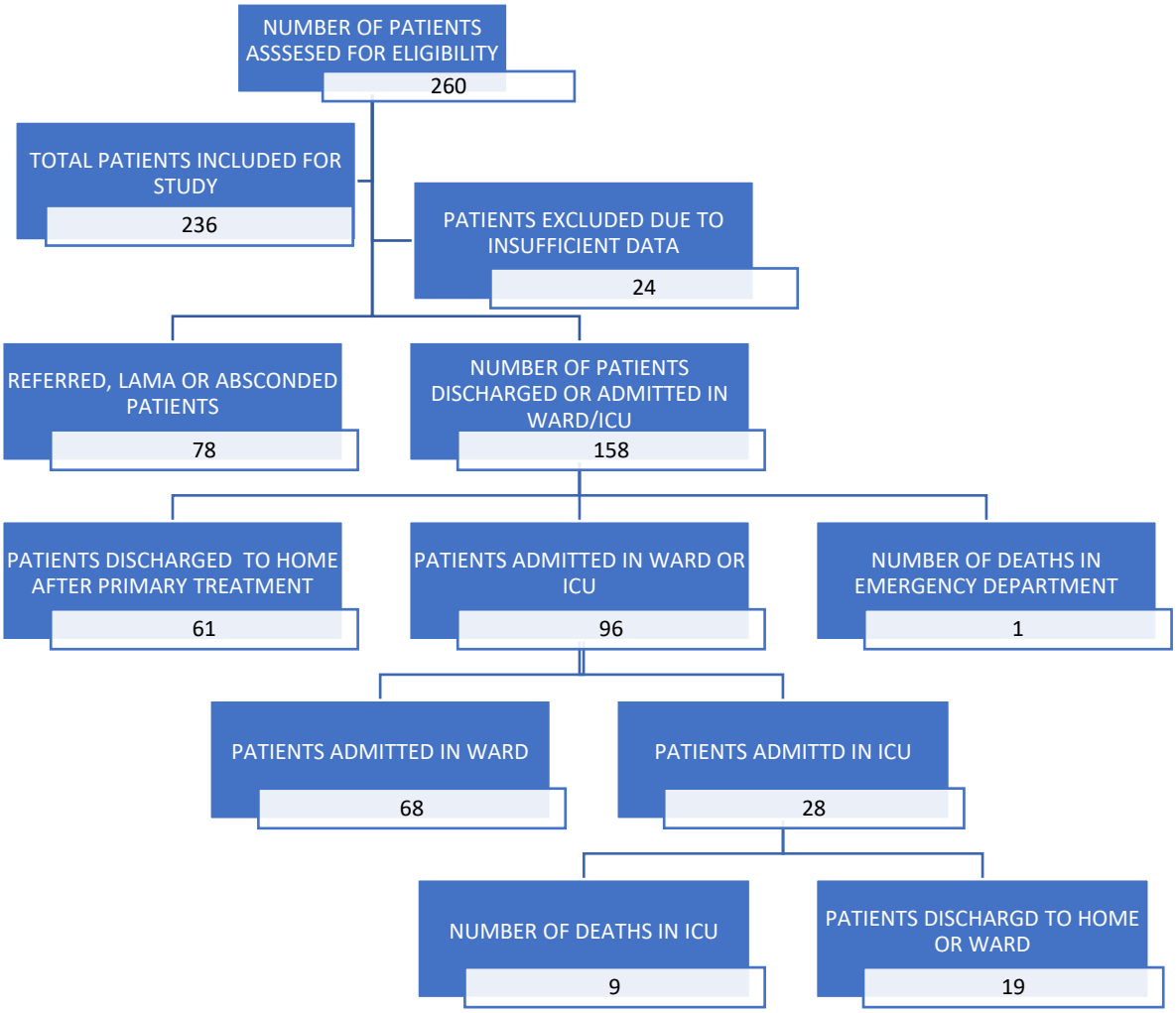


Figure 1-: Flow diagram of participants in the study

RESULTS

Population characteristics

The study was conducted in a tertiary centre in western Rajasthan. A total of 260 patients with acute poisoning presented to emergency department during the study period. Twenty-four patients were excluded from the study due to insufficient data. Subsequently 236 patients were enrolled in the study.

Demographic and clinical characteristics of patients

During study period from Jan 1 2021 to June 30 2022 total of 48218 patients attended emergency department, out of which, in 260 cases poisoning was suspected. Therefore, the prevalence rate of poisoning in the study was 0.53%.

The mean age of patients was 27.8 ± 16.1 years. The proportion of male patients was 60.5%, and the proportion of female patients is 39.5%, as shown in table 2.

Demographic details	
Age- years (Mean ± SD)	27.8 ± 16.1
Gender	
Male [n (%)]	143 (60.5%)
Female [n (%)]	93 (39.5%)

Table 2: Demographic and clinical characteristics among the study population

AGE DISTRIBUTION

Out of the 236 patients, maximum number of patients were from 21-30 age group (n-88), and only 3 patients were from a group of 71-80.

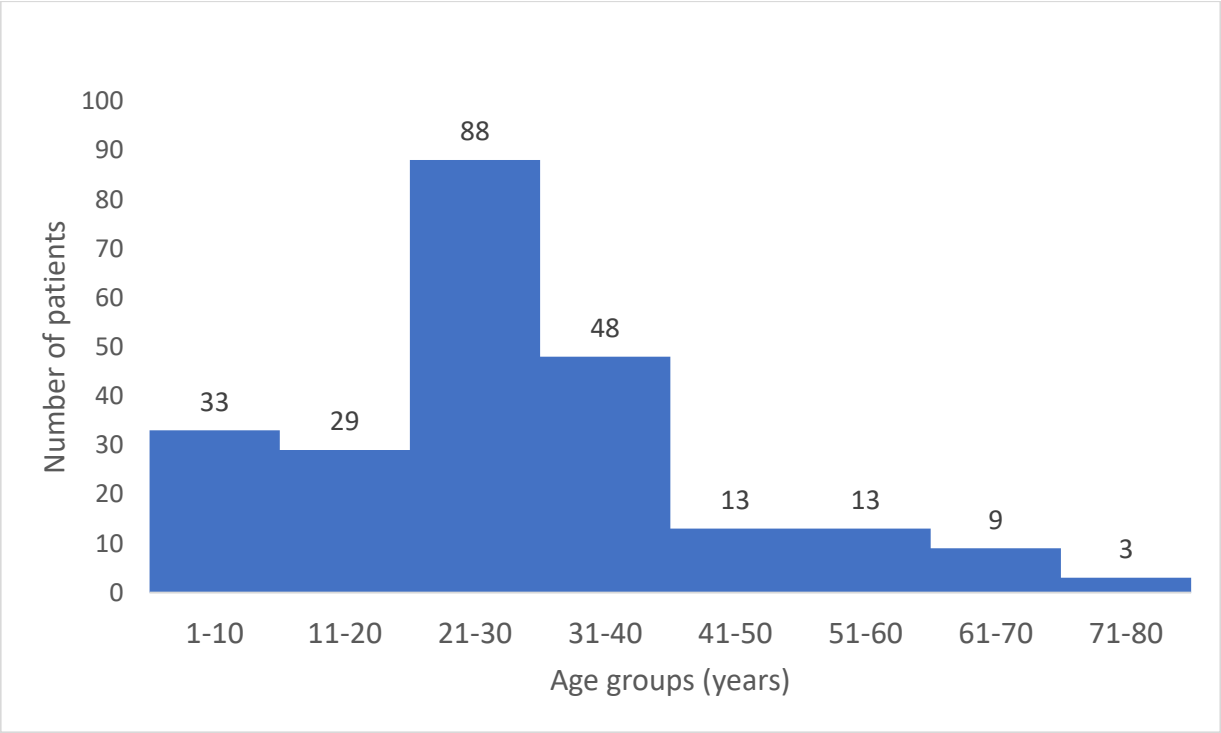


Figure 2: Histogram showing age distribution in the study population

AGE DISTRIBUTION (YEARS)	NUMBER (%)
1-10	33 (13.9%)
11-20	29 (12.2%)
21-30	88 (37.2%)
31-40	48 (20.3%)
41-50	13 (5.5%)
51-60	13 (5.5%)
61-70	9 (3.8%)
71-80	3 (1.2%)

Table- 3: Age distribution in the study population

SEX DISTRIBUTION

Out of the 236 patients 143 were male and 93 were female**.**

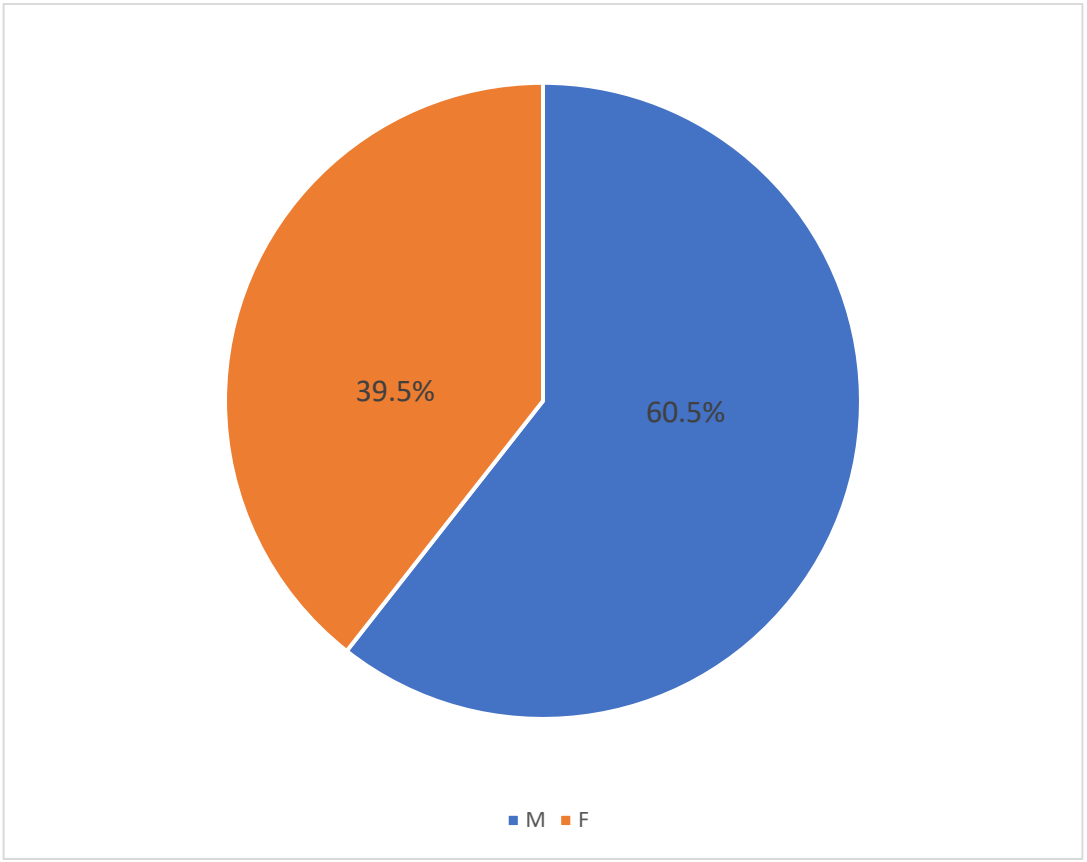


Figure 3: Sex distribution in the study population

EDUCATIONAL STATUS

Out of the 236 patients included in the study, 190 patients were aged more than 18 years. These patients were included to compare the educational status of the patient. Out of 190 patients, 88 patients were graduates and postgraduates and 83 were having high school and higher secondary school as educational status.

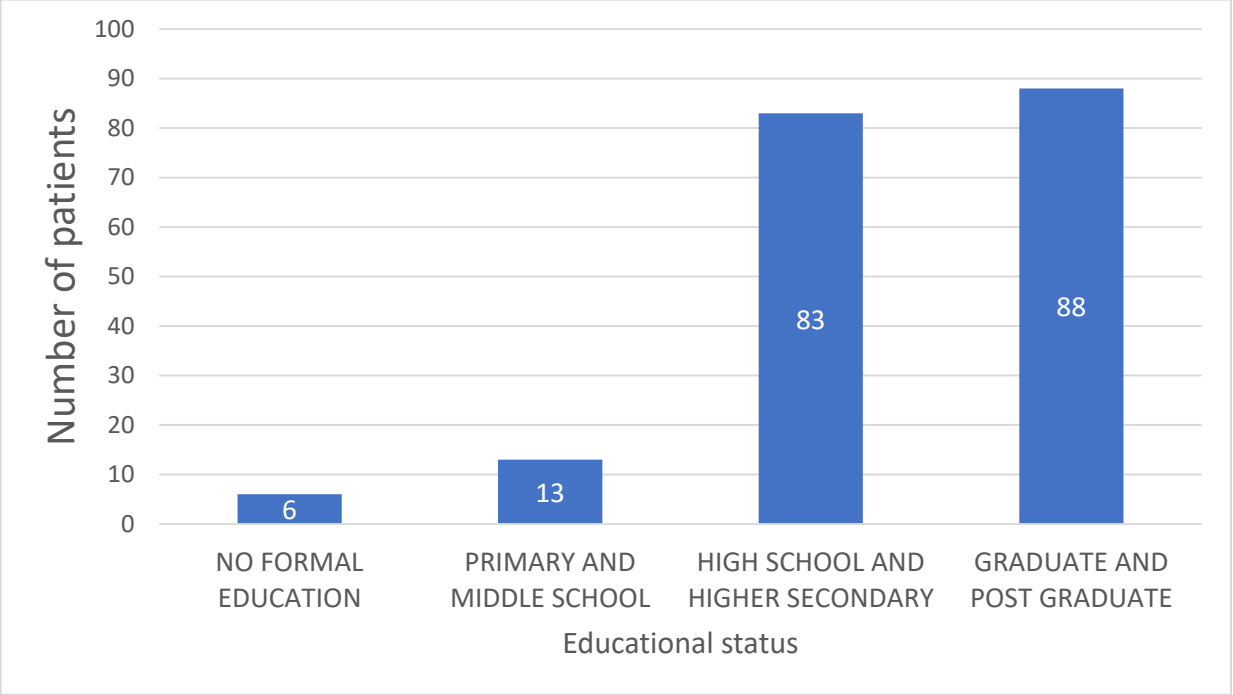


Figure 4: Educational status in the study population (Age > 18 years)

EDUCATION	NUMBER (%)
NO FORMAL EDUCATION	6 (3.1%)
PRIMARY AND MIDDLE SCHOOL	13(6.8%)
HIGH SCHOOL AND HIGHER SECONDARY	83 (43.6%)
GRADUATE AND POSTGRADUATE	88 (46.3%)

Table 4: Educational status of the study population (Age > 18 years)

MARITAL STATUS

Out of the 236 patients included in the study, 190 patients were aged more than 18 years. These patients were included to compare the marital status of the patient. 112 out of 190 patients were married (58.95%).

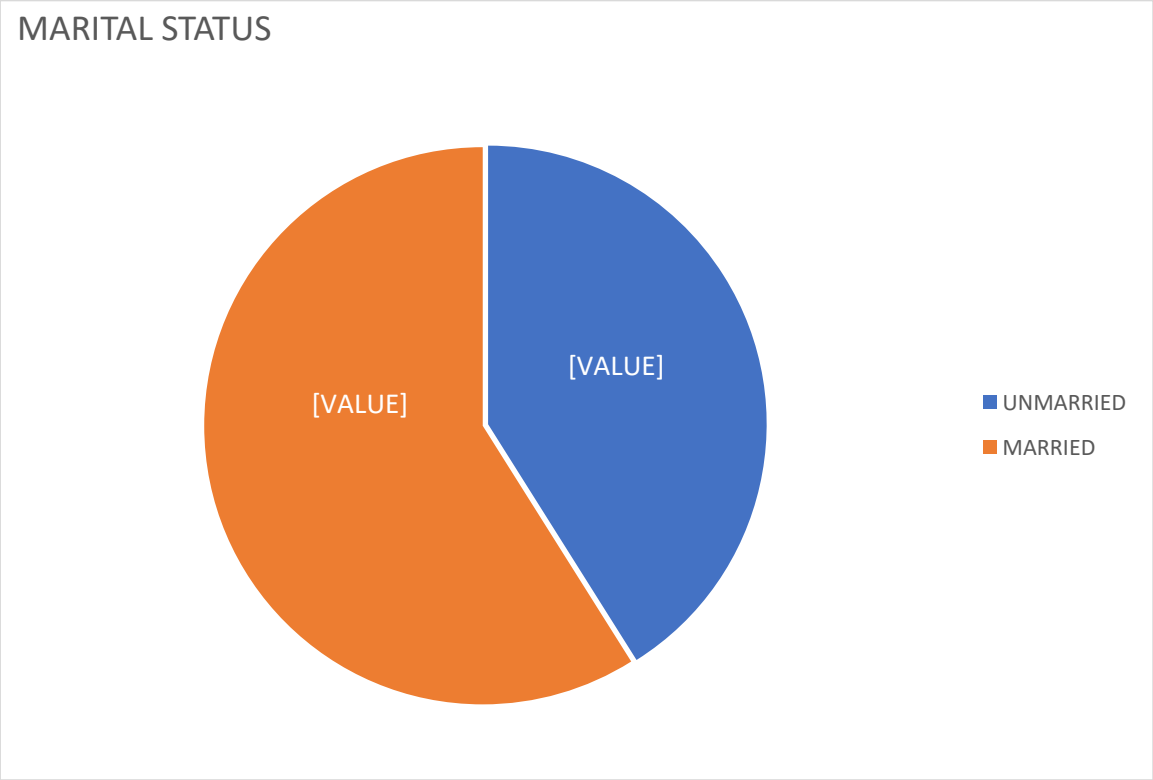


Figure 5: Marital status in the study population (Age > 18 years)

MARITAL STATUS	NUMBER (%)
UNMARRIED	78 (41.05%)
MARRIED	112 (58.95%)

Table- 5: Marital status of the study population (Age > 18 years)

POPULATION DISTRIBUTION

Out of 236 patients, 149 (63.1%) patients belonged to urban areas, as shown in figure-6

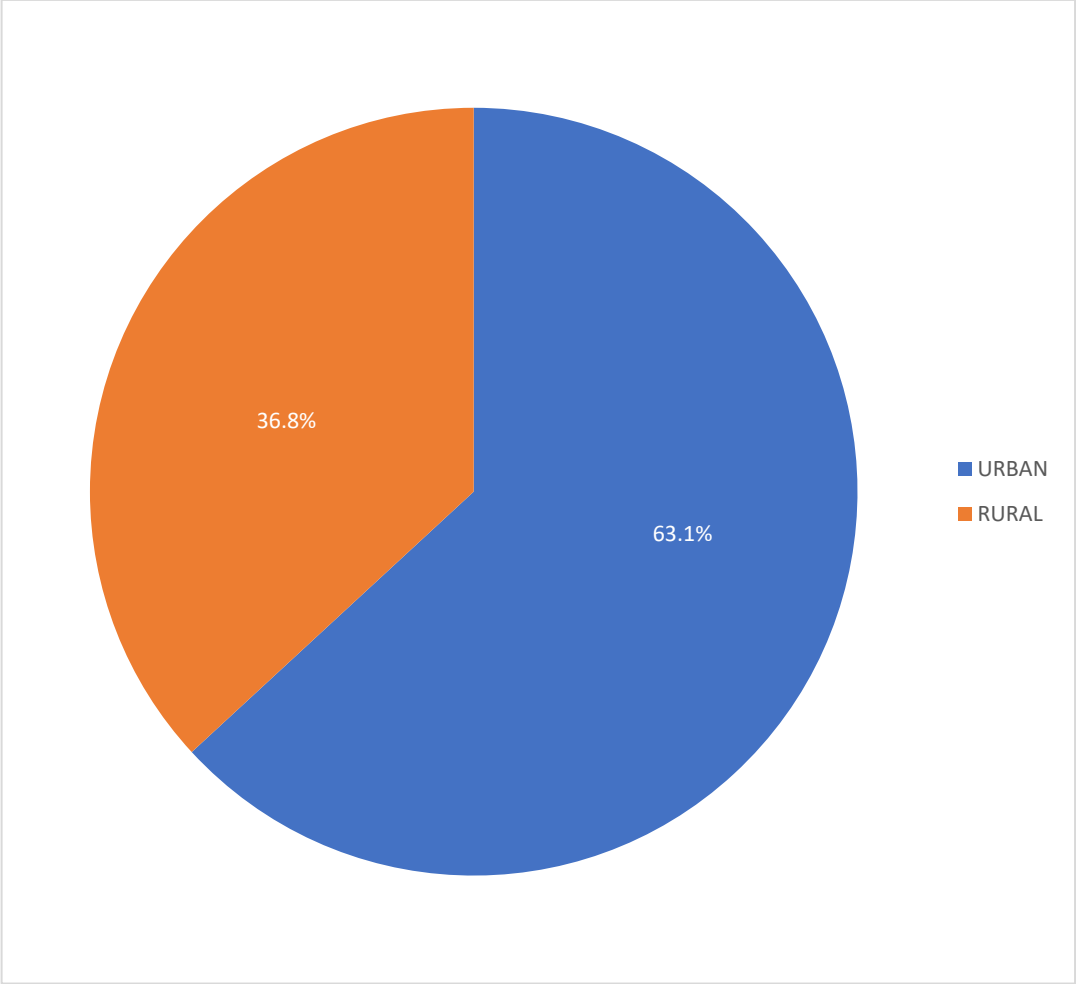


Figure 6: Population distribution of the study participants

POPULATION DISTRIBUTION	NUMBER (%)
URBAN	149 (63.1%)
RURAL	87 (36.8%)

Table 6: Population distribution of the study participants

COMORBIDITIES

Comorbidities were present in 32 patients. Psychiatric illness was the most common comorbidity in 18 out of 32 patients.

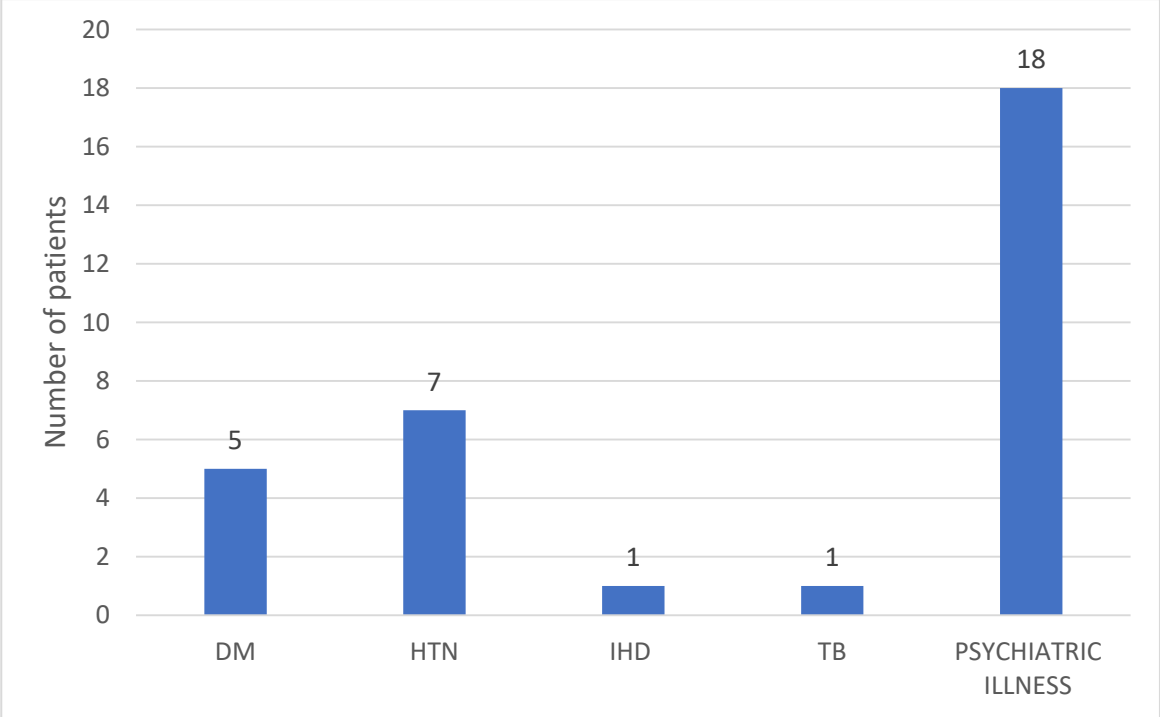


Figure 7: Comorbidities in the study population

COMORBIDITIES	NUMBER (% out of total population)
PSYCHIATRIC ILLNESS	18 (7.6%)
HYPERTENSION (HTN)	7 (2.9%)
DIABETES MELLITUS (DM)	5 (2.1%)
TUBERCULOSIS (TB)	1 (0.4%)
ISCHEMIC HEART DISEASE (IHD)	1 (0.4%)

Table- 7: Comorbidities in the study population

PSYCHIATRIC ILLNESS

Eighteen patients in the study population had previously been diagnosed with psychiatric illness. Depression was the most common psychiatric illness, followed by insomnia.

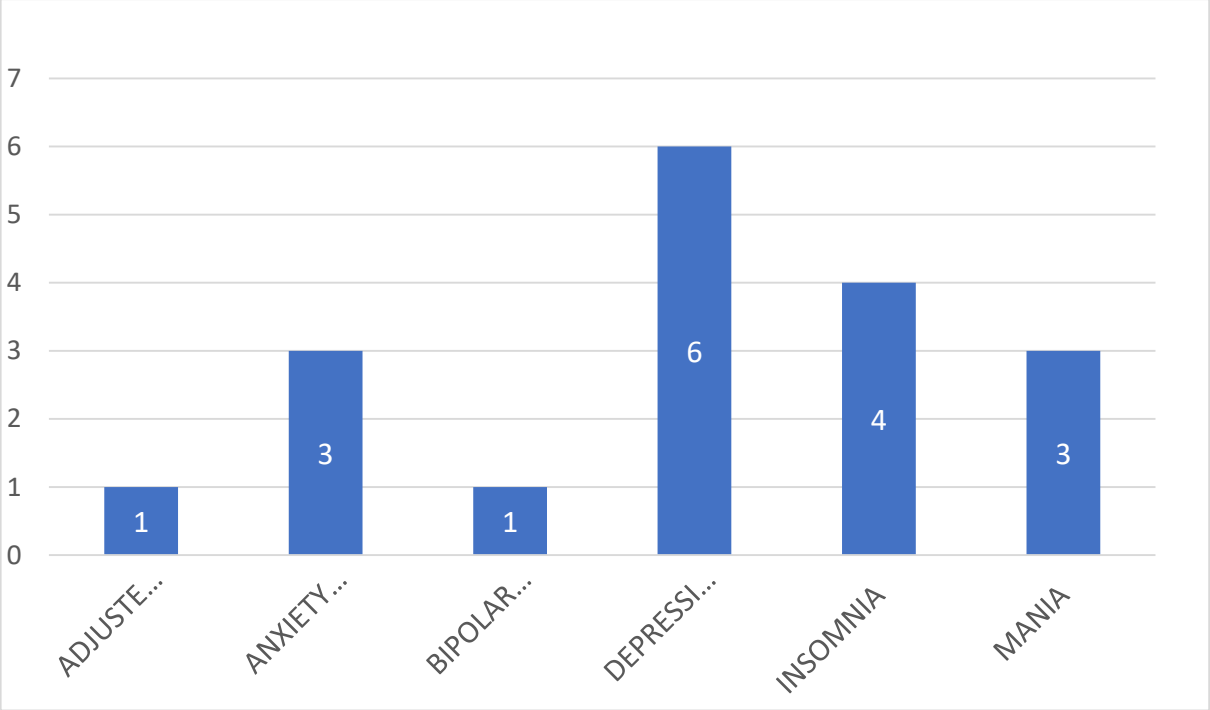


Figure 8: Psychiatric illnesses in the study population

PSYCHIATRIC ILLNESS	NUMBER OF PATIENTS WITH PSYCHIATRIC ILLNESS (%)
DEPRESSION	6 (33.3%)
INSOMNIA	4 (22.2%)
MANIA	3 (16.6%)
ANXIETY DISORDER	3 (16.6%)
BIPOLAR DISORDER	1 (5.5%)
ADJUSTMENT DISORDER	1 (5.5%)

Table- 8: Psychiatric illnesses in the study population

INTENTION OF POISONING

The intention of poisoning was suicidal in 185 patients (78.4%) and accidental in 51 patients (21.6%). None of the cases had homicidal intentions.

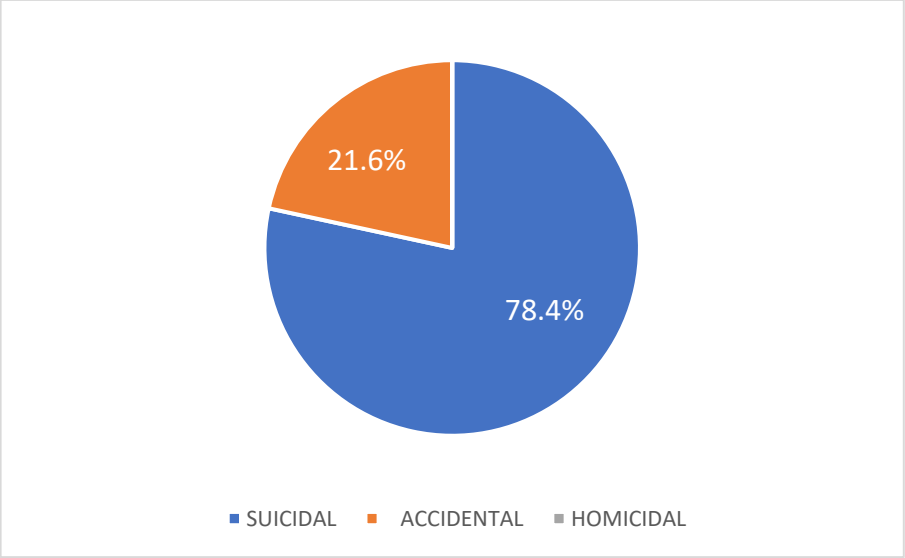


Figure 9: Intention of poisoning

INTENTION	NUMBER (%)
SUICIDAL	185 (78.4%)
ACCIDENTAL	51 (21.6%)

Table- 9: Intention of poisoning

ROUTE OF INGESTION

The oral route was the most common route of ingestion seen in 234 patients (99.1%). The inhalation route of poisoning was seen in only two patients (0.9%). The 2 cases of inhalational poisons were aerosol of organophosphate and fumes of cadmium and sulphur.

ROUTE	NUMBER (%)
ORAL	234 (99.1%)
INHALATION	2 (0.9%)

Table 10: Route of ingestion

CAUSATIVE AGENTS OF PATIENTS WITH ACUTE POISONING

Organophosphate was the most common poisoning presenting to emergency seen in 55 patients (23.3%), followed by drug ingestion seen in 44 patients (18.6%). In both the sexes, organophosphate was the most common with 30 and 25 cases in male and female respectively.

POISON		NUMBER OF PATIENTS	PERCENTAGE
ORGANOPHOSPHATE		55	23.3%
DRUGS		44	18.6%
DETERGENTS AND CORROSIVES		38	16.1%
ALUMINIUM PHOSPHIDE		24	10.1%
ALCOHOL (ETHANOL)		10	4.2%
CANNABIS		10	4.2%
HYDROCARBON	PAINT THINNER	9	3.8%
	PETROL AND DIESEL	6	2.5%
	KEROSENE	5	2.1%
	TURPENTINE OIL	2	0.85%
	CAMPHOR	2	0.85%
OPIUM		5	2.1%
HAIR DYE (PARA-PHENYLENEDIAMINE)		2	0.85%
COPPER SULPHATE		1	0.42%
POTTASIUM PERMANGANATE		1	0.42%
PAINT		1	0.42%
MERCURY		1	0.42%
CADMIUM SULPHIDE		1	0.42%
AMMONIUM DICHROMATE		1	0.42%
UNKNOWN		18	7.6%

Table - 11: COMMON DRUGS OF INGESTION

There were 44 patients who presented with a history of drug ingestion.

DRUGS	NUMBER OF PATIENTS INGESTED THE DRUG
BENZODIAZEPINES	18
PARACETAMOL	2
CYCLOPAM	2
TRAMADOL	2
AMLODIPINE	2
PHENOBARBITONE	2
TRIHEXYPHENIDYL	1
METHAMPHETAMINE (MDMA)	1
TERBUTALINE	1
MINOXIDIL	1
IRON SYRUP	1
ESCITALOPRAM	1
MULTIDRUG INGESTION	10

Table -12: Ingestion of Drugs in poisoning patients

DURATION OF STAY IN HOSPITAL

Out of 158 (excluding the patients who were referred, absconded, leave against medical advice), the maximum duration of stay in the hospital was 600 hours, and the minimum was 3 hours. The median duration of stay was 48 (3-96) hours.

SYMPTOM DISTRIBUTION

Out of the 236 patients in the study population, 219 were symptomatic. The most common symptom at the time of presentation was nausea and vomiting (37.1%), followed by altered sensorium (17.3%).

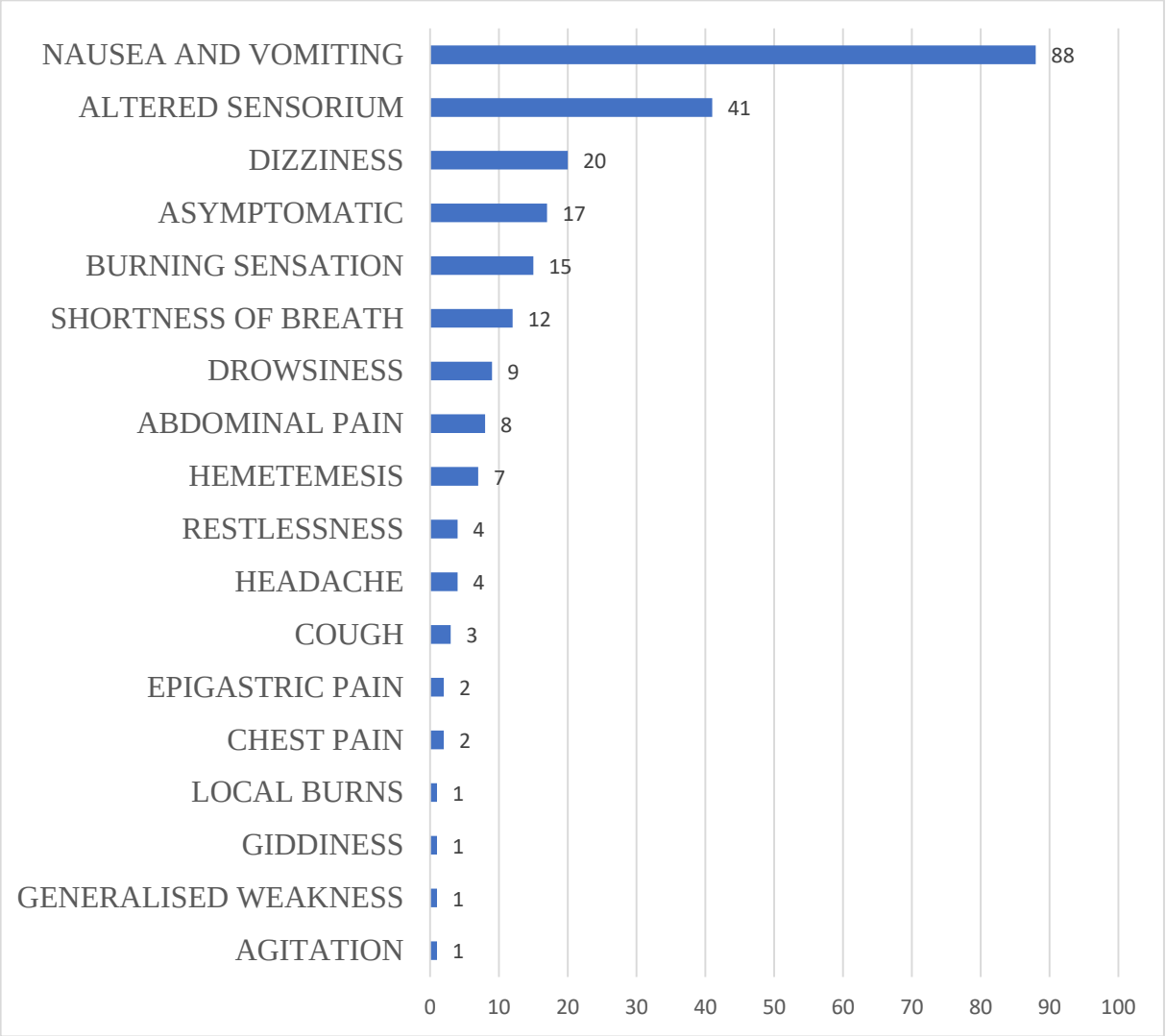


Figure 10: Symptom distribution in the patients with acute poisoning

GLASGOW COMA SCALE (GCS) AT PRESENTATION

Out of 236 patients, 195 patients had a GCS of 15 at presentation, and 41 patients had a GCS of <15 at presentation.

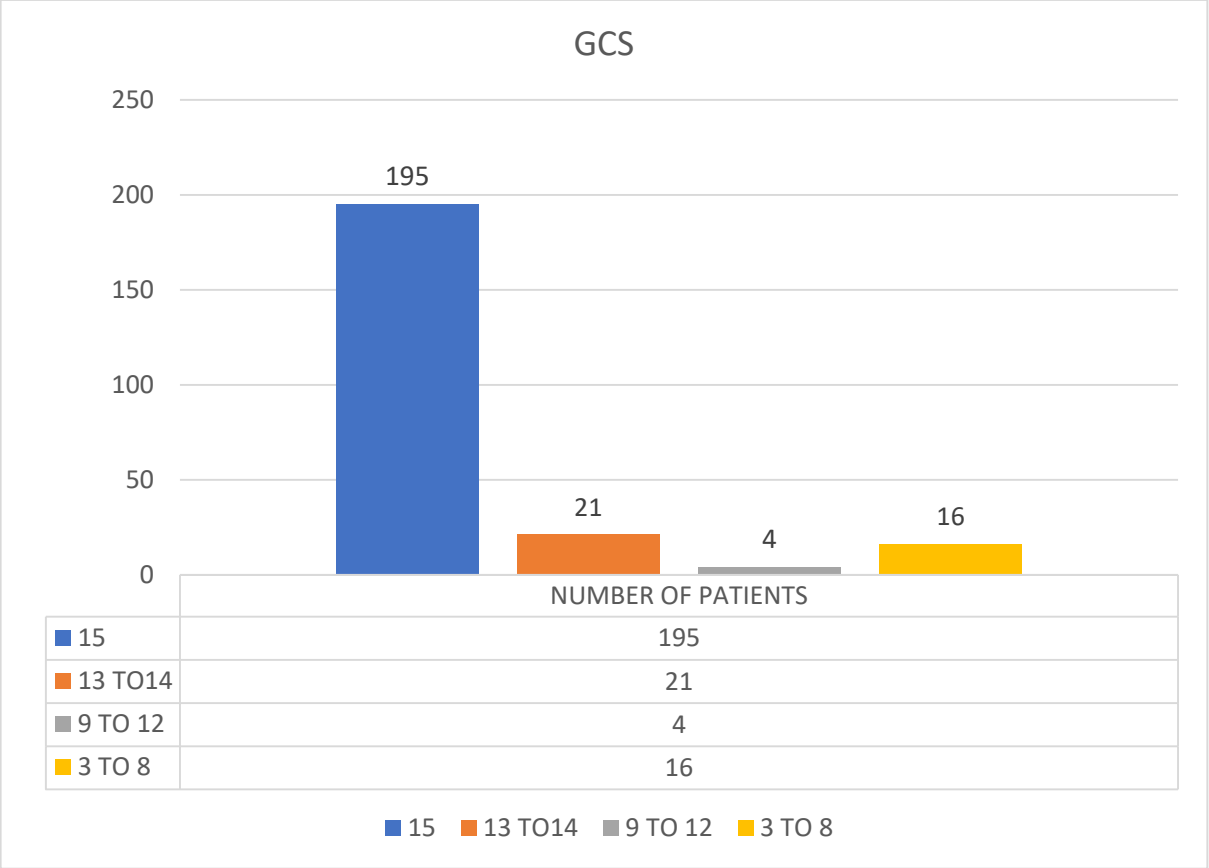


Figure 11: GCS at presentation among acute poisoning patients

GCS AT PRESENTATION	NUMBER OF PATIENTS (%)
15	195 (82.6%)
13 TO14	21 (8.9%)
9 TO 12	4 (1.7%)
3 TO 8	16 (6.8%)

Table - 13: GCS at presentation among acute poisoning patients

GASTRIC LAVAGE

Gastric lavage was given to 68 patients. Gastric lavage was given to 30 patients with organophosphate and 18 patients with Aluminium phosphide poisoning.

GASTRIC LAVAGE IN DIFFERENT POISONING PATIENTS

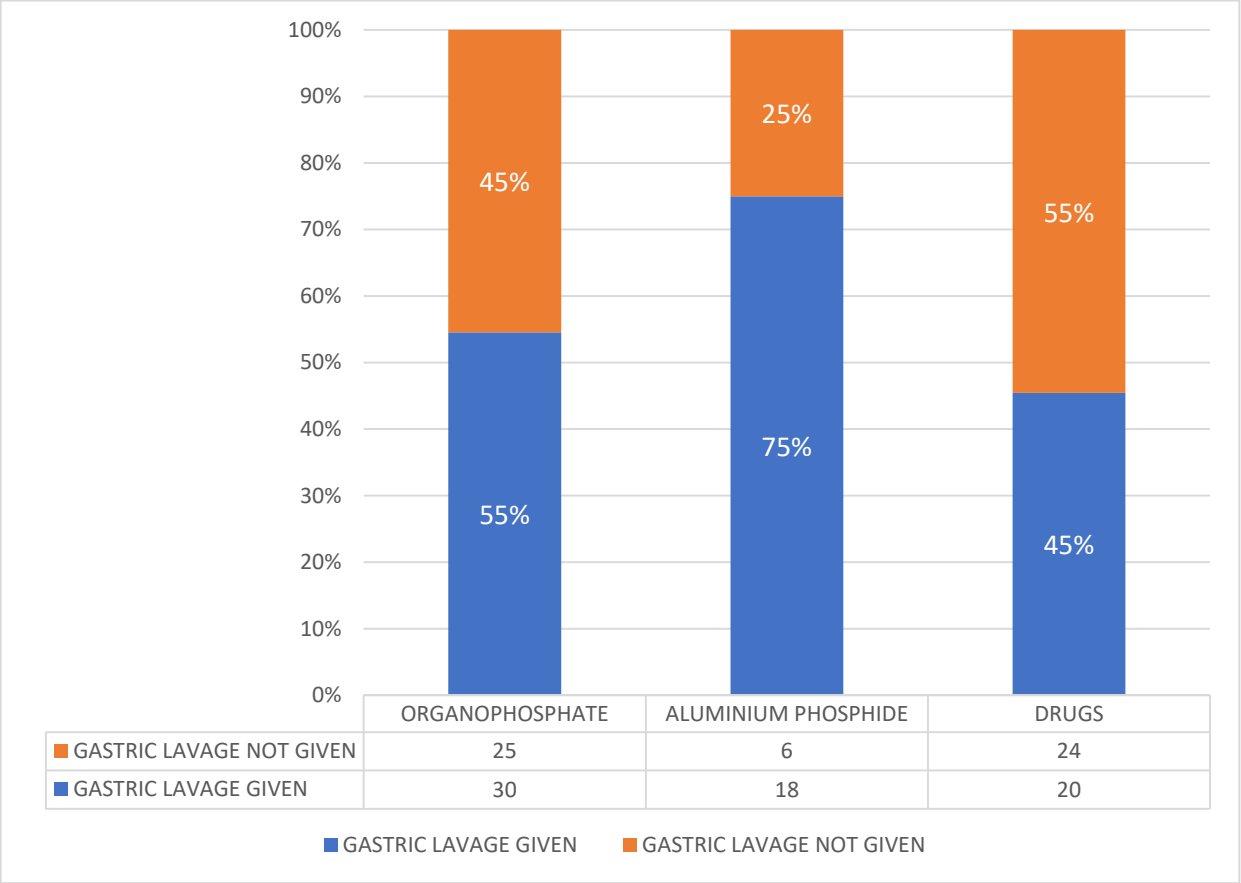


Figure 12: Gastric lavage in different poisoning patients

ACTIVATED CHARCOAL

Activated charcoal was given to 44 patients. The poisoning in which activated charcoal was given mostly is an organophosphate (n=23; 52.2%).

ACTIVATED CHARCOAL IN DIFFERENT POISONING PATIENTS

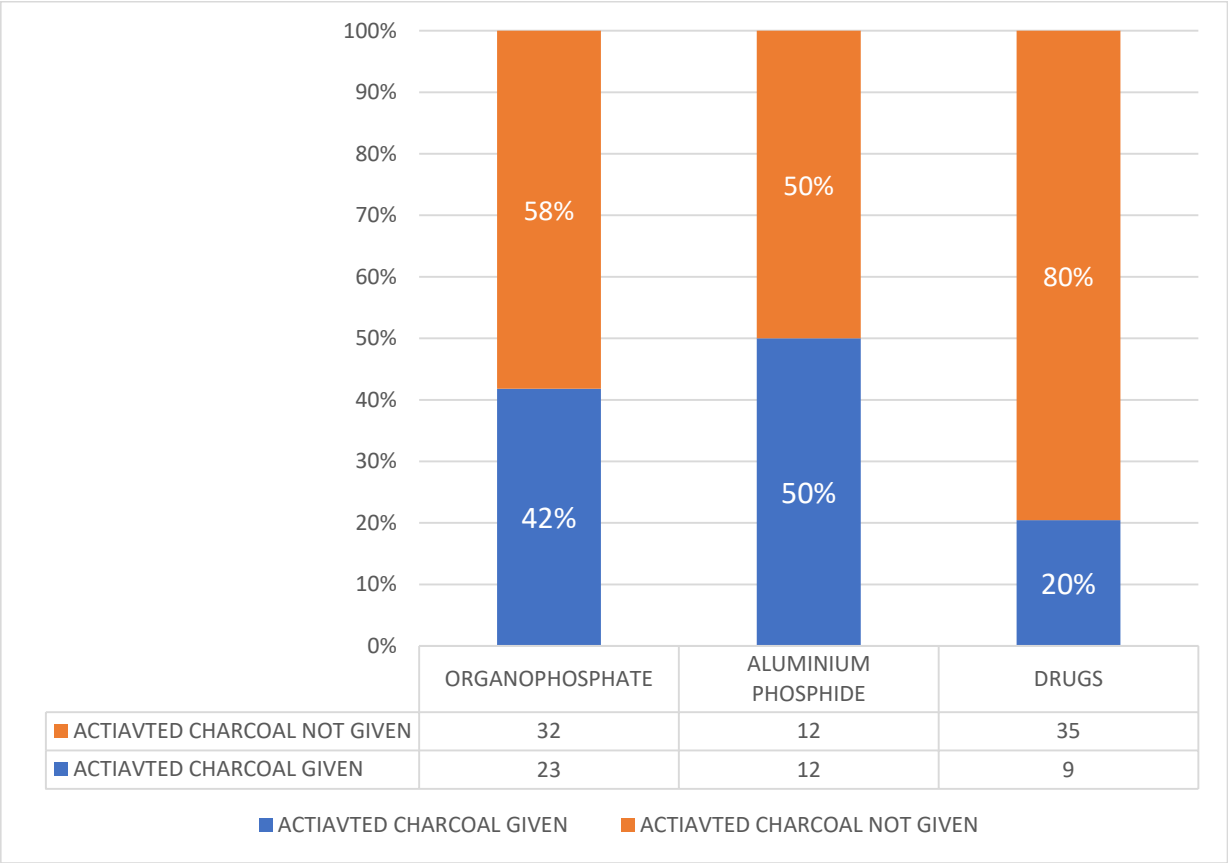


Figure 13: Activated charcoal in different poisoning patients

NEED OF VASOPRESSOR SUPPORT IN EMERGENCY IN DIFFERENT POISONINGS

Of 236 patients, 21 (8.8%) required vasopressor support. Thirteen out of 24 (54%) Aluminium phosphide poisonings required vasopressor support.

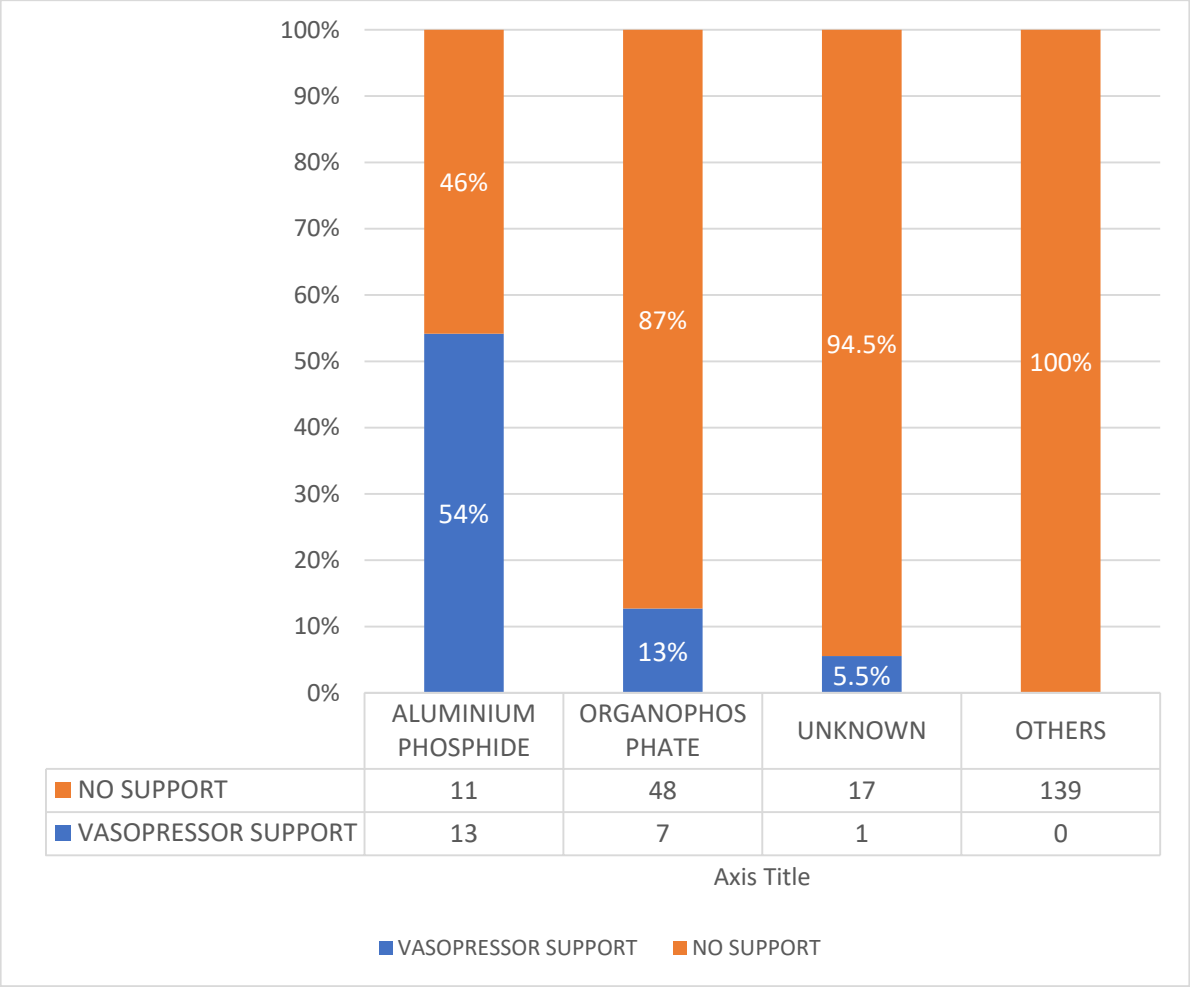


Figure 14: Need of vasopressor support in different poisoning patients

PERCENTAGE OF PATIENTS WITH VARIOUS POISONING REQUIRED VASOPRESSOR SUPPORT

Thirteen out of 21 (61.9%) of the patients who required vasopressor support was Aluminium phosphide poisoning, and 33.3% patients had organophosphate poisoning, as shown in figure-14.

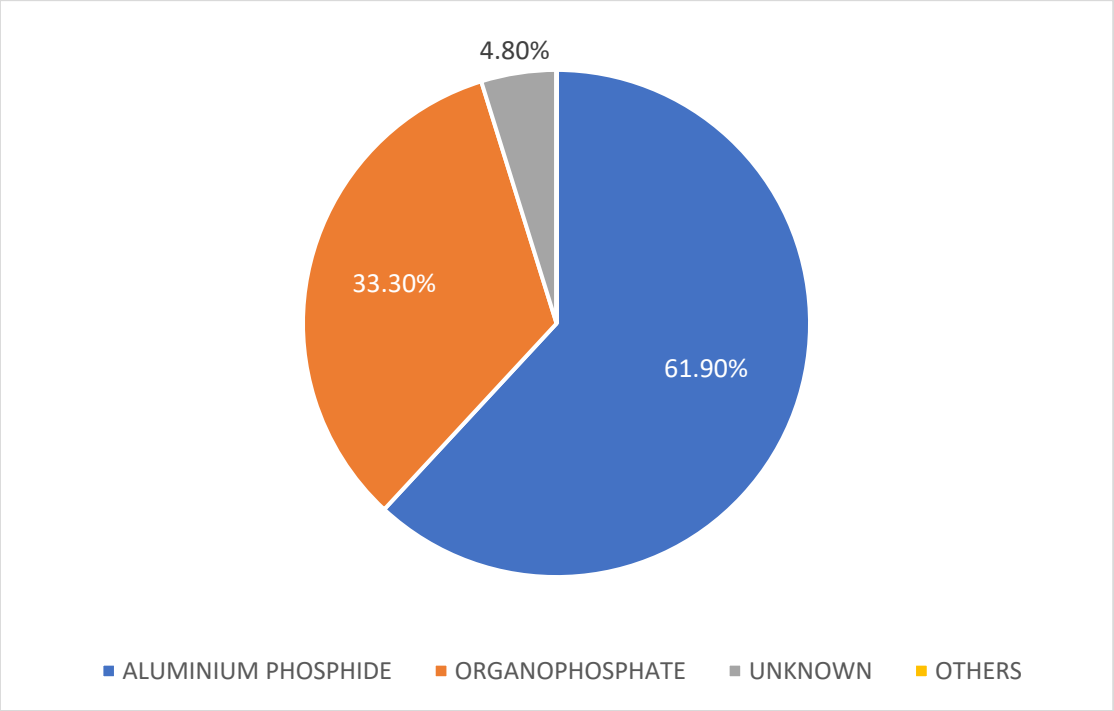


Figure 15: Percentage of patients with various poisoning requiring vasopressor support

POISON	PERCENTAGE OF THE PATIENTS WITH VARIOUS POISONING REQUIRED VASOPRESSOR SUPPORT (NUMBER)
ALUMINIUM PHOSPHIDE	61.9% (13)
ORGANOPHOSPHATE	33.3% (7)
UNKNOWN	4.8% (1)

Table -14: Percentage of patients with various poisoning requiring vasopressor support

ORGANOPHOSPHATE POISONING

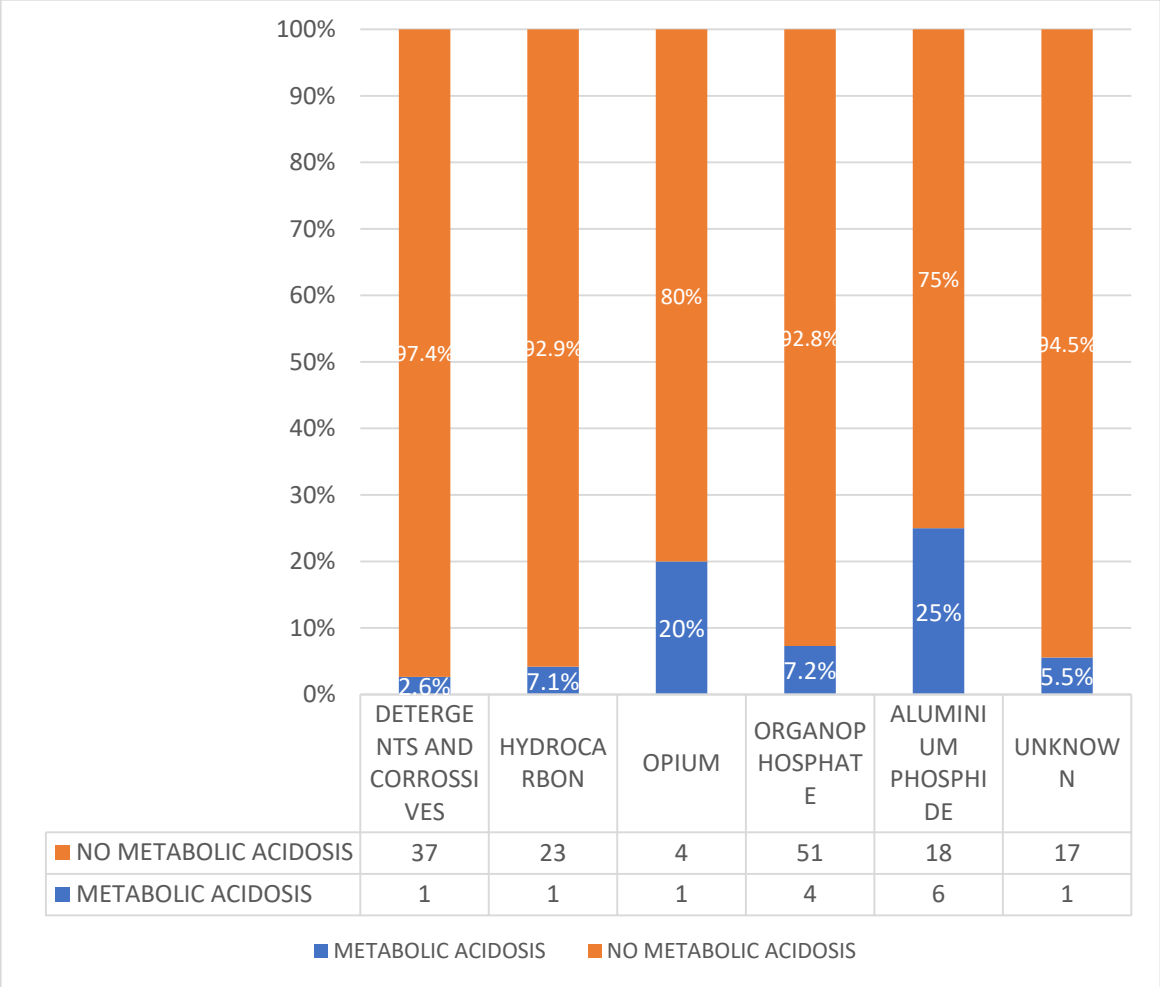
Out of 55 Organophosphate poisoning cases, atropine and pralidoxime were given to 26 (47%) and 8 (15%) patients, respectively.

ALUMINIUM PHOSPHIDE POISONING

Out of 24 Aluminum phosphide poisoning cases, N acetylcysteine, magnesium sulphate, coconut oil and activated charcoal were given to 22 (91.6%), 20 (83%), 15 (62%), 12 (50%) patients, respectively.

METABOLIC ACIDOSIS IN DIFFERENT POISONING PATIENTS

Of 236 poisoning cases, 14 patients (5.9%) had metabolic acidosis. Metabolic acidosis was most commonly seen in Aluminium phosphide poisoning.



	DETERGENTS AND CORROSSIVES	HYDROCARBON	OPIUM	ORGANOPHOSPHATE	ALUMINIUM PHOSPHIDE	UNKNOWN
NO METABOLIC ACIDOSIS	37	23	4	51	18	17
METABOLIC ACIDOSIS	1	1	1	4	6	1

Figure 16: Percentage of metabolic acidosis in different poisoning patients

PERCENTAGE OF VARIOUS POISONINGS IN PATIENTS WITH METABOLIC ACIDOSIS

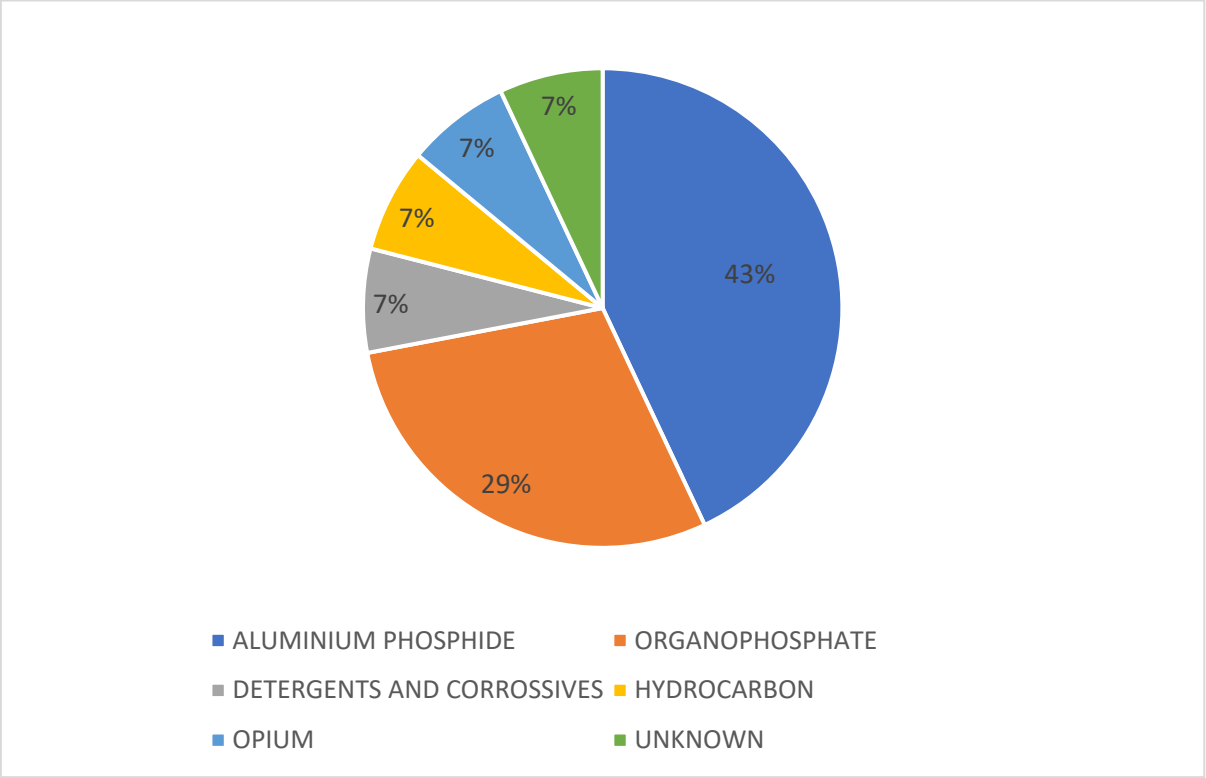


Figure 17: Percentage of various poisoning in patients with metabolic acidosis

POISON	PERCENTAGE OF VARIOUS POISONING IN PATIENTS WITH METABOLIC ACIDOSIS (NUMBER)
ALUMINIUM PHOSPHIDE	43% (6)
ORGANOPHOSPHATE	29% (4)
DETERGENTS AND CORROSIVES	7% (1)
HYDROCARBON	7% (1)
OPIUM	7% (1)
UNKNOWN	7% (1)

Table - 15: Percentage of various poisoning in patients with metabolic acidosis

PERCENTAGE OF RENAL DYSFUNCTION IN PATIENTS PRESENTED WITH DIFFERENT POISONINGS

Out of 236 poisoning cases, eight patients (3.4%) had renal dysfunction. It was seen in 4 out of 24 Aluminium phosphide poisoning patients.

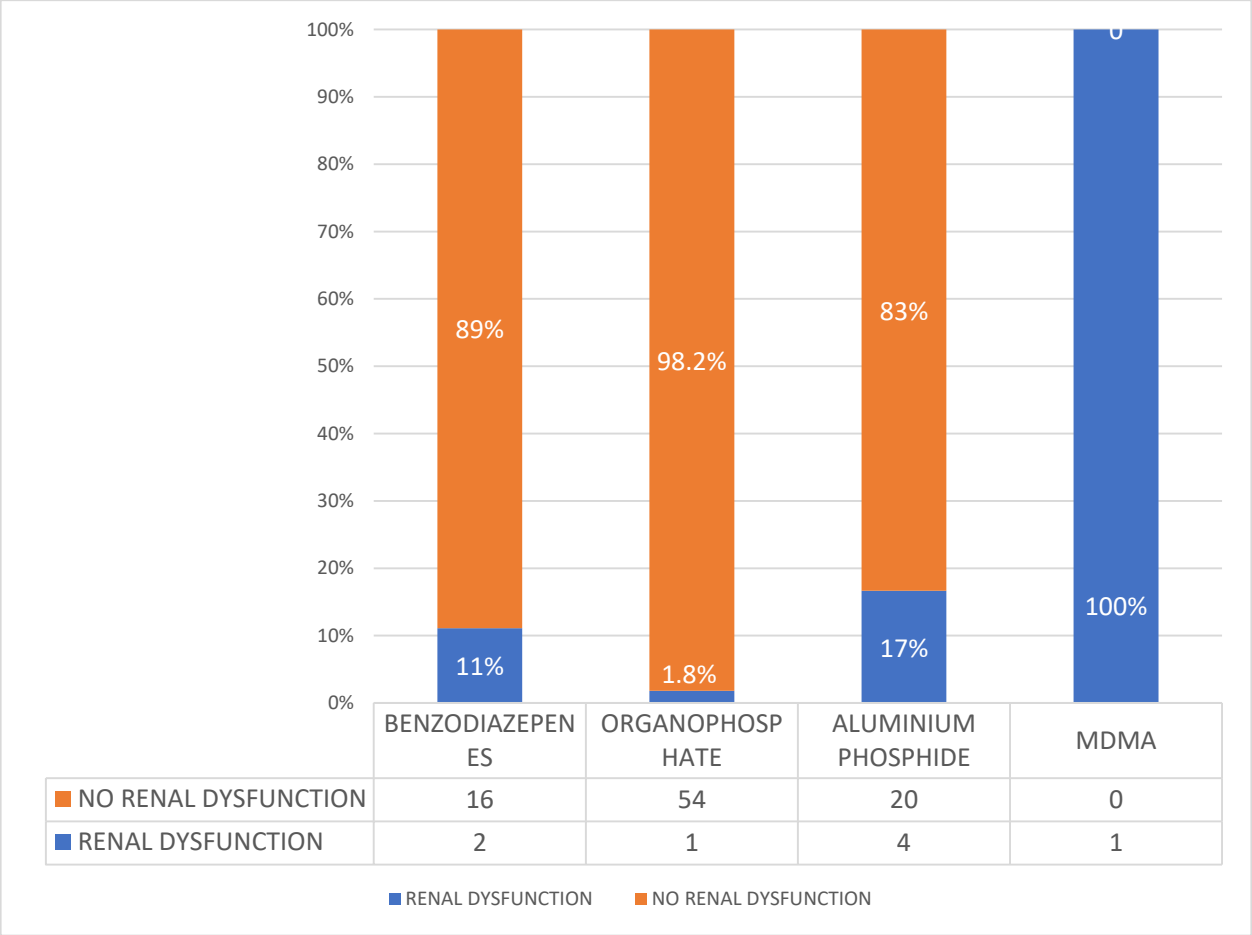


Figure 18: Percentage of renal dysfunction in patients with different poisonings

PERCENTAGE OF VARIOUS POISONINGS IN PATIENTS WITH RENAL DYSFUNCTION

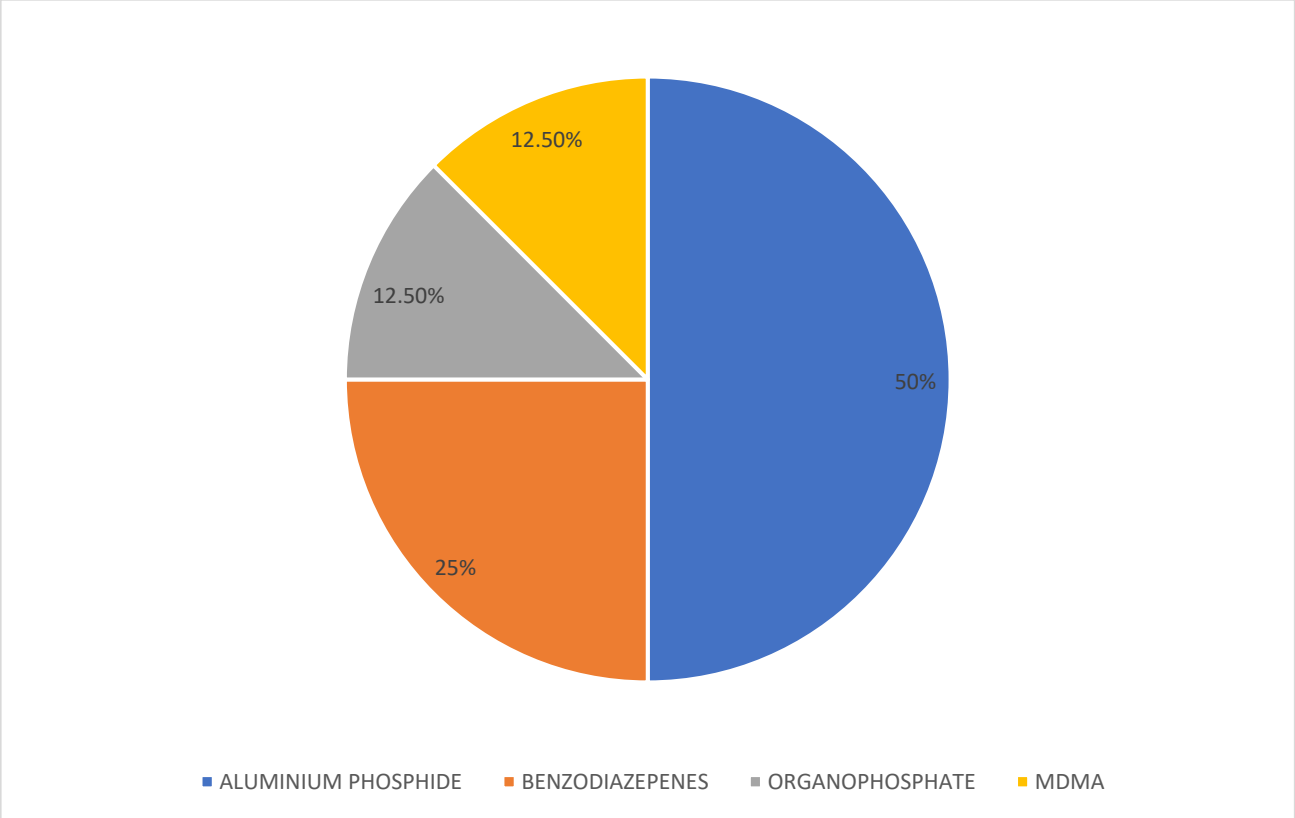


Figure 19: Percentage of various poisoning in patients with renal dysfunction

POISON	PERCENTAGE OF VARIOUS POISONING IN PATIENTS WITH RENAL DYSFUNCTION (NUMBER)
ALUMINIUM PHOSPHIDE	50% (4)
BENZODIAZEPINES	25% (2)
ORGANOPHOSPHATE	12.5% (1)
MDMA	12.5% (1)

Table- 16: Percentage of various poisoning in patients with renal dysfunction

PERCENTAGE OF LIVER DYSFUNCTION IN PATIENTS PRESENTED WITH DIFFERENT POISONINGS

Out of 236 poisoning cases, eight patients had liver dysfunction, as shown in figure-.

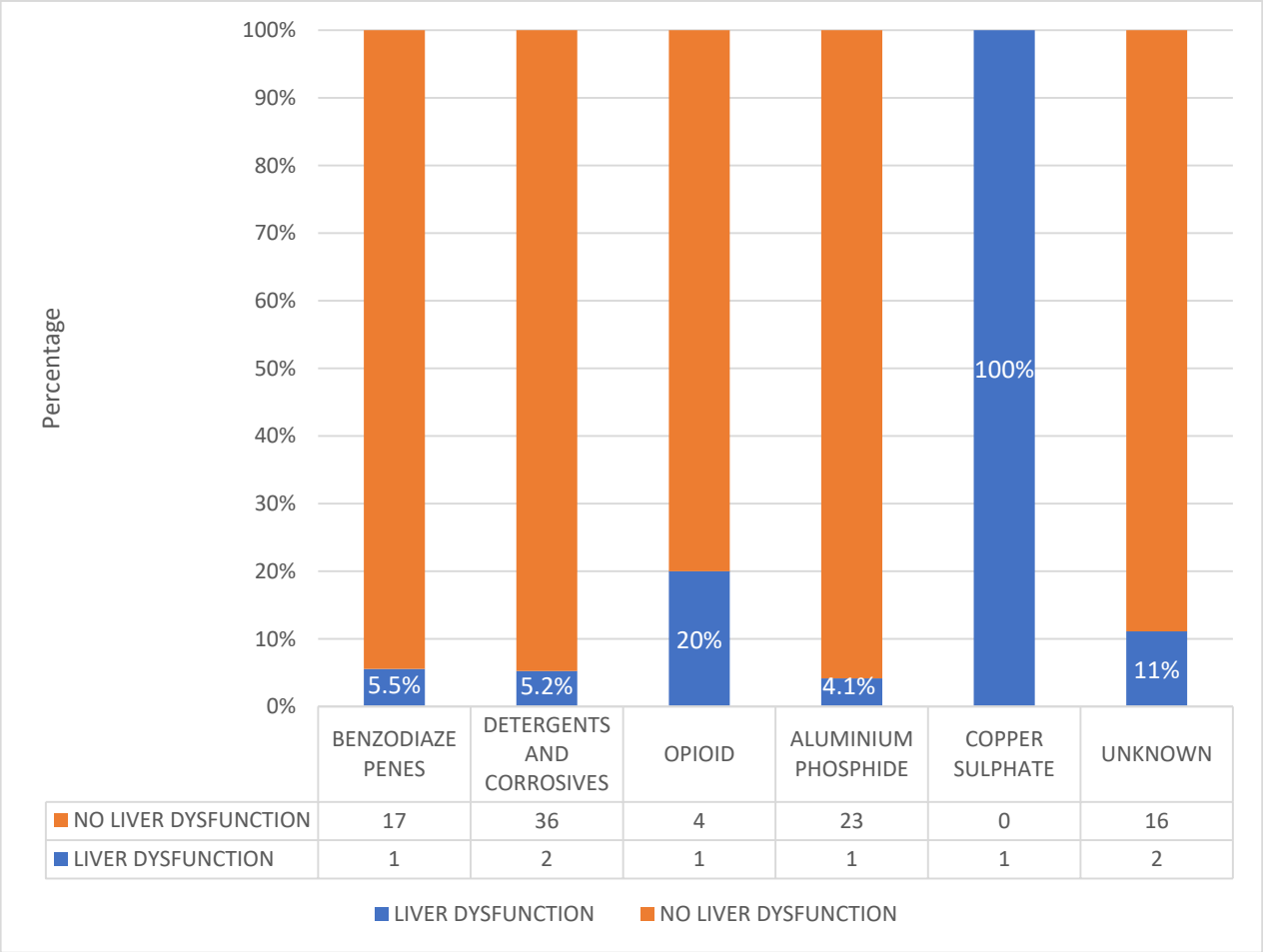


Figure 20: Percentage of liver dysfunction in different poisoning patients

PERCENTAGE OF VARIOUS POISONINGS IN PATIENTS WITH LIVER DYSFUNCTION

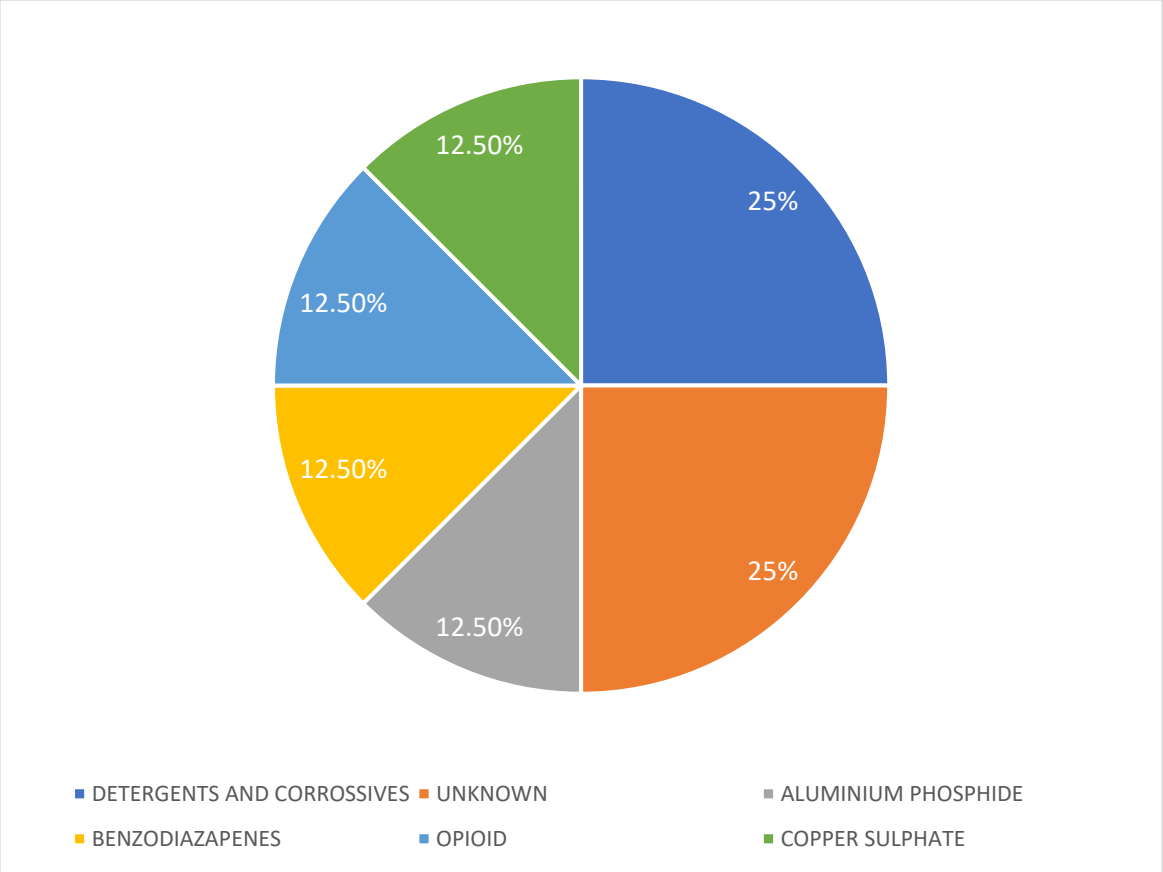


Figure 21: Percentage of various poisoning in patients with liver dysfunction

POISON	PERCENTAGE OF VARIOUS POISONING PATIENTS WITH LIVER DYSFUNCTION (NUMBER)
DETERGENTS AND CORROSIVES	25% (2)
UNKNOWN	25% (2)
ALUMINIUM PHOSPHIDE	12.5% (1)
BENZODIAZEPINES	12.5% (1)
OPIOID	12.5% (1)
COPPER SULPHATE	12.5% (1)

Table - 17: Percentage of various poisoning in patients with liver dysfunction

PERCENTAGE OF COAGULOPATHY IN PATIENTS PRESENTED WITH DIFFERENT POISONINGS

Of 236 poisoning cases, nine patients (0.038%) had coagulopathy. Four out of 24 (16.6%) aluminium phosphide poisoning patients had coagulopathy.

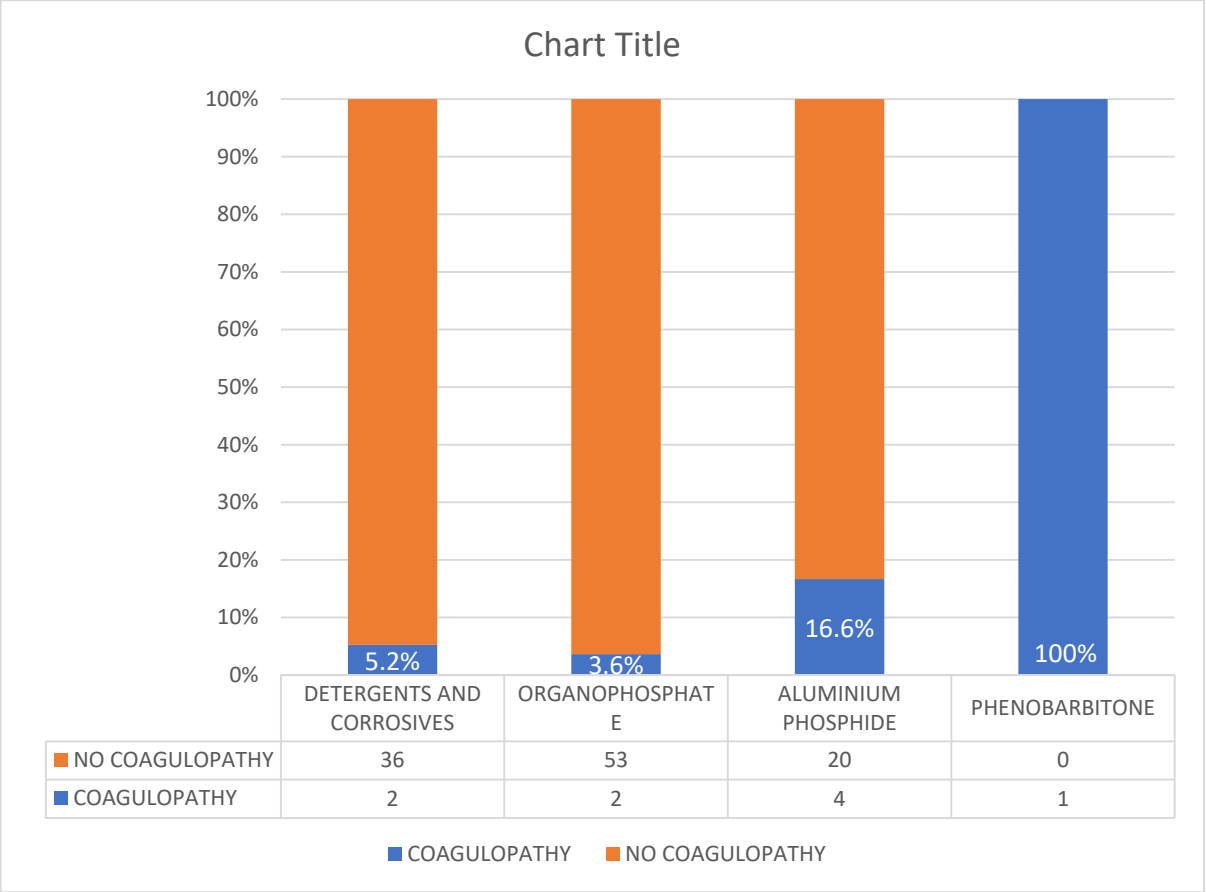


Figure 22: Percentage of coagulopathy in different poisoning patients

PERCENTAGE OF VARIOUS POISONINGS IN PATIENTS WITH COAGULOPATHY

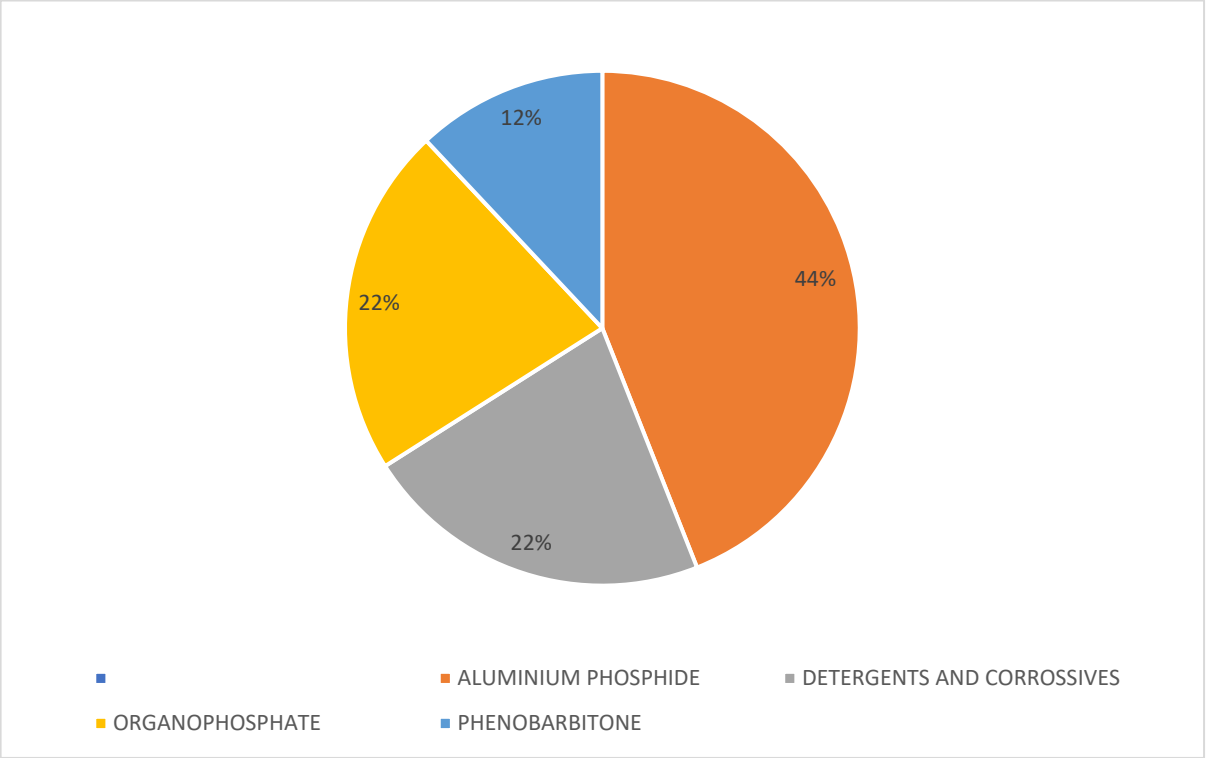


Figure 23 Percentage of various poisoning in patients with coagulopathy

POISON	**PERCENTAGE OF VARIOUS POISONING PATIENTS WITH COAGULOPATHY (NUMBER)**
ALUMINIUM PHOSPHIDE	44% (4)
DETERGENTS AND CORROSIVES	22% (2)
ORGANOPHOSPHATE	22% (2)
PHENOBARBITONE	12% (1)

Table- 18: Percentage of various poisoning in patients with coagulopathy

ICU ADMISSION IN DIFFERENT POISONING

Of 158 (excluding the patients referred, absconded, leave against medical advice), 28 were admitted to ICU.

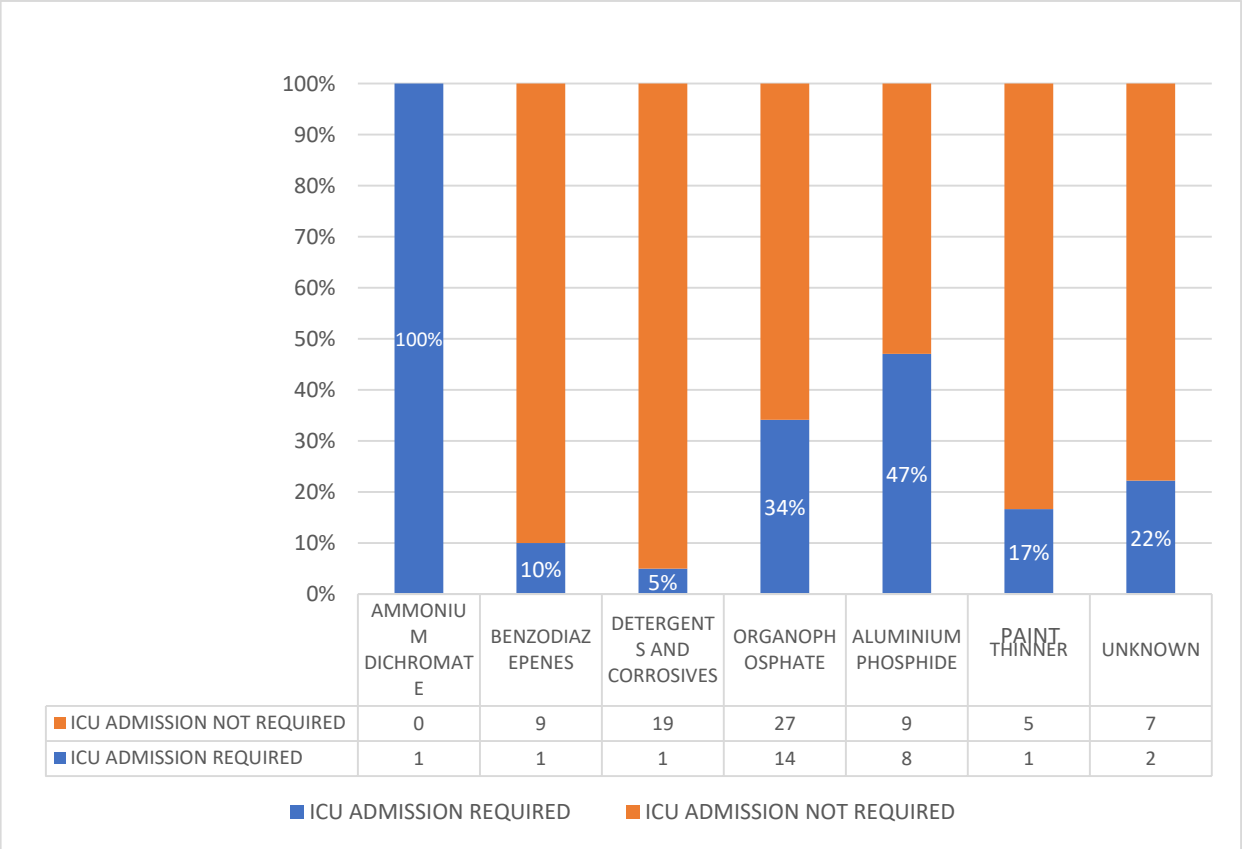


Figure 24: Patients requiring ICU admission in different poisoning cases

PERCENTAGE OF VARIOUS POISONINGS IN PATIENTS REQUIRING ADMISSION

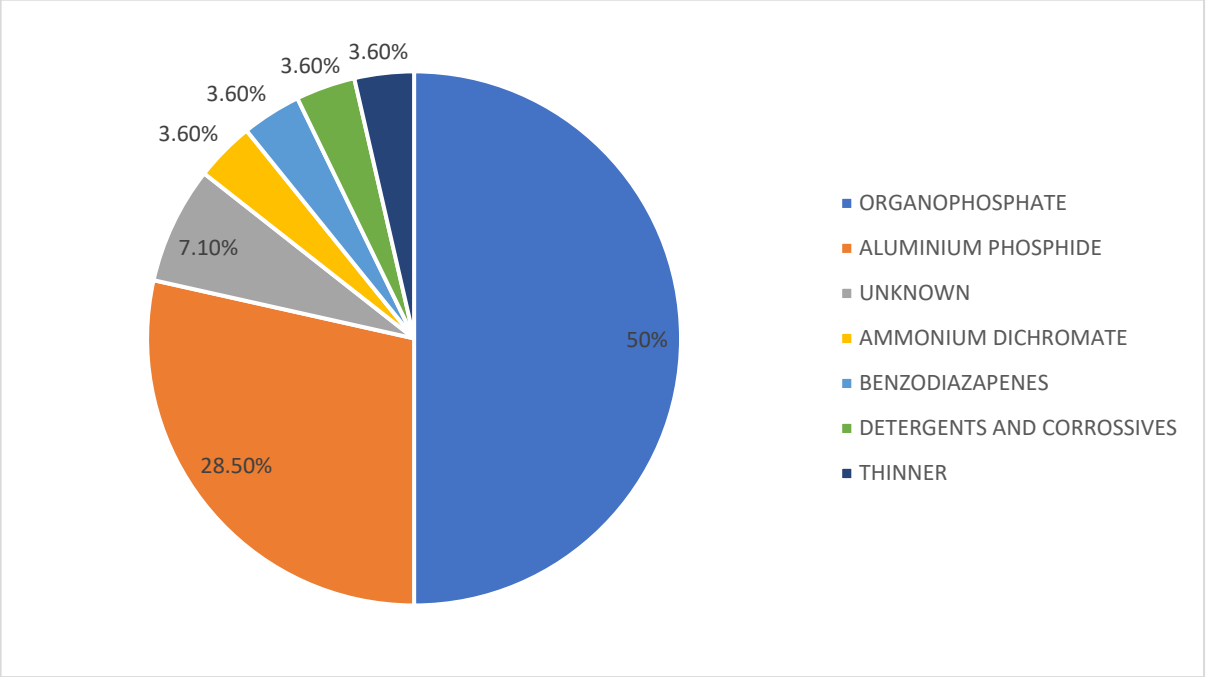


Figure 25: Percentage of various poisoning patients requiring ICU admission

POISON	PERCENTAGE OF VARIOUS POISONING PATIENTS REQUIRING ICU ADMISSION (NUMBER)
ORGANOPHOSPHATE	50% (14)
ALUMINIUM PHOSPHIDE	28.5% (8)
UNKNOWN	7.1% (2)
AMMONIUM DICHROMATE	3.6% (1)
BENZODIAZEPINES	3.6% (1)
DETERGENTS AND CORROSIVES	3.6% (1)
THINNER	3.6% (1)

Table- 19: Percentage of various poisoning patients requiring ICU admission

MORTALITY

(Excluding the patients who were referred, absconded, leave against medical advice)

A total of 10 patients died within 28 days of ingestion of poison. Six out of 10 patients who died were male. Out of these 10 patients, nine had died after admission in ICU. One death occurred in an emergency itself. Overall, 28 days mortality rate was 6.3%.

Out of 17 Aluminium phosphide poisoning with proper follow-up, five patients died with a mortality rate of 29.4%. Out of 41 organophosphate poisoning with proper follow-up, five patients died, with a mortality rate of 12%.

QSOFA AND ICU ADMISSION

Twenty one out of 48 patients with qSOFA ≥ 1 required ICU admission. Patients with qSOFA ≥1 had statistically significant higher requirement of ICU admission (p <0.001).

	qSOFA SCORE = 0	**qSOFA SCORE ≥ 1**	**TOTAL**	**P value***
NO ICU ADMISSION	103	27	130	
ICU ADMISSION	7	21	28	**< 0.001**
TOTAL	110	48	158	

Table –20: Significance of qSOFA in context to ICU admission

LACTATE AND DURATION OF STAY IN THE HOSPITAL

Lactate levels were elevated in 21 out of 158 patients. Patients with elevated lactate levels (>2.2mmol/L) didn't have statistically significant longer duration of stay in the hospital. (p <0.001).

	LACTATE	NUMBER OF PATIENTS	MEDIAN IN HOURS (IQR)	P value
DURATION OF STAY IN HOSPITAL	Not elevated	137	48 (24-72)	0.065
	Elevated	21	72 (18-216)	

Table –21: Significance of elevated lactate levels and duration of stay in hospital

TIME OF PRESENTATION AND MECHANICAL VENTILATION

Out of 158 patients, 19 patients required mechanical ventilation. There was a statistically significant association between the time of presentation to emergency and the requirement of mechanical ventilation. (p <0.001).

MECHANICAL VENTILATION	NUMBER OF PATIENTS	MEDIAN DURATION IN MINUTES (IQR)	P value
Not required	139	110 (60-190)	**0.044**
Required	19	160(110-1340)	

Table -:22 Statistical significance between the time of presentation and mechanical ventilation

AGE AND DURATION OF STAY IN HOSPITAL

There was no statistically significant correlation between age and duration of stay in the hospital. [rho (ρ)= 0.041, p= 0.60]

COMPARISON BETWEEN SURVIVORS AND NON-SURVIVORS IN POISONING CASES

(Excluding the patients who were referred, abscond, leave against medical advice)

Median value of qSOFA was statistically significantly higher in patients who did not survive (p <0.001). There was no statistically significant association between age, sex and survival of the patient.

		Survivors (n-148)	Non-Survivors (n-10)	P value	Test of significance
AGE (Years) Median (IQR)		25 (18-36)	24 (19-35)	0.825	Mann Whitney U test
qSOFA SCORE Median (IQR)		0 (0-1)	2 (1-2)	**<0.001**	
SEX	**FEMALE**	55 (37.2%)	4 (40.0%)	1.0	Fisher Exact test
	MALE	93 (62.8%)	6 (60.0%)		

Table 23- : Significance of age, sex, qSOFA in mortality

Renal dysfunction (P= 0.047) and need of vasopressor support (P= <0.001) had statistically significant association with survival of the patient, but liver dysfunction and coagulopathy did not have significant association with survival of the patient,

LIVER DYSFUNCTION	NO	142 95.9%	10 100%	-	Fisher Exact test
	YES	6 4.1%	0		
RENAL DYSFUNCTION	NO	144 97.3%	8 80.0%	**0.047**	
	YES	4 2.7%	2 20.0%		
COAGULOPATHY	NO	142 95.9%	9 90.0%	0.373	
	YES	6 4.1%	1		
REQUIRED VASOPRESSOR SUPPORT	NO	141 95.3%	2 20.0%	**<0.001**	
	YES	7 4.7%	8 80.0%		

Table 24- : Significance of coagulopathy, need of vasopressor support, liver and renal dysfunction in mortality

COMPARISON BETWEEN ORGANOPHOSPHATE AND NON-ORGANOPHOSPHATE POISONING CASES

Demographic variables, qSOFA score and various outcomes were compared between organophosphorus and non-organophosphorus poisoning. There was no statistically significant association except qSOFA.

		Organophosphate (n-41)	Non-Organophosphate (n-117)	P value	Test of significance
AGE (Years) Median (IQR)		25 (19-40)	26 (18-35)	0.508	Mann Whitney U test
qSOFA SCORE Median (IQR)		0 (0-1)	0 (0-0)	**0.004**	
SEX	**FEMALE**	17 10.8%	42 26.6%	0.526	Chi-Square test
	MALE	24 15.2%	75 47.5%		

Table 25 - : Comparison of variables like age, sex and qSOFA in organophosphate and non-organophosphate poisoning

	YES/NO	Organophosphate	Non-Organophosphate	P value	Test of significance
LIVER DYSFUNCTION	NO	41 25.9%	111 70.3%	1.0	Fisher Exact test
	YES	0 0	6 3.8%		
RENAL DYSFUNCTION	NO	40 25.3%	112 70.9%	-	
	YES	1 0.6%	5 3.2%		
COAGULOPATHY	NO	39 24.7%	112 70.9%	1.0	
	YES	2 1.3%	5 3.2%		
VASOPRESSORS	NO	36 22.8%	107 67.7%	0.539	
	YES	5 3.2%	10 6.3%		

Table 26 -: Comparison of variables like coagulopathy, need of vasopressor support, liver and renal dysfunction in organophosphate and non-organophosphate poisoning

DISCUSSION

As per Shah S et al., 2% to 3% of all hospital admissions in India are due to acute poisoning cases (79). However in the present study the prevalence of poisoning was 0.53%. A study by Singh et al. from 2001 to 2003 showed that the majority (36%) of the cases belonged to the age group 21-30 years(80). Thapa et al. did a study in 2007-2008 that showed the most common age group was 21-30 years (81). Tanuj et al. (2000-2006) showed that the victims varied in age from 2 to 82 years, with the third decade of life being shown as the highest prevalence (82). In our study, the maximum number of patients were from the age group of 21-30 years (37.2%), followed by age groups 31-40 years (20.3%). The mean age of the patients in our study was 27.8± 16.1 years, comparable to the above studies. The tendency of increasing instances in the 21–40 age range is brought on by increased work stress, family issues, financial strain, and other life-settling issues in this age group.

In a study done by Patel et al. in 2011-2013, 59.2% out of 135 patients were male(3). Another study done in 2013-14 by Shah et al. had 216 males among 340 cases(79). In a study by Indu et al. in 2018-2013 on 1860 patients, 62% of patients were male. In a systematic review by Bhagavathula et al., the gender ratio was found to be 2.1:1 (female: male). In our study of 236 participants, 143 (60.5%) were male. The study by Shah S et al. mentioned that most male instances of poisoning might be due to their greater exposure to stress in everyday life and work dangers compared to females(79).

According to one study comparing the education status of patients with poisoning, the majority of patients with acute poisoning were illiterate (59.2%), followed by primary school education (21.4%), secondary school (16.5%), and higher education (2.9%) level (83). 190 of the 216 individuals who participated in our research were older than 18 years. These patients were taken into account while comparing the patients' educational backgrounds. In our study comparing the education status of patients, the majority were graduates and postgraduates (46.3%), followed by high school and higher secondary education (43.6%), primary and middle school education (6.8%) and 3.1% patients having no formal education.

Indu et al. conducted a study showing the predominance of suicidal poisoning in married people. The greater poisoning prevalence among married people might be attributed to financial strain, dysfunctional families, and stress (84). In our study comparing the marital status of 190 patients aged more than 18 years, we found that 112 patients (58.95%) were married. Dispute (family or marital) (75.9%), psychiatric issue (14.8%), and substance

addiction (9.3%) were often mentioned among the 54 patients who provided their cause of poisoning in the study by Eyosias et al. (103).

In our study, psychiatric illness was the most common comorbidity seen in 18 out of 236 patients (7.6%), followed by hypertension in 7 patients (2.9%). Out of the 18 patients with psychiatric illness, depression is the most common, seen in 6 patients (33.3%), followed by insomnia seen in 4 patients (22.2%), mania and anxiety disorders seen in 3 patients (16.6%), bipolar disorder and adjustment disorder seen in 1 patient (5.5%).

As per Shah et al., the most common route of exposure is ingestion (71.4%), followed by dermal and inhalation routes (79). In a study by Pannu et al., ingestion for self-harm remained the predominant method of poisoning. In their study the ratio of intentional self-harm to unintentional exposure was 4:1(86). Intentional poisoning was the most common mode of poisoning (50.5%) in the study done by Eyosias et al. The most typical underlying cause of intentional poisonings was a brief argument with a family member or spouse (n=41, 75.9%) (85). In a study by Senarathna et al., the primary causes of self-poisoning attempts were alcoholism, relationship and financial issues, family conflicts, physical and psychological abuse, and family disagreements(5). A study by Bjornaas et al. showed that among the fatalities, 32 cases were suicides and 71 cases were accidents comprising 31% and 69%, respectively (66). In the study by Pannu et al., both males and females had comparable intentions (self-harm versus accidental) (77.8% vs 22.2% and 79.7% vs 20.3%, respectively). Adolescents (13-19 years) were more likely to be exposed accidentally, while young adults (20-39 years) regularly used substances for self-harm(86). In our study, the most common route of ingestion is by oral route (99.1%). Among the intentions, suicidal intention (n=185, 78.4%) was more common than accidental (n=51, 21.6%).

A study by Indu et al. (2008-2013) including 1860 patients showed organophosphate as the most common poisoning with 461 patients (24.78%), followed by paraquat in 211 patients(11.34%), rat poison in 158 patients(8.49%), dye in 173 patients (9.29%), pyrethroid in 167 patients (8.98%), drugs in 125 patients(6.72%), unknown in 303 patients (16.29%), alcohol in 44 patients(2.37%), corrosives in 29 patients(1.56%), household cleaning agents in 93 patients(5%) and others in 96 patients(5.16%)(84).

Mittal et al. had a systematic review and meta-analysis from 2010 to 2020 in the Indian population that showed that pesticides are the most frequent kind of poisoning. Studies have found that limiting the use of particularly toxic pesticides significantly lowers the number of

fatalities(87).A study by Pannu et al. showed that about two-thirds of the poisoning cases included pesticides, with organophosphate (22.6%), aluminium phosphide (18.9%) paraquat (4.7%) being the most prevalent substances. A study by Tanuj et al. mentioned that organophosphates continue to be the most widely utilised agrochemicals in India. While the majority of males (82.2%) continued to choose organophosphates exclusively, females (58.3%) also preferred zinc phosphide, drugs (11.1%), and carbamates (5.6%) in addition to organophosphates(88). In our study also, organophosphate was the most common cause of poisoning in 55 patients (23.3%). Drugs were the second most common in 44 patients (18.6%), followed by detergents and corrosives in 38 patients (16.1%), aluminium phosphide in 24 patients (10.1%), unknown in 18 patients (7.6%), alcohol and cannabis in 10 patients each (4.2%), paint thinner in 9 patients (3.8%), petrol and diesel in 6 patients (2.5%), opium and kerosene in 5 patients each (2.1%) and others in 12 patients (5.04%).

In a study by Lalit et al., 21% of all poisonings were caused by prescription medicines. Paracetamol was the cause of nearly half of them (45.4%; 359/790). The other medications belonged to the therapeutic groups of analgesic, antipsychotic, anti-asthmatic, and antihypertensives(5). In our study, benzodiazepines were the most common drug of ingestion, followed by escitalopram, paracetamol, cyclopam, tramadol, amlodipine, cetirizine, trihexyphenidyl, methamphetamine, clonazepam, terbutaline, phenobarbitone, minoxidil, iron syrup.

In the study by Eyosias et al., the most common presenting symptom was diarrhoea and vomiting (49.5%), which was followed by altered consciousness (16.5%) and epigastric discomfort (13.6%). In our study, nausea and vomiting were the most common symptoms seen in 37.1% of patients, followed by altered sensorium in 17%, burning sensation in 6.3%, shortness of breath in 5%, drowsiness in 3.8%, hematemesis in 2.9%, headache and restlessness each in 1.7%, cough in 1.2%, chest pain and epigastric pain in 0.8%, local burns, giddiness, generalised weakness and agitation in 0.4% patients.

A study by Mehrpour et al. showed that most patients with poisoning presented to the emergency had a GCS score of less than 9(9). In the present study, most patients had normal GCS (n=195, 82.6%). The number of patients with GCS 13 to 14 was 21 (8.9%), 9 to 12 was 4 (1.7%), and 3 to 8 was 16 (6.8%).

A study by Pannu et al. showed that the length of stay of patients with poisoning in the hospital was two days. In their study, only 13.7% (n = 55) of those stayed for seven days

or longer(86). A study by M Islambulchilar et al. showed that the mean duration of stay in the hospital was 3.02 ± 2.8 days(89). In our study Median duration of stay was 48 (3-96) hours.

A study by Adinew et al. showed that most patients with poisoning received supportive care, whereas 27% and 24% of patients received gastrointestinal decontamination and particular antidotes. The most widely used remedies were atropine and activated charcoal. Among other forms of treatment, 3.0% (7 instances) got both stomach lavage and the administration of charcoal for decontamination, whereas 17.6% (130 patients) had intestinal lavage, and 6.4% were given activated charcoal (90). In our study, gastric lavage and activated charcoal were given to 68 and 44 patients, respectively. The maximum number of patients in which gastric lavage done was organophosphate poisoning (n=30), followed by aluminium phosphide. The maximum number of patients in which activated charcoal given was organophosphate poisoning (n=23), followed by aluminium phosphide(n=12). In our study, N-acetyl cysteine, magnesium sulphate, coconut oil and activated charcoal were provided to 22 (91.6%), 20 (83%), 15 (62%), and 12 (50%) patients, respectively, out of 24 cases of aluminium phosphide poisoning.

Research by Kumar et al. showed that patients with non-organophosphorus poisoning reported greater rates of multiorgan failure and death (11.8%)(86). According to a study by Mathai and Bhanu, patients with hemodynamic instability who presented late, had early signs of organ failure, had acidosis, and required vasoactive medications had a poor prognosis(4). Ahuja et al. showed that patients with high SOFA scores had greater mortality. In the same research, patients who required mechanical ventilation and vasoactive support also had a greater death rate(4). In the present study,14 out of 236 poisonings (5.9%) cases exhibited metabolic acidosis. They were primarily seen in aluminium phosphide poisoning (6 patients), followed by organophosphate poisoning (4 patients).A study by Rajbanshi et al. showed that 16.5% of survivors had acute renal failure(4).In our study, 8 out of 236 patients (3.4%) had renal dysfunction. Aluminium phosphide poisoning (n=4) had the maximum number of patients with renal dysfunction, followed by benzodiazepines (n=2).Due to its extensive usage and ease of availability, acetaminophen overdose continues to be the most significant cause of acute liver failure in North America, Europe, and Australia; however, our data showed that it is discovered seldom in reports from LMIC-like India(86). In our study, 8 (3.4%) out of 236 patients had liver dysfunction, and nine (3.1%) had coagulopathy. In the present study, 28 patients (17.7%) were admitted to ICU out of 158 patients (excluding those

who were referred, absconded, or LAMA). Organophosphate was the poisoning with maximum ICU admission (n=14, 50%), followed by aluminium phosphide (n=8, 28.5%).

As per the study by Rajbanshi et al., patients who had non-organophosphorus poisoning had 1.6 times more ICU mortality and shorter stay (P = 0.020). Age, GCS, SOFA score, length of hospital stays, the requirement for mechanical ventilation, abnormal coagulation profile, hepatic failure, and need for vasoactive medications were among the variables that statistically significantly affected the patients' ICU mortality. A study by Mathai et al. found that characteristics such as delayed presentation, early signs of organ failure, acidosis, and the requirement for vasoactive medications in patients with hemodynamic instability contributed to their poor outcome(91). In our study mortality rate was 6.3%. Aluminium phosphide has the highest mortality rate (29.4%), followed by organophosphate (12%). Median value of qSOFA was statistically significantly higher in non-survivor patients (p <0.001). Renal dysfunction (P= 0.047) and need of vasopressor support (P= <0.001) had statistically significant association with survival of the patient. Age, sex, liver dysfunction, and coagulopathy didn’t have a significant association with the mortality of the patient. We also compared clinical and outcome parameters of organophosphorus and non-organophosphorus poisoning and found no significant association except with qSOFA (p=0.023).

Study Strengths and limitations

This research will contribute to the generation of regional clinico-epidemiological data on poisoning. As a result, it might be helpful in planning the optimal use of available resources for the prevention and treatment of poisoning cases in western Rajasthan.

It did, however, have certain limitations.

1. The research was conducted in a single centre.

2. The epidemiological data do not incorporate socioeconomic condition, cultural and religious information, or occupational data, which might have given further insight about the clinical spectrum of poisoning.

3. Because our hospital is a tertiary care centre, individuals with poisoning might have appeared late after obtaining first aid at the local level, thus missing the precise identification of toxins.

4. We did not include patients with snake/unknown bites.

5. Multicentre studies with larger sample sizes are required in the future for generalizability of our results.

CONCLUSION

Acute poisoning is one of the primary reasons of hospitalization in emergency department. The current research revealed that it is prevalent among men, young individuals, literate and married people. Suicidal poisonings by oral route are more prevalent than accidental poisonings. Organophosphorus poisoning is one of the most common types of poisoning in Western Rajasthan, followed by ingestion of drugs. In our study, nausea and vomiting were the most common symptoms at presentation, followed by altered sensorium. In the present study, 3.4% of patients with poisoning had renal dysfunction, 3.3% had liver dysfunction, 3.1% had coagulopathy, 8.8% needed vasopressor support, and 17.7% required ICU admission. Aluminum phosphide was associated with the highest mortality rate, followed by organophosphate poisoning.

Recommendations

To decrease morbidity and mortality due to poisoning, we propose stringent enforcement of the pesticide act to monitor pesticide usage, which is the most prevalent poisoning in Western Rajasthan. To raise public awareness of various toxins, educational initiatives with a focus on prevention are required. A multicentric nationwide study might fulfill our study objectives more effectively.

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ANNEXURES

ANNEXURE – 1

IEC CERTIFICATE

अखिल भारतीय आयुर्विज्ञान संस्थान, जोधपुर
All India Institute of Medical Sciences, Jodhpur
संस्थागत नैतिकता समिति
Institutional Ethics Committee

No. AIIMS/IEC/2021/3534 **Date: 12/03/2021**

ETHICAL CLEARANCE CERTIFICATE

Certificate Reference Number: **AIIMS/IEC/2021/3369**

Project title: **"Clinico-epidemiological profile and outcome of patients with acute poisoning patients presenting to emergency of a tertiary care centre in Western Rajasthan"**

Nature of Project:	**Research Project Submitted for Expedited Review**
Submitted as:	**M.D. Dissertation**
Student Name:	**Dr. Akhil V George**
Guide:	**Dr. Ankur Sharma**
Co-Guide:	**Dr. Ashok Puranik, Dr. Mahaveer Rodha, Dr. Gopal Krishna Bohra & Dr. Bharat Chaudhary**

Institutional Ethics Committee after thorough consideration accorded its approval on above project.

The investigator may therefore commence the research from the date of this certificate, using the reference number indicated above.

Please note that the AIIMS IEC must be informed immediately of:

- Any material change in the conditions or undertakings mentioned in the document.
- Any material breaches of ethical undertakings or events that impact upon the ethical conduct of the research.

The Principal Investigator must report to the AIIMS IEC in the prescribed format, where applicable, bi-annually, and at the end of the project, in respect of ethical compliance.

AIIMS IEC retains the right to withdraw or amend this if:

- Any unethical principle or practices are revealed or suspected
- Relevant information has been withheld or misrepresented

AIIMS IEC shall have an access to any information or data at any time during the course or after completion of the project.

Please Note that this approval will be rectified whenever it is possible to hold a meeting in person of the Institutional Ethics Committee. It is possible that the PI may be asked to give more clarifications or the Institutional Ethics Committee may withhold the project. The Institutional Ethics Committee is adopting this procedure due to COVID-19 (Corona Virus) situation.

If the Institutional Ethics Committee does not get back to you, this means your project has been cleared by the IEC.

On behalf of Ethics Committee, I wish you success in your research.

Dr. Praveen Sharma
Member Secretary

Member secretary
Institutional Ethics Committee
AIIMS,Jodhpur

Basni Phase-2, Jodhpur, Rajasthan-342005; **Website:** www.aiimsjodhpur.edu.in; **Phone:** 0291-2740741 Extn. 3109
E-mail : ethicscommittee@aiimsjodhpur.edu.in; ethicscommitteeaiimsjdh@gmail.com

APPENDIX - 2

All India Institute of Medical Sciences Jodhpur, Rajasthan

Patient Informed Consent Form (PICF)

Title of the project: **"CLINICO-EPIDEMIOLOGICAL PROFILE AND OUTCOME OF PATIENTS WITH ACUTE POISONING PATIENTS PRESENTING TO EMERGENCY OF A TERITIARY CARE CENTRE IN WESTERN RAJASTHAN"**

Name of the PG Student: Dr. AKHIL V GEORGE

Tel. No.: 9633495444

Patient/Volunteer Identification No.: _____

I, _____ S/o or D/o _____

R/o

_____

give my full, free, voluntary consent to be a part of the study **"CLINICO-EPIDEMIOLOGICAL PROFILE AND OUTCOME OF PATIENTS WITH ACUTE POISONING PATIENTS PRESENTING TO EMERGENCY OF A TERITIARY CARE CENTRE IN WESTERN RAJASTHAN"** the procedure and nature of which has been explained to me in my own language to my full satisfaction. I confirm that I have had the opportunity to ask questions.

I understand that my participation is voluntary and am aware of my right to opt out of the study at any time without giving any reason.

I understand that the information collected about me and any of my medical records may be looked at by responsible individual from AIIMS, Jodhpur. I give permission for these individuals to have access to my records.

I agree to take part in the above study.

Date: _____

_____

Place: _____ Signature/Left thumb impression

This to certify that the above consent has been obtained in my presence.

APPENDIX - 3

ऑल इं डया इंस्टिट्यूट ऑफ मै डकल साईं सस जोधपुर, राजस

रोगी सू चत सहमति प्रपत्र

प्रोजेक्ट का शीर्षक: "पश्चिमी राजस्थान के तृतीयक स्वास्थय केंद्र के आपातकालीन वभाग में आने वाले आकस्मिक वषाक्तता रो गयों का नैदानिक-जनापदिक रोग वज्ञान वर्णन एवं उनके निष्कर्ष का अध्ययन "

प्रमुख अन्वेषक: डॉ अ खल व जोर्गे टेल. संख्या: 9633495444

रोगी पहचान सं. : _____

मैं, _____ पुत्र/,पुत्री _____

निवासी_____

"पश्चिमी राजस्थान के तृतीयक स्वास्थय केंद्र के आपातकालीन वभाग में आने वाले आकस्मिक वषाक्तता रो गयों का नैदानिक-जनापदिक रोग वज्ञान वर्णन एवं उनके निष्कर्ष का अध्ययन का हिस्सा बनने के लए मेरी पूर्ण, स्वतंत्र, स्वैच्छिक सहमति देता हूँ, जिसकी प्र क्रया और प्रकृति मुझे अपनी भाषा में अपनी पूर्ण संतुष्टि के लए समझाया गया है। मैं पुष्टि करता हूं क मुझे प्रश्न पूछने का अवसर मला है।

मैं समझता हूं क मेरी भागीदारी स्वैच्छिक है और कसी भी कारण के बिना कसी भी समय अध्ययन से बाहर निकलने के मेरे अ धकार से अवगत हूं।

मैं समझता हूं क मेरे और मेरे कसी भी मे डकल रिकॉर्ड के बारे में एकत्र की गई जानकारी एम्स, जोधपुर से जिम्मेदार व्यक्ति द्वारा देखी जा सकती है। मैं इन व्यक्तियों के लए अपने रिकॉर्ड तक पहुंचने की अनुमति देता हूं।

मैं उपरोक्त अध्ययन में भाग लेने के लए सहमत हूं।

तारीख: _____

_____

स्थान: _____ हस्ताक्षर / बाएं अंगूठे चह्न

यह प्रमा णत करने के लए क उपर्युक्त सहमति मेरी उपस्थिति में प्राप्त की गई है।

APPENDIX - 4

All India Institute of Medical Sciences Jodhpur, Rajasthan

PATIENT INFORMATION SHEET

Name of the patient: **Patient ID.:**

"CLINICO-EPIDEMIOLOGICAL PROFILE AND OUTCOME OF PATIENTS WITH ACUTE POISONING PATIENTS PRESENTING TO EMERGENCY OF A TERITIARY CARE CENTRE IN WESTERN RAJASTHAN"

1. You are participating a study to understand the various parameters associated with patients presented at emergency with poison exposure.

2. We will be collecting information regarding agent, quantity, route of exposure of poison local and systemic examination findings, and relevant investigations.

3. **Study procedure** : We will ask you to fill a proforma regarding the exposure to poisoning

4. **Likely benefit**: The current study will contribute substantial additional information regarding epidemiology and outcome of poisoning in tertiary hospitals.

5. **Confidentiality**: All the data collected from you will be kept highly confidential.

6. **Risk:** Enrolment in above study poses no substantial risk to you as all procedures performed are part of routine clinical care. You can withdraw from the study at any point of time without any consequences to yourself.

For further information / questions, the following personnel can be contacted

Dr. Akhil V George

Junior Resident, Department of Emergency Medicine, All India Institute of Medical Sciences, Jodhpur, Rajasthan.

Ph: 9633495444

APPENDIX - 5

ऑल इं डया इंस्टिट्यूट ऑफ मै डकल साईं सस जोधपुर, राजस्थान

रोगी सूचना पत्रक

मरीज का नाम रोगी पहचान सं :

"पश्चिमी राजस्थान के तृतीयक स्वास्थय केंद्र के आपातकालीन वभाग में आने वाले आकस्मिक वषाक्तता रो गयों का नैदानिक-जनापदिक रोग वज्ञान वर्णन एवं उनके निष्कर्ष का अध्ययन "

1. आप जहर खाने या उसके संपर्क में आने के बाद आपातकालीन स्थिति में आने वाले रो गयों से जुड़े व भन्न मापदंडों को समझने के लए एक अध्ययन में भाग ले रहे हैं।

2. हम जहर के एजेंट, मात्रा, र स्थानीय और प्रणालीगत परीक्षा के निष्कर्षों, जो खम के मार्ग और प्रासं गक जांच के बारे में जानकारी एकत्र करेंगे।

3. अध्ययन प्र क्रया :हम आपको वषाक्तता के संपर्क के बारे में एक प्रोफार्मा भरने के लए कहेंगे

4. संभा वत लाभ: वर्तमान अध्ययन तृतीयक अस्पतालों में वषाक्तता के परिणाम के बारे में अतिरिक्त जानकारी का योगदान देगा।

5. गोपनीयता: आपके द्वारा एकत्र कए गए सभी डेटा को अत्य धक गोपनीय रखा जाएगा।

6. जो खम**:** उपरोक्त अध्ययन में नामांकन आपके लए कोई महत्वपूर्ण जो खम नहीं है क्यों क सभी प्र क्रयाएँ निय मत नैदानिक देखभाल का हिस्सा हैं। आप बिना कसी परिणाम के कसी भी समय अध्ययन से पीछे हट सकते हैं।

अ धक जानकारी / प्रश्नों के लए, निम्न ल खत क र्मयों से संपर्क कया जा सकता है

डॉ। अ खल वी जॉर्ज**,** जूनियर रेजिडेंट, आपातकालीन च कत्सा वभाग, अ खल भारतीय आयु र्वज्ञान संस्थान, जोधपुर, राजस्थान। Ph: 9633495444

PROFORMA

Name of the patient: Date:

Age: Sex: REG. ID:

Address:

Education:

Marital Status: Occupation:

Mobile No:

Urban or Rural:

Premorbid conditions:

Psychiatric illness-

GCS at presentation:

Sucidal/Homicidal/Accidental:

Type of Poison:

Quantity of poison:

Route of exposure:

Intention, if voluntary:

Time of Exposure:

Management given at home:

Treatment given by previously visited hospital, if any:

Transport facility used:

Time of Presentation:

Reason for delay, if any:

If bite/prick, known /unknown:

If known, source:

Clinical presentation:

Local Examination:

Vitals:

qSOFA score:

Treatment given:

Duration of mechanical ventilation:

Vasopressors given:

ICU admission, yes or no:

If yes, management given:

Duration of stay:

Investigations

RBS:

Blood Gas Analysis:

CBC:

KFT:

Serum Electrolytes:

LFT:

Coagulation Profile:

Samples Collected and Findings:

Akhil Thesis Plag Check

ORIGINALITY REPORT

9%
SIMILARITY INDEX

PRIMARY SOURCES

1	Mahendra Lodha, Darshan Patel, Mayank Badkur, Satya Prakash Meena et al. "Assessment of Quality of Life After Ventral Hernia Repair: A Prospective Observational Study at a Tertiary Care Centre", Cureus, 2022 Crossref	63 words — 1%
2	Bhoopendra Singh, B. Unnikrishnan. "A profile of acute poisoning at Mangalore (South India)", Journal of Clinical Forensic Medicine, 2006 Crossref	55 words — 1%
3	Eda Süzen-Orhan, Edin Botan, Emrah Gün, Hasan Özen et al. "Colchicine Poisoning Cases in a Pediatric Intensive Care Unit", Pediatric Emergency Care, 2022 Crossref	36 words — < 1%
4	"61st Annual Conference of Indian Society of Hematology & Blood Transfusion (ISHBT) November 2020", Indian Journal of Hematology and Blood Transfusion, 2020 Crossref	34 words — < 1%
5	"Posters", Clinical Microbiology and Infection, 4/2007 Crossref	32 words — < 1%
6	www.science.gov Internet	26 words — < 1%