

A dissertation on
**A STUDY TITLED "CLINICAL PROFILE OF RODENTICIDE
POISONING AND ITS OUTCOME IN A TERTIARY CARE
CENTRE"**

Submitted in partial fulfilment of requirements for

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MADRAS MEDICAL COLLEGE

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MAY 2020

CERTIFICATE

This is to certify that the dissertation entitled “**CLINICAL PROFILE OF RODENTICIDE POISONING AND ITS OUTCOME IN A TERTIARY CARE CENTRE**” is a bonafide work done by **Dr.ARVIND KUMAR.V**, at Madras Medical College, Chennai in partial fulfilment of the university rules and regulations for award of M.D., Degree in General Medicine (Branch-I) under our guidance and supervision during the academic year 2017-2020.

Prof. Dr. T.S. SHANTHI M.D.,
Guide & Research Supervisor,
Institute of Internal Medicine,
MMC &RGGGH,
Chennai - 3.

Prof. Dr. S. RAGUNANTHAN, M.D.,
Director (I/c) and Professor,
Institute of Internal Medicine,
MMC &RGGGH,
Chennai - 3.

Prof.Dr. R. JAYANTHI M.D., FRCP (Glasg)
The Dean
MMC & RGGGH,
Chennai – 03

DECLARATION

I, **Dr. ARVIND KUMAR.V**, Register No: **201711003** solemnly declare that this dissertation entitled “**CLINICAL PROFILE OF RODENTICIDE POISONING AND ITS OUTCOME IN A TERTIARY CARE CENTRE**” was done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai during 2017-2020 under the guidance and supervision of my Chief Prof. **Dr. T.S. SHANTHI M.D.** This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree in General Medicine (Branch-I).

Place: Chennai-3

Signature of Candidate

Date:

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Lastly, I thank all my professional colleagues for their support and valuable criticism.

LIST OF ABBREVIATIONS

AKI – Acute Kidney Injury

ALT – Alanine Transaminase

ALF – Acute Liver Failure

aPTT – Activated Partial Thromboplastin Time

ARDS – Acute Respiratory Distress Syndrome

AST -Aspartate Transaminase

BP – Blood Pressure

D.B – Direct Bilirubin

FFP – Fresh Frozen Plasma

GCS – Glasgow Coma Scale

INR – International Normalized Ratio

LFT – Liver Function Test

MELD – Model for End-stage Liver Disease

NAC – N Acetyl cysteine

PR – Pulse Rate

PSS – Poison Severity Score

PT – Prothrombin Time

RBS – Random Blood Sugar

RFT – Renal Function Test

T.B – Total Bilirubin

TABLE OF CONTENTS

S.NO	TITLE	PAGE NO
1	INTRODUCTION	8
2	REVIEW OF LITERATURE	9
3	AIMS AND OBJECTIVES	36
4	MATERIALS AND METHODS	37
5	RESULTS AND OBSERVATIONS	39
6	DISCUSSION	71
7	CONCLUSION	82
8	LIMITATIONS OF STUDY	84
9	BIBLIOGRAPHY	85
	ANNEXE Proforma Ethical Committee Approval Plagiarism Screenshot Plagiarism Certificate Information Sheet Consent form Master Chart	

INTRODUCTION

Poisoning is one of the frequent causes of admission in emergency department. Diseases borne due to rodents are an important public health issue e.g. leptospirosis, in a country like India. Therefore, the need for rodenticide has been commercialized and various types and forms of rodenticide is being sold.

These are manufactured by companies which sell them in packets containing information about the contents and warning signs in case of accidental exposure to humans. On the other hand, they are also made locally without information regarding contents

Toxicity ranges from asymptomatic patients to death with complications like Acute Liver Failure, Hepatic Encephalopathy, Bleeding Manifestations due to Coagulopathy, Acute Respiratory Distress Syndrome, Acute kidney injury etc.

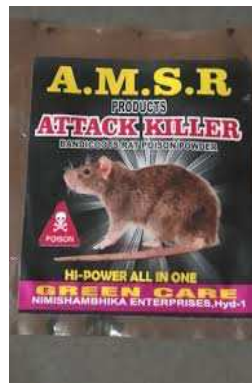
There has been previous studies and reports regarding the magnitude of the effects of different rodenticides in humans. This study is about Clinical profile of Rodenticide poisoning in humans and their Outcome in a tertiary care center.

REVIEW OF LITERATURE

Rodenticide is a chemical which kills rodents like rat, squirrels, mice and other small rodents. An ideal rodenticide kills the rodents effectively and is not toxic to humans and pets, when accidentally exposed to them. Such an ideal rodenticide is yet to be identified. Various rodenticide differs from each other in composition, mechanism of action, lethal dose, and toxicity spectrum.

Figure 1. Commercially available rodenticide in India

Locally Made



Company Made



There are various forms of rodenticide available namely:

1. Paste
2. Cake
3. Powder
4. Pellet

Rodenticides like warfarin's and super warfarin's are commonly available in cake forms. They usually cause major toxicity if taken chronically multiple times. Acute toxicity is seen in metal phosphides, yellow and white phosphorous, Thallium. It is either due to suicidal intention or Accidental. Accidental poisoning is most common in pediatric age group⁽¹⁸⁾.

Table 1: CLASSIFICATION OF RODENTICIDES⁽¹⁹⁾

S.No	Inorganic Compounds	Organic Compounds
1	Arsenic	Sodium Monofluoroacetate
2	Thallium	Alpha Naphthyl Thiourea
3	Phosphorus	Warfarin
4	Barium	Strychnine
5	Zinc	Norbormide
6		Vacor
7		Scilliroside

Other different ways of classification are based on:

1. By animal activity
2. By nature and onset of their symptoms
3. Based on their Lethal dose in rats.

Rat killer Cake:

They are usually synonymous with warfarin and superwarfarins. There are two groups of anticoagulants 1) Hydroxy Coumarin 2) Indanediones. Warfarin were initially marketed as rodenticide and later used for therapeutic purpose⁽²¹⁾.

Table 2: Types of Anticoagulants⁽²⁰⁾

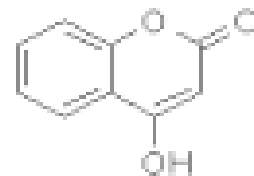
S.No	HYDROXYCOUMARINS	INDANEDIONES
1	Warfarin	Chlorphacinine
2	Defenacoum	Pindone
3	Panwarfarin	Pivalyn
4	Warficide	Diphacinone
5	Coumachlor	Phenindione
6	Oumafuryl	Asinindione
7	Prolin	Brodifacoum

Coumarin consists of benzene ring, ester and alkene. Indanediones has a chemical formula of $C_9H_6O_2$

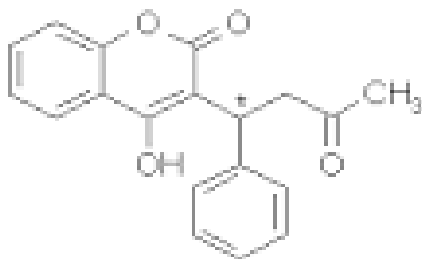
Figure 2: Structure of Coumarin and Indanediones



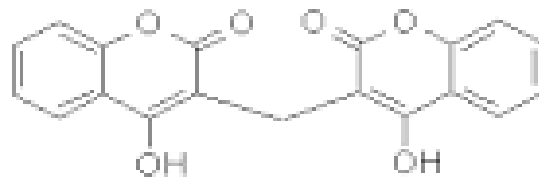
coumarin



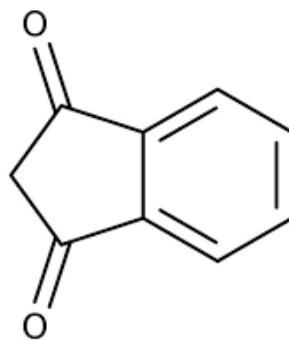
4-hydroxycoumarin



warfarin
* chiral center



bishydroxycoumarin
(dicoumarol)



Indanedione

Superwarfarins were developed to overcome the resistance to warfarin in rats ⁽¹⁾. They are long acting, lipid soluble chemical. Half life ranges from weeks to months with an average of 24 days ⁽²⁾

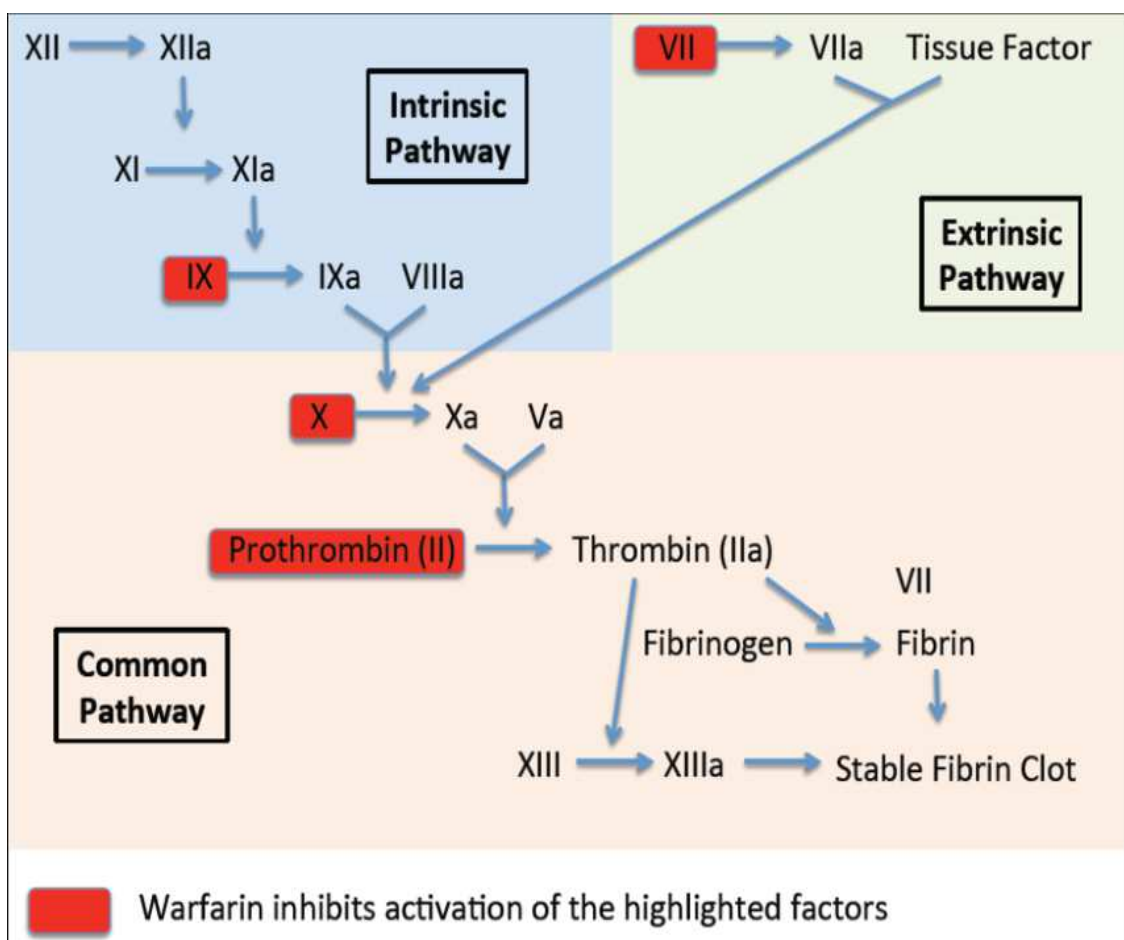
Figure 3: Commonly available Rat Killer Cake



Mechanism of Action:

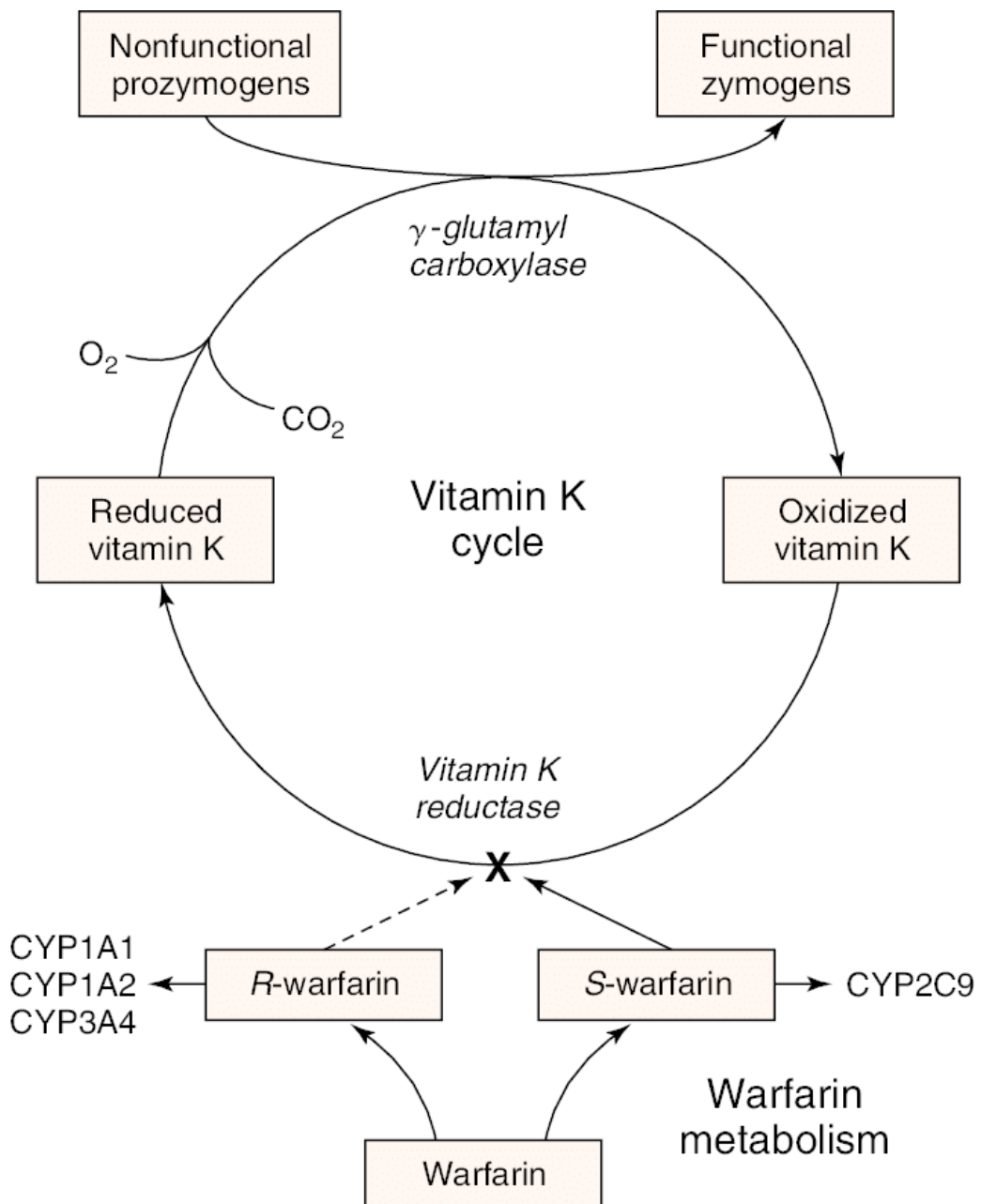
Inhibition of vitamin k dependent enzymes is major site of action. Vitamin K is a vital cofactor for forming functional clotting factor II, VII, IX, X IN liver. Gamma carboxylation of these factors enable them to get attached to calcium therefore rendering them functional.

Figure 4: Coagulation Cascade Inhibition site by Warfarin



Reduced vitamin k is essential for gamma carboxylation of precursor factor, simultaneously to carboxylation vitamin k is oxidized by epoxide enzyme.

Figure 5: Mechanism of action



Pharmacology:

Warfarin and superwarfarins are completely absorbed in gastrointestinal tract. New active clotting factor production is immediately stopped but preformed factor is circulating. Half-life of circulating coagulation factors inhibited by warfarin are

factor II-60 hours, VII- 6 hours, IX- 24 hours, X- 72 hours. Hence effect of warfarin takes around 48 to 60 hours to reflect in prothrombin time and international normalized ratio. To prolong international normalized ratio, clotting factor levels should go below 25% of their normal value.

Toxicity:

- Ingestion of small quantity does not cause any serious side effects.
- Chronic repeated ingestions can cause increased risk of bleeding manifestation especially if they have previous liver damage or chronic malnutrition.

Clinical features:

Most of the clinical features of superwarfarins are due to reduced tendency to clot. There can be local irritation of gastrointestinal system causing vomiting. Minor bleeding like subconjunctival hemorrhage, petechiae, purpura, ecchymosis can be seen. Major Bleeding manifestation include gastrointestinal bleed. But intracranial bleed has not been documented in literature till date.

Diagnosis:

-Specific levels of the suspected compound in blood.

-Baseline and frequent measurement from every 6 hours of prothrombin time and international normalized ratio.

-If after 2 days there is no raise in prothrombin time, then significant level of toxin ingestion can be excluded.

- Measuring clotting factors is not necessary.

Management:**A) Gastric Lavage:**

It should be done within 1 hour of ingestion, beyond 2 hours there is no significant change in outcome to the patient

B) Activated Charcoal:

Multidose activated charcoal should be given within 1 hour of ingestion after gastric lavage. The dose to be given is 1g/kg body weight.

C) Vitamin K₁ (Phytonadione):

- It is a specific antidote for warfarin toxicity.
- It should be given only if prothrombin time is increased.
- If given prophylactically, prothrombin time at the end of 48 hours could not be considered as non-toxic dose ingestion if found normal.

- Dose to be given is 50mg / dose to 600mg/day every 6 hours, preferred route is subcutaneous more than intramuscular.
- Prothrombin concentrate or Fresh frozen plasma or whole blood may be needed if there is active bleeding
- Patient may need prolonged duration of vitamin k supplementation if superwarfarins effect last for months⁽³⁾.



Figure 6: Yellow phosphorus

Yellow Phosphorus:

- It is one of the rodenticides with worse outcome compared to others.
 - It is commonly available in paste form and the frequent manufacturer product brought to hospital in India is Ratol.

-This is a highly cellular toxin which are still used in firework, fertilizer manufacturing along with rodenticides.

- It has elemental phosphorus which is highly toxic.
- Since it is in paste form, accidental ingestion in children is seen predominantly.
- It has corrosive properties.

Stages of Toxicity:

1st Stage:

- This stage is between ingestion of the poison and within 24 hours.
- It is mostly asymptomatic
 - If symptoms are present, it is mostly due to local gastrointestinal tract irritation with features like vomiting and abdominal pain.

2nd Stage:

- This stage lasts between 24 hours to 72 hours
- This stage to for the most part asymptomatic
- There can be mild increase in bilirubin, aspartate and alanine transaminase

3rd Stage:

- It constitutes duration greater than 72 hours till resolution or death
- There can be hepatomegaly and jaundice.
- Acute fulminant liver failure is a major complication

- Other complications include bleeding tendencies due to increased prothrombin time and/ thrombocytopenia
- Acute tubular necrosis can also occur though a rare complication.
- Hepatic encephalopathy occurs.

Lethal dose:

Dose varies depending on the route of absorption of the toxin as penetration into systemic circulation can occur due to ingestion, inhalation and through skin.

LD 50 through ingestion - 1mg/kg⁽⁴⁾

LD 50 through inhalation – 5 mg/kg.

Clinical Features^(5,6):

1)Inhalation:

-It causes local irritation leading to conjunctivitis, mucus membrane necrosis

- Wheezing and chemical pneumonitis if the toxin reaches the lower respiratory airways.

-If the exposure is high may cause non cardiogenic pulmonary edema

- Phossy jaw occurs if the patient has chronic exposure to yellow phosphorus.

2)Dermal:

- Chemical burns on areas of exposure may even lead to necrosis

3)Ingestion:

- Local irritation cause vomiting and abdominal pain.
- Cardiovascular toxicity causes arrhythmia like ventricular tachycardia, ventricular fibrillation, atrial fibrillation etc., shock.
- Hepatotoxicity causes jaundice, hepaticencephalopathy, coma and seizures.
- Bleeding manifestation due to abnormal coagulation profile orthrombocytopenia.
- Renal failure due to acute tubular necrosis.

4) Metabolic derangements:

- Hypocalcemia
- Hyperphosphatemia

Sometime spontaneous recovery occurs without any possible explanations⁽⁷⁾.

Management:

1) Decontamination:

- Removal of clothing
- Washing exposed area with soap and water
- Irrigate exposed eyes with water
- Cover exposed area to prevent spontaneous combustion.

2)Supportive Measures:

- Initial survey and managing the airway, breathing and circulation.

- IV fluids may be necessary for hypotension due to persistent vomiting due to gastrointestinal symptoms.

3) Gastric Lavage:

- Lavage should be given within one hour of ingestion with potassium permanganate 0.1% to convert the phosphorus into a harmless oxide.

4) Activated Charcoal:

- Should be given after gastric lavage at a dose of 1mg/kg body weight.

- No clear advantage in giving MDAC after 2 hours of ingestion.

Patient should avoid fatty diet as it increases phosphorus absorption.

5) For Acute liver failure^(23,24,25,26):

- Monitoring of liver function and renal function test periodically.

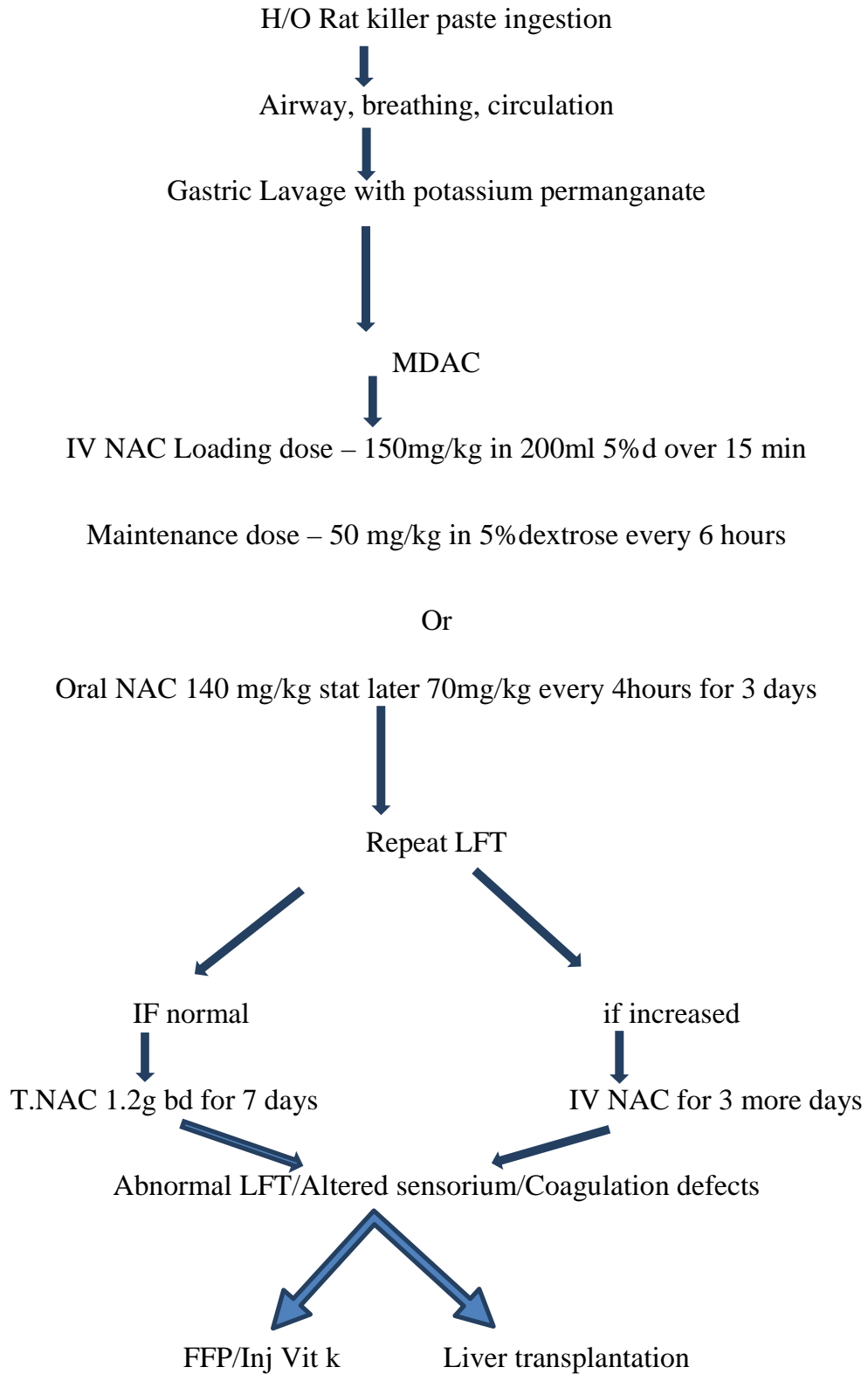
- IV N acetyl cysteine is of significant importance in management of yellow phosphorus poisoning

- N acetyl cysteine is available in intravenous and oral forms

-N acetyl cysteine scavenges free radicals and replenishes mitochondrial and cytosols glutathione reserves, therefore preventing damage to liver.

-It has been proven NAC is of positive therapeutic significance in acetaminophen related acute liver failure but significance in non-acetaminophen related acute liver failure is yet to be proven beyond doubt even though studies suggest usefulness.

Management Protocol⁽¹⁹⁾:



Liver Transplant:

-Liver transplantation is the replacement of native and diseased liver by a normal organ.

- The most recent and advanced procedure is orthotopic transplantation, where the native organ is being removed and the donor organ is replaced in the same anatomic location.

- Success rate as expressed in 1-year survival has improved from around 30% in the 1970s and it is more than 90% nowadays

- These longer survival rates are due to improving operative techniques, better organ procurement and preservation, breakthrough in immunosuppressive therapy, and careful patient selection and timing of surgery.

-Timing of the operation is of critical importance.

Indication for Liver Transplant

- Primary biliary cirrhosis
- Secondary biliary cirrhosis
- Primary sclerosing cholangitis
- Autoimmune hepatitis
- Caroli's disease
- Cryptogenic cirrhosis
- Chronic hepatitis with cirrhosis

- Hepatic vein thrombosis
- Fulminant hepatitis
- Alcoholic cirrhosis
- Chronic viral hepatitis
- Primary hepatocellular malignancies
- Hepatic adenomas
- Non-alcoholic steatohepatitis
- Familial amyloid polyneuropathy

Liver Transplant Criteria:

Criteria of King's College, London ⁽²⁷⁾

Acetaminophen Cases

-Arterial pH < 7.25 more than 24 hours after drug ingestion

All of the following:

-Prothrombin time > 100 sec or INR > 6.5

-Serum creatinine level > 3.4 mg/dL (300 µmol/L) or anuria

-Grade 3 to 4 encephalopathy

Non -acetaminophen Cases

-Prothrombin time > 100 sec or INR > 6.7

Any 3 of the following:

-Unfavorable etiology (seronegative hepatitis or drug reaction)

-Age < 10 or > 40 years

-Acute or subacute category (duration of jaundice > 7 days)

-Serum bilirubin level > 17.5 mg/dL (300 μ mol/L)

-Prothrombin time > 50 sec or INR > 3.5

Complications of Liver Transplantation:

- Prehepatic:
 - Pigment load
 - Haemolysis may occur
 - Blood collections (hematomas, abdominal collections)
- Intrahepatic:
 - Early
 - Hepatotoxic drugs and anaesthesia
 - Hypoperfusion (hypotension, shock, sepsis)
 - Benign postoperative cholestasis

- Late
- Transfusion-associated hepatitis
- Exacerbation of primary hepatic disease
- Post hepatic
- Biliary obstruction
- Reduced Renal clearance of conjugated bilirubin (renal dysfunction)
- Primary graft may not function after transplant
- Portal vein obstruction may occur
- Hepatic artery can be thrombosis
- Anastomosis can get leaked with can lead to intraabdominal bleeding
- Bile duct can become stenosed, obstructed or leak
- Transplant Rejection
- Recurrence of primary hepatic disease

Zinc Phosphide:

- They are grey crystalline powder and are not water soluble
- It has a characteristic Rotten fish odor⁽⁹⁾
- It is available in different composition and a minimum of 32% zinc phosphide⁽¹¹⁾

Mechanism of action:

-On exposure to water or stomach hydrochloric acid phosphine gas is produced.

-But delay in development of systemic toxicity has led to alternate mechanism like production of phosphonium as an intermediate product and get absorbed through stomach and later gets converted into phosphine in particular organs.

- Phosphine inhibits cytochrome c oxidase resulting in renal and liver failure⁽¹⁰⁾

- Oxygen uptake in liver mitochondria is affected^(12,13)

- It also has an anticholinesterase effects and can also cause denaturation of oxyhemoglobin molecules⁽¹⁴⁾

Fatal dose⁽¹⁰⁾:

It is around 40 mg/kg bodyweight.

Clinical Features:

-Gastrointestinal symptoms like vomiting and abdominal pain

-Cardiovascular – shock, arrhythmias

- Hepatobiliary symptoms -Acute liver failure,encephalopathy,bleeding

- CNS manifestations like altered sensorium, seizure and coma

Treatment:

- There is no specific antidote for zinc phosphide poisoning.
- Symptomatic treatment is the mainstay of management
- Airway, breathing and circulation is secured
- Test for phosphine gas detection⁽¹⁵⁾:

5 ml of lavage fluid is mixed with 15 ml of water and heated for about 20 minutes at 50⁰ Celsius.

Two filter papers coated with silver nitrate and lead acetate separately is kept in the mouth of container.

	Silver Nitrate	Lead acetate
Phosphine gas	Turns Black	No change
Hydrogen Sulphide	Turns Black	Turns Black

- Magnesium sulphate⁽¹⁶⁾:

The exact mechanism of action in zinc phosphide poisoning is not known. It is hypothesized that magnesium stabilizes cell membranes and prevents arrhythmias.

There has been reduction in mortality rate and fewer complications.

-Liver function and renal function has to be monitored frequently

-Vitamin k1 supplements should be given if prothrombin time increases⁽¹⁷⁾.

MELD Score⁽³⁶⁾:

-The Model for End-stage Liver Disease was firstformed to predict the survival in patients who undergoes elective procedures like placementoftransjugular intrahepatic portosystemic shunts which is a complication of portal hypertension.

- MELD using only objective variables was validated later as an accurate predictor of survival among various populations of patients with advanced liver disease.

- The primary use of the MELD score has been the allocation of organs for liver transplantation. But the MELD score has been shown to predict survival in patients with cirrhosis in whomcomplications like infections, variceal bleeding, patients with fulminant hepatic failure and alcoholic hepatitis.

- MELD can also be used in selection of patients for surgery other than liver transplant and also in determining appropriate treatment for patients with HCC (hepatocellular carcinoma) who are not candidates for liver transplantation.

- Approximately around 15% to 20% of patient's survival cannot be predicted accurately by the MELD score.

MELD Formula:

Parameters Includes

1. Serum Creatinine[mg/dl]

2. PT/INR

3. Serum Bilirubin[mg/dl]

$$\text{MELD}(i) = 0.957 \times \log(\text{Cr}) + 0.378 \times \log(\text{bilirubin}) + 1.120 \times \log(\text{INR}) + 0.643$$

Laboratory values less than 1.0 will be set to 1.0 when calculating a candidate's MELD score

For candidates with an initial MELD score greater than 11, the MELD score is then

re-calculated as follows:

$$\text{MELD} = \text{MELD}(i) + 1.32 \times (137 - \text{Na}) - [0.033 \times \text{MELD}(i) \times (137 - \text{Na})]$$

The following candidates will receive a creatinine value of 4.0 mg/dL:

- Candidates with a creatinine value greater than 4.0 mg/dL
- Candidates who received two or more dialysis treatments within the prior 7 days

- Candidates who received 24 hours of continuous veno-venous hemodialysis (CVVHD) within the prior 7 days.

Rounded to the tenth decimal place and then multiplied by 10

Maximum Meld score=40

Poison Severity Score ⁽³⁷⁾:

-Toxicology research lacks a universal and well accepted method to assess the severity of poisoning.

the PSS was developed as a tool to document encounters with poisoned patients

-A standardized scale to grade the severity of poisoning allows qualitative evaluation of morbidity due to poisoning and also for comparability of data.

- PSS is a classification scheme for poisoning cases in adults and children.

- PSS can be used for the classification of acute poisonings regardless of the type and the number of agents involved.

-Due to limitations a modified scheme in future may be required for certain poisonings, but this scheme may serve as a model for them.

- PSS should take into account overall clinical course and then be applied according to the most severe symptoms (including subjective symptoms as well as objective signs).

- Hence it is usually a retrospective process which requires follow-up of cases.

- Timing when the score is must be clearly stated when the data is being presented.

- The presence of a particular symptom is checked in the chart and the severity grading is assigned based on the most severe symptom(s) and or sign(s) observed.

- Severity grading should not consider the risks based on parameters like amount ingested or serum/plasma concentration of the poison.

- Prophylactic use of antidotes should not influence the grading, but should be mentioned when data is being collected

- Death is being given a separate grade

Severity Grades:

- NONE (0): No symptoms or signs related to poisoning
- MINOR (1): Mild, transient and spontaneously resolving symptoms
- MODERATE (2): Pronounced or prolonged symptoms

- SEVERE (3): Severe or life-threatening symptoms
- FATAL (4): Death

Hepatic Encephalopathy:

- Portosystemic encephalopathy is a major complication

defined as an alteration in mental status and cognitive function which occurs in the presence of liver failure.

- Development of encephalopathy is a requirement in acute liver injury for a diagnosis of fulminant failure.

- Encephalopathy is more common in patients with chronic liver disease.

- Liver fails to remove the gut-derived neurotoxins because of vascular shunting and decreased functional liver mass, which enters the brain and cause symptoms which duped as hepatic encephalopathy.

- Ammonia levels are typically elevated in patients with hepatic encephalopathy, but severity of liver failure cannot be quantified with ammonia levels.

- Other compounds and metabolites may contribute to the development of encephalopathy which include false neurotransmitters and mercaptans.

AIMS/ OBJECTIVES

- 1) To study various types of rodenticide poisoning admitted in RGGGH and to compare clinical profile and biochemical outcomes among them.
- 2) Descriptive analysis of rodenticide poisoning based on
 - a) Age
 - b) Sex
 - c) Marital status
 - d) Mode of poisoning
 - e) Type of poisoning
 - f) Amount of poison consumed
 - g) Time from consumption to admission
 - h) Clinical features of each type of poison
 - i) Outcomes of all the types of poison.

MATERIALS AND METHODS

Place of study:

They are carried out in toxicology ward and general ward of Institute of Internal Medicine, RGGGH, Chennai.

Study Duration:

One-year duration from 1st April 2018 to 31st March 2019

Study Design:

Observational Study

Ethical Committee Approval:

Approval has been obtained to conduct this study

Inclusion Criteria:

- Age > 12 years
- All patients with Clinical features or History of Rodenticide poisoning as per ICD 10 T60.4⁽²⁸⁾
- Patient willing for study
- If patient has altered mental status or unconscious, consent from closest kin can be included.

Exclusion Criteria:

- Patient not willing for study
- Patient <18 years of age
- If rodenticide mixed with other poisons/Toxic substance including but not limited to alcohol
- If patient has preexisting liver pathology which includes acute/chronic hepatitis B/C infection

RESULTS AND OBSERVATION

Table 3: Age Distribution

Age group	No of Patients	%
12 to 18 Years	35	17.2
19-28 Years	98	48.0
29-39 Years	43	21.1
40-49 Years	19	9.3
50& Above	9	4.4
Total	204	100.0

Figure 7: Age distribution

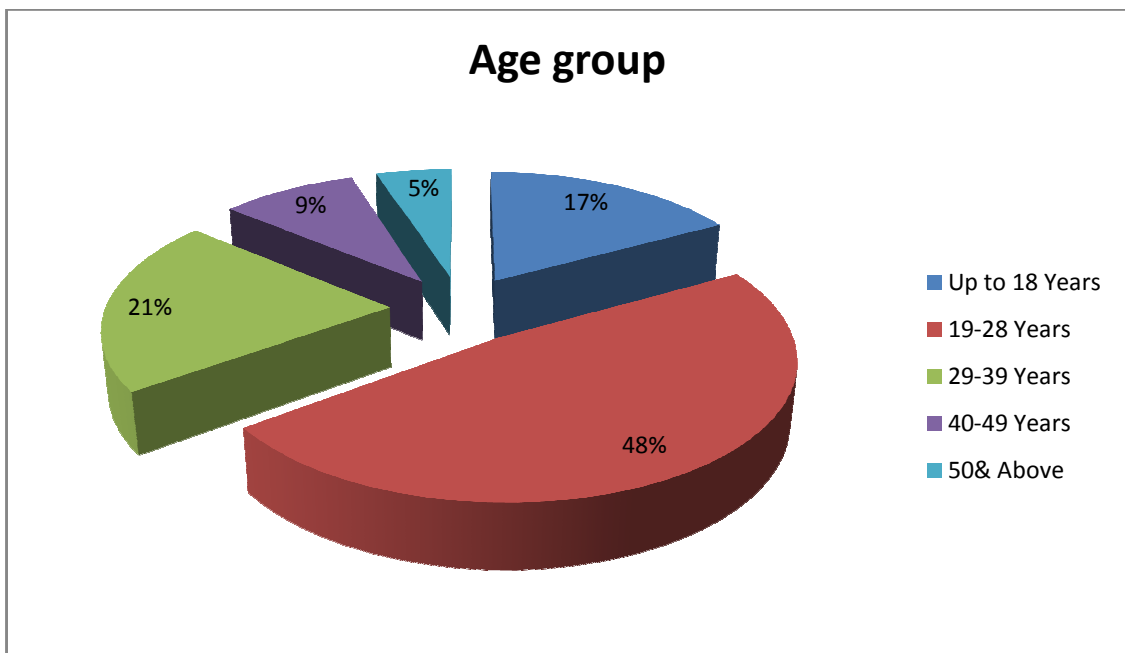


Table 4: Sex Distribution

Sex	No of Patients	%
Male	95	46.6
Female	109	53.4
Total	204	100.0

Figure 8: Sex Distribution

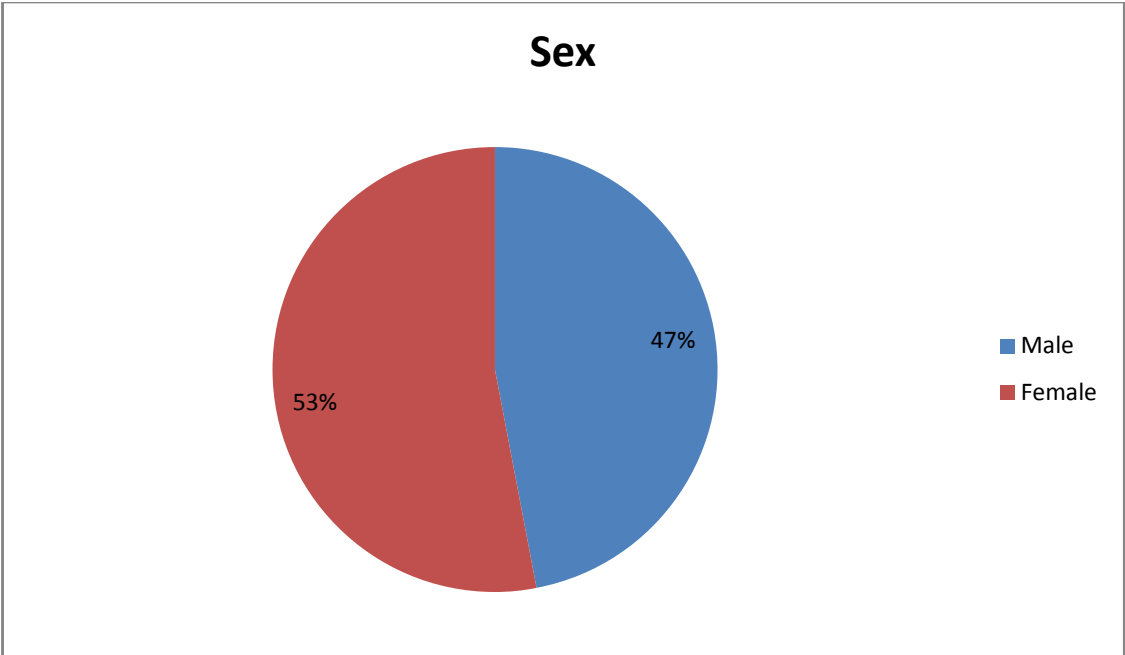


Table 5: Distribution based on Marital Status

Marital status	No of Patients	%
Unmarried	73	35.8
Married	131	64.2
Total	204	100.0

Figure 9: Distribution based on Marital Status

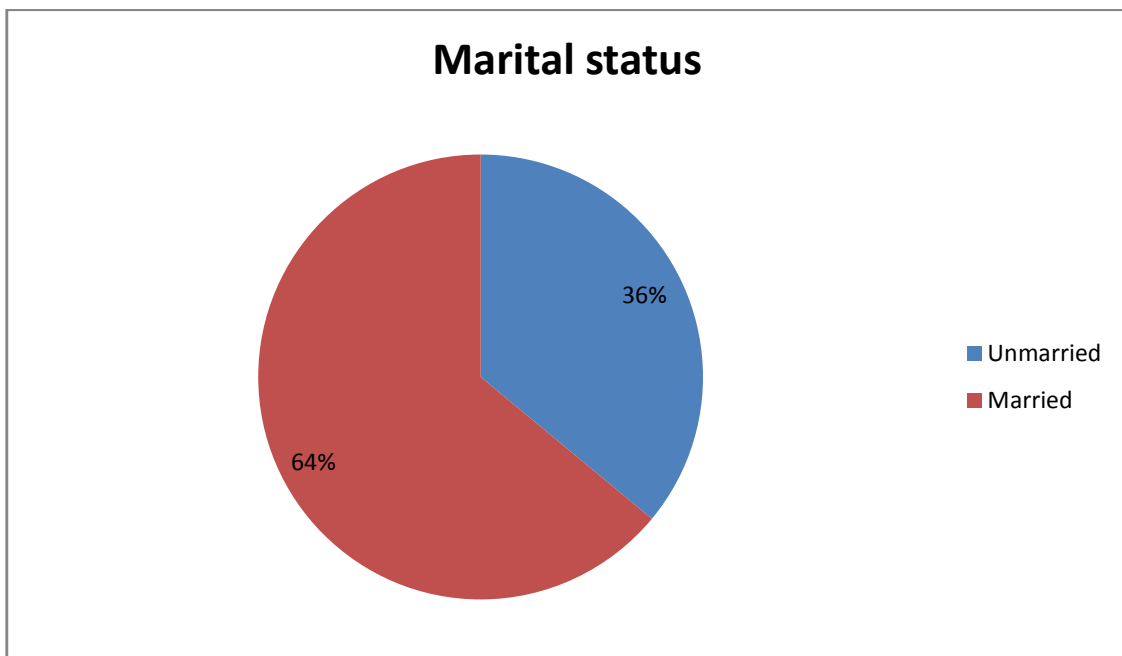


Table 6: Time Interval between Poisoning and Admission

Time to admission	No of Patients	%
<6 Hours	49	24.0
6-24 Hours	88	43.1
>24 Hours	67	32.8
Total	204	100.0

Figure 10: Time Interval between Poisoning and Admission

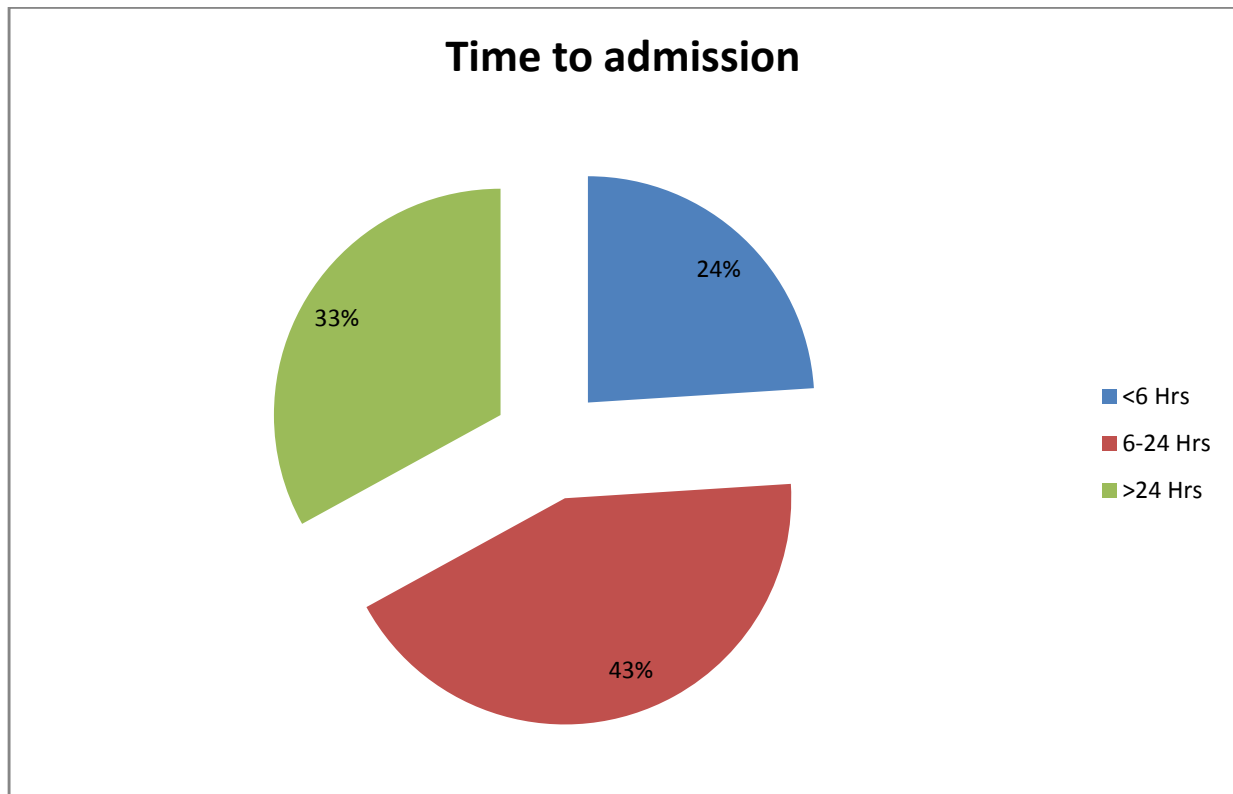


Table 7: Mode of Poisoning

Mode	No of Patients	%
Accident	1	.5
Suicide	203	99.5
Total	204	100.0

Figure 11: Mode of Poisoning

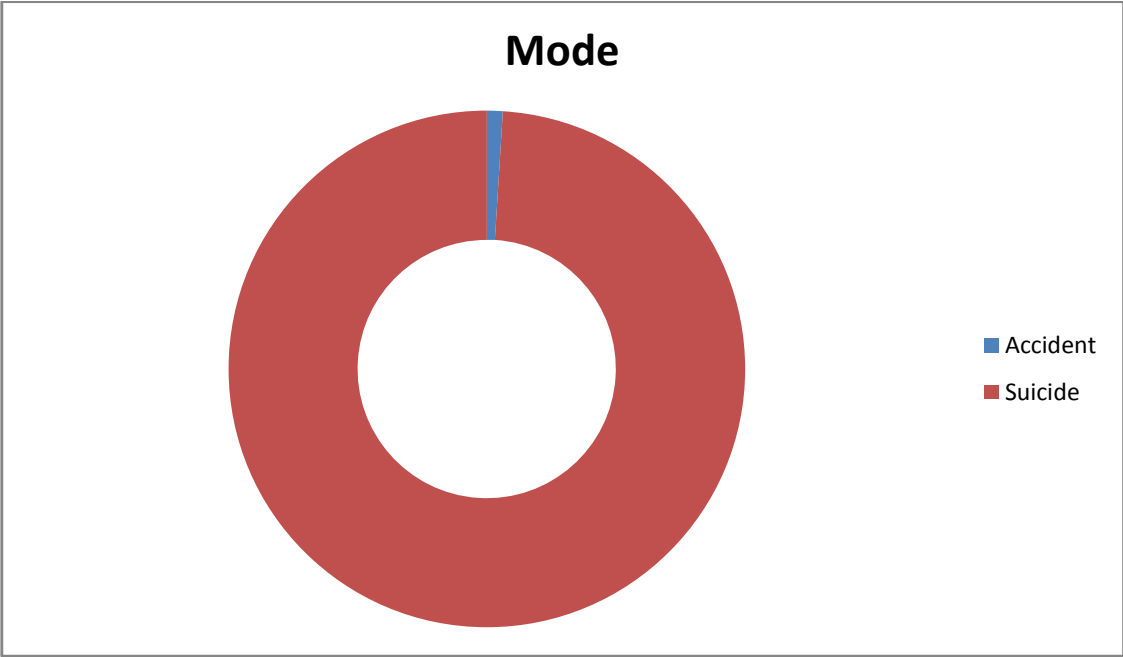


Table 8: DISTRIBUTION AMONG TYPES OF POISON

Type	No of Patients	%
YELLOW PHOSPHOROUS	111	54.4
SUPER WARFARIN	37	18.1
ZINC PHOSPHIDE	56	27.5
Total	204	100.0

Figure 12: DISTRIBUTION AMONG TYPES OF POISON

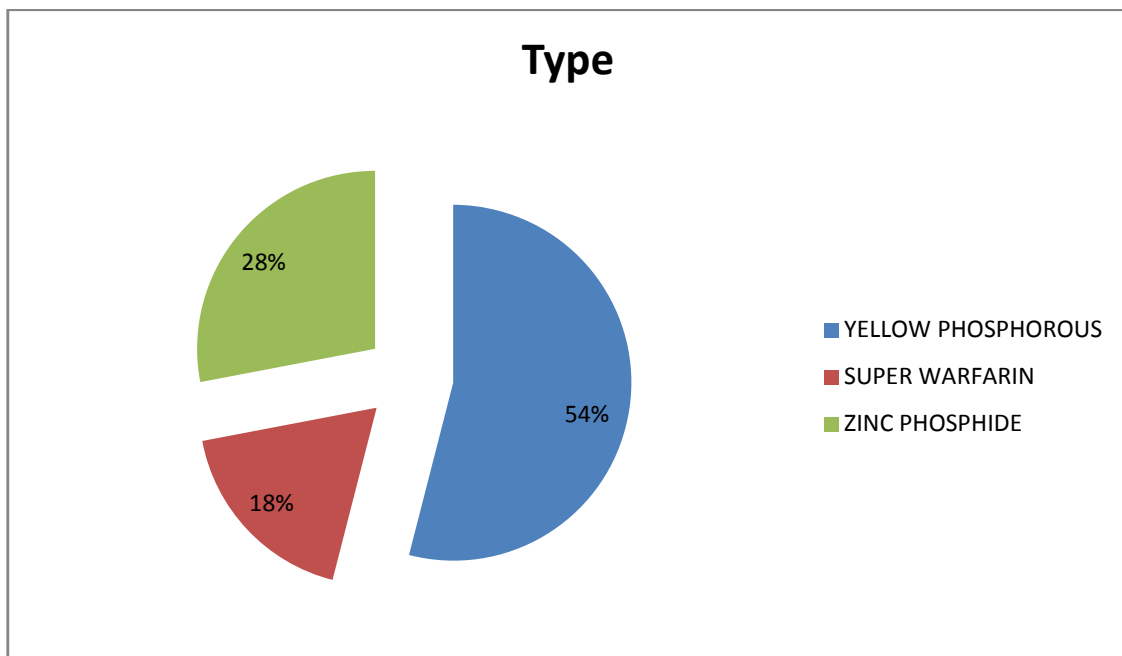


Table 9: Amount of Poison Consumed

Amount	No of Patients	%
<5 grams	104	51.0
5 to 10 grams	60	29.4
11 to 25 grams	40	19.6
Total	204	100.0

Figure 13: Amount of Poison Consumed

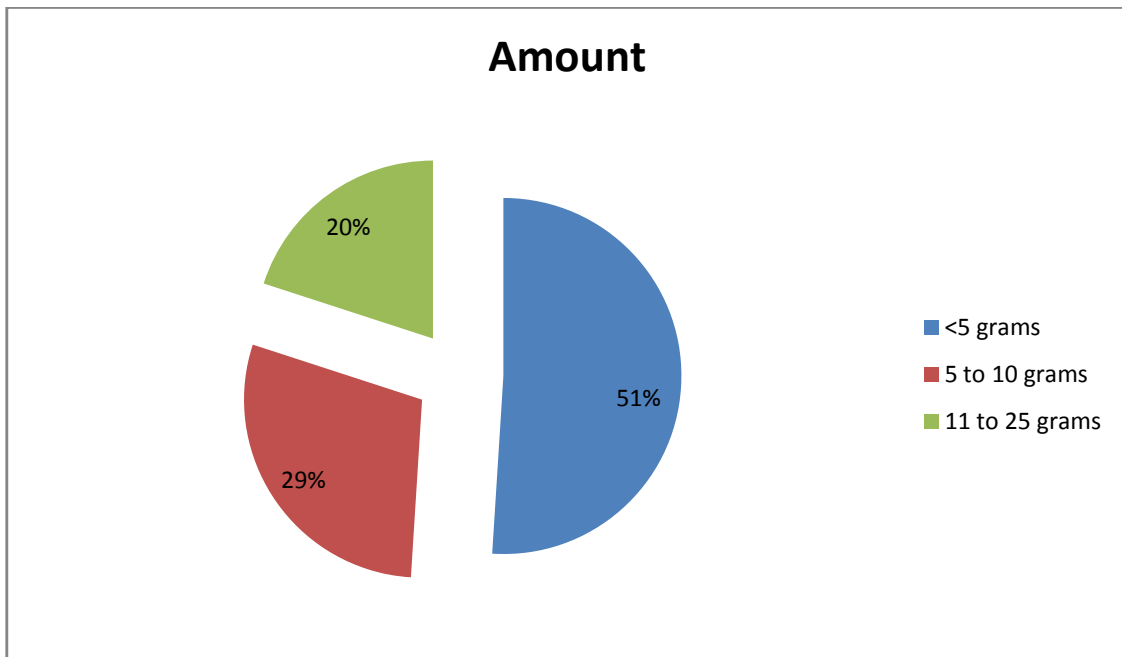


Table 10: Distribution of Blood pressure at admission

BP	No of Patients	%
Normal	181	88.7
Low	23	11.3
Total	204	100.0

Figure 14: Distribution of Blood pressure at admission

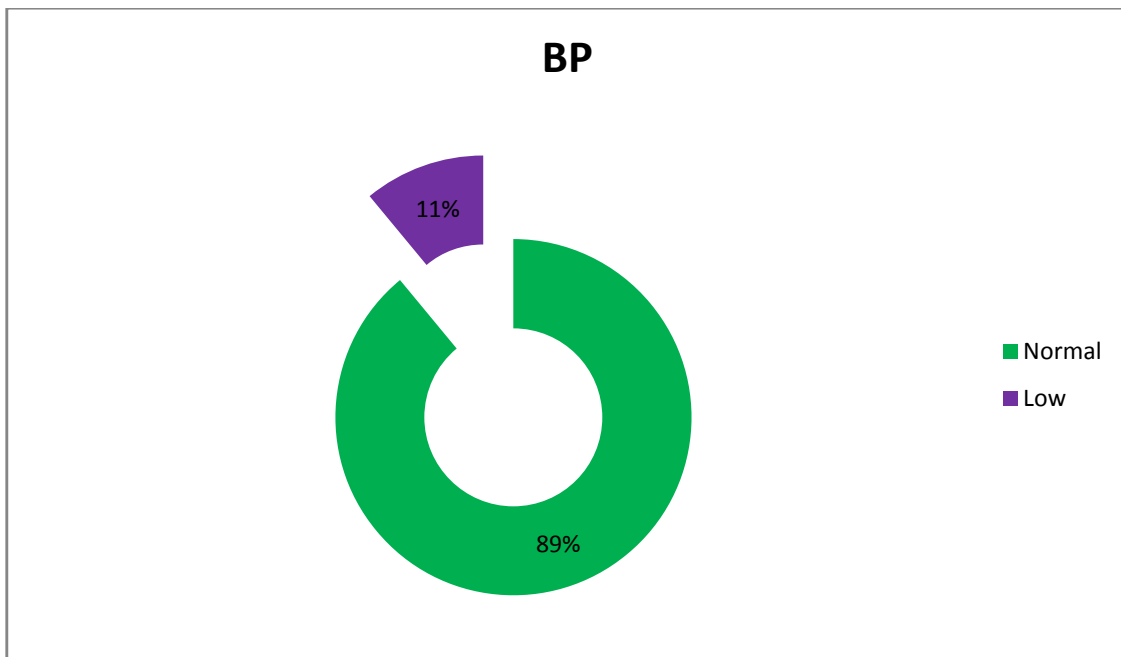


Table 11: Distribution of Pulse Rate at admission

PR	No of Patients	%
Normal	151	74.0
Low	8	3.9
High	45	22.1
Total	204	100.0

Figure 15: Distribution of Pulse Rate at admission

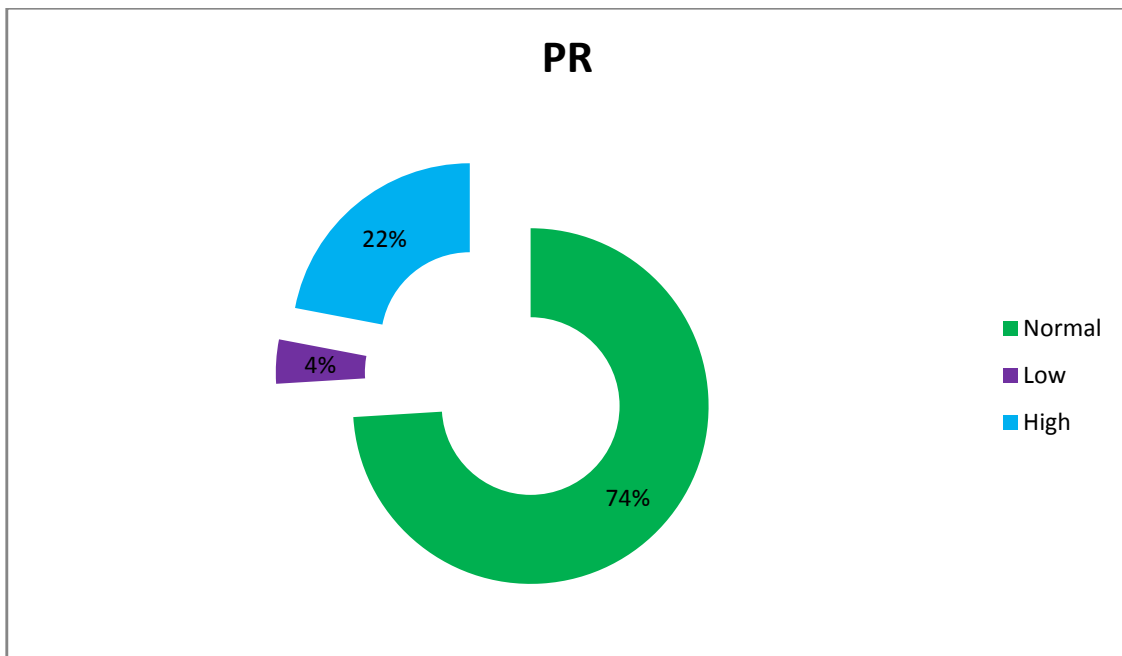


Table 12: GCS at Time of Admission

GCS	No of Patients	%
14&15	148	72.5
8 to 13	38	18.6
<8	18	8.8
Total	204	100.0

Figure 16: GCS at Time of Admission

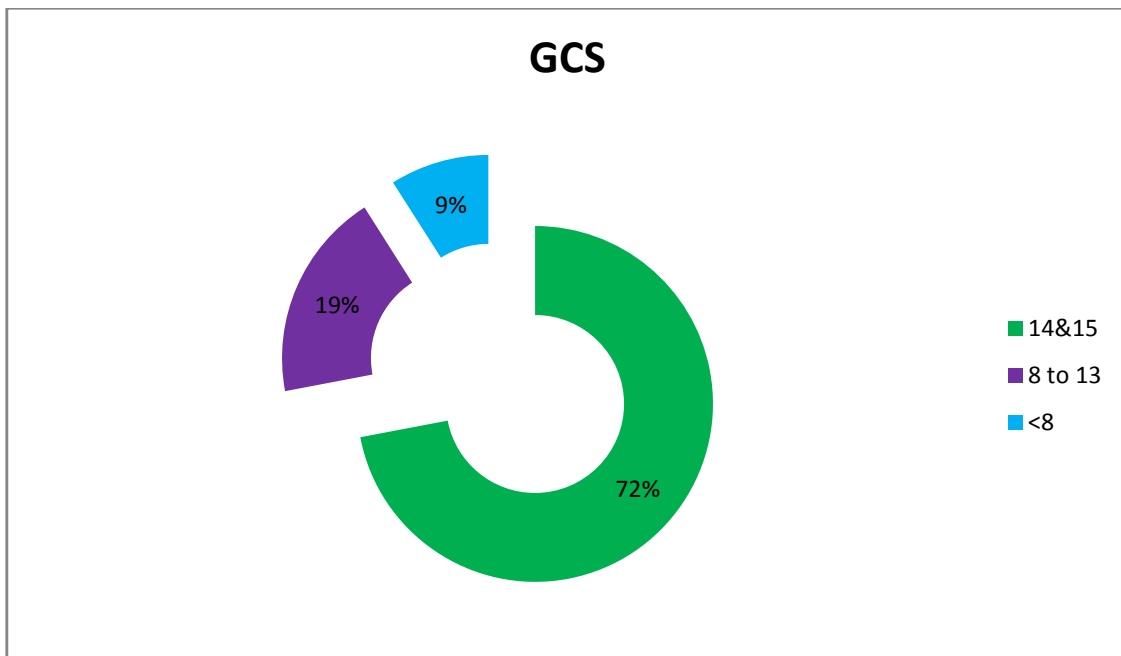


Table 13: Relation between Time to admission and Outcome of patients.

Time to admission and Outcome Crosstabulation						
			Outcome			Total
			Recovery without complications	Recovery with complications	Death	
Time to admission	<6 Hours	Count	44	4	1	49
		% within Outcome	42.3%	7.5%	2.1%	24.0%
	6-24 Hours	Count	53	23	12	88
		% within Outcome	51.0%	43.4%	25.5%	43.1%
	>24 Hours	Count	7	26	34	67
		% within Outcome	6.7%	49.1%	72.3%	32.8%
Total		Count	104	53	47	204
		% within Outcome	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=82.858** p<0.001

Figure 17: Relation between Time to admission and Outcome of patients.

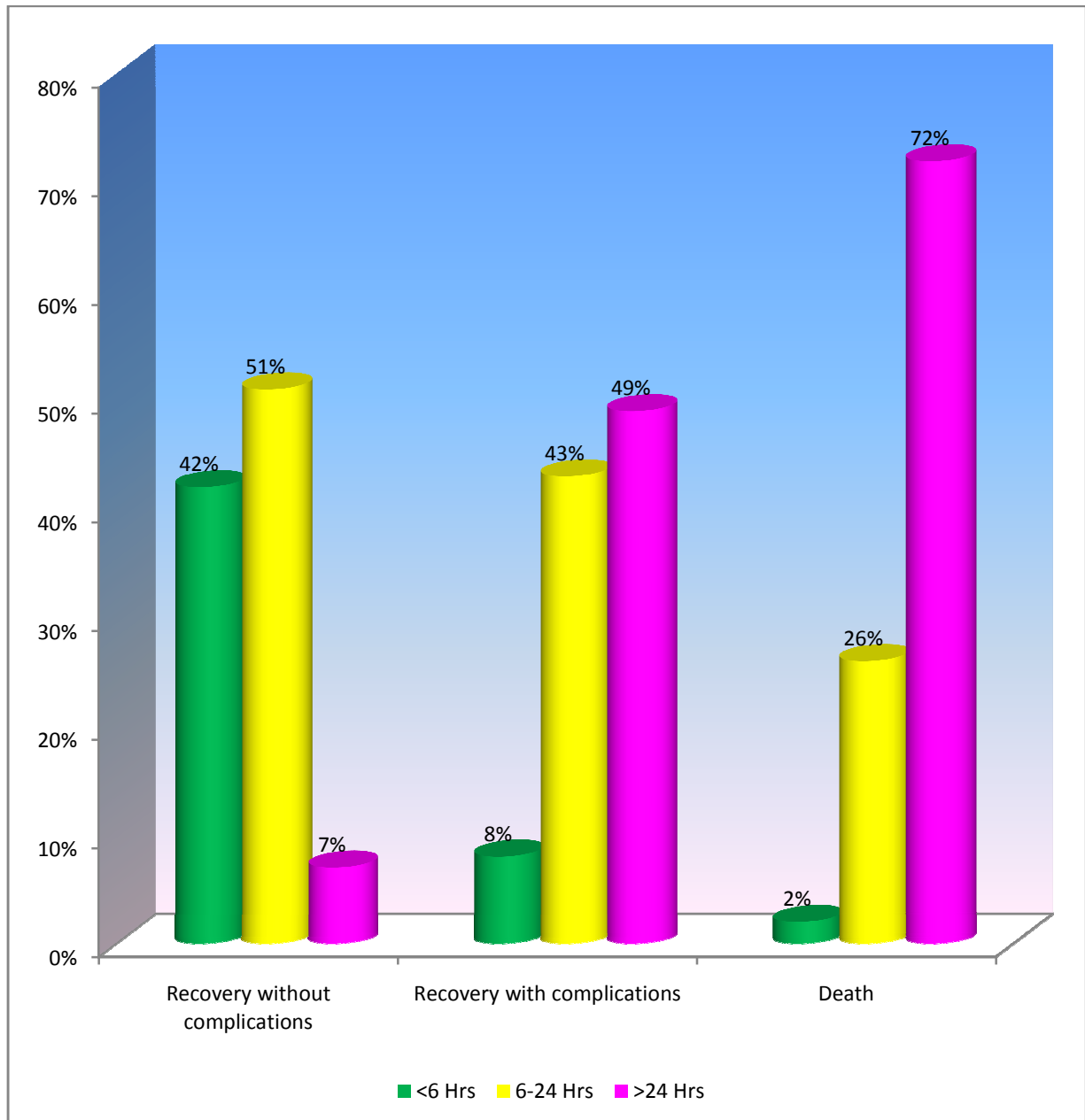


Table 14: Relation between Amount of poison and Outcome of patients

		Outcome			Total
		RECOVERY WITHOUT COMPLICATIONS	RECOVERY WITH COMPLICATIONS	DEATH	
Amount	<5 grams Outcome	Count 72 69.2%	Count 27 50.9%	Count 5 10.6%	Count 104 51.0%
	5 to 10 grams Outcome	Count 11 10.6%	Count 21 39.6%	Count 28 59.6%	Count 60 29.4%
	11 to 25 grams Outcome	Count 21 20.2%	Count 5 9.4%	Count 14 29.8%	Count 40 19.6%
	Total Outcome	Count 104 100.0%	Count 53 100.0%	Count 47 100.0%	Count 204 100.0%

Pearson Chi-Square=56.060** p<0.001

Figure 18: Relation between Amount of poison and Outcome of patients.

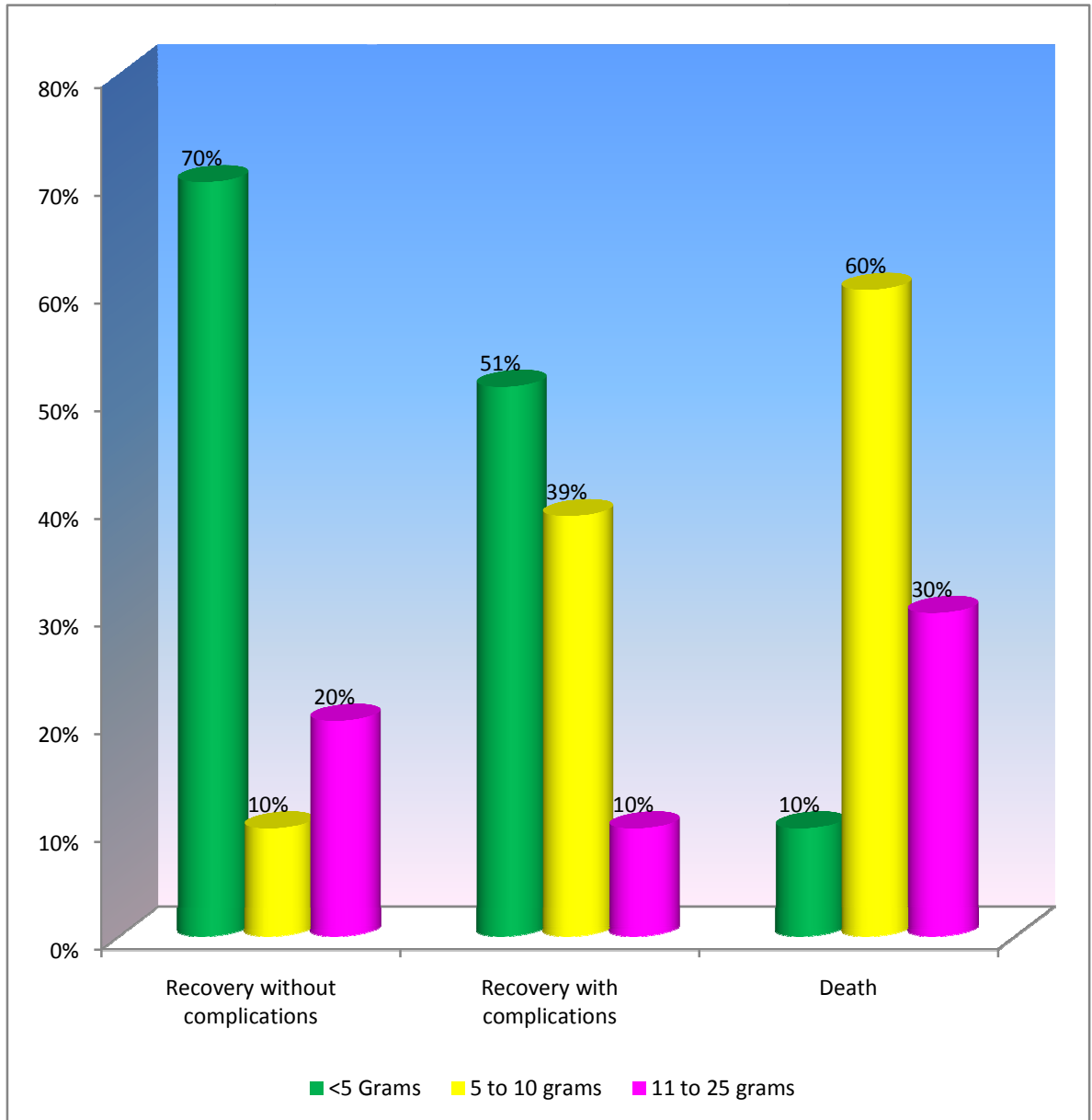


Table 15: Complication Distribution

		No of Patients	% of Total
Vomiting	No	118	57.8%
	Yes	86	42.2%
Abdominal Pain	No	138	67.6%
	Yes	66	32.4%
Bleeding manifestation	No	191	93.6%
	Yes	13	6.4%
Altered Sensorium	No	165	80.9%
	Yes	39	19.1%
Jaundice	No	156	76.5%
	Yes	48	23.5%
Others	No	184	90.2%
	Yes	20	9.8%

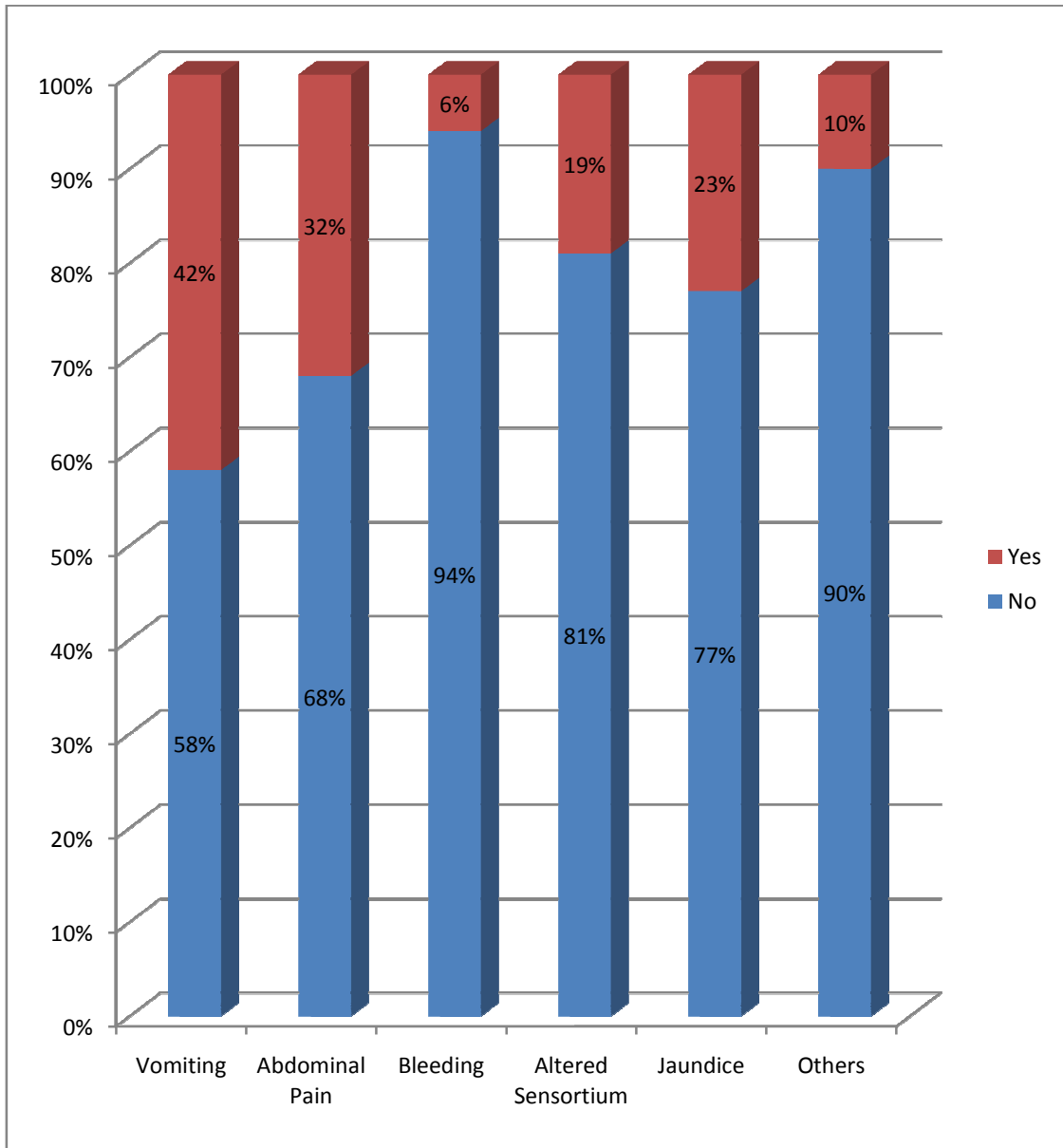


Figure 19: Complication Distribution

Table 16: Distribution of RBS, Total bilirubin and INR among patients

		No of Patients	% of Total
RBS	>90 mg/dl	128	63.1%
	55 to 90 mg/dl	56	27.6%
	<55 mg/dl	19	9.4%
Total Bilirubin	Normal	141	69.1%
	Low	0	0.0%
	High	63	30.9%
Creatinine	Normal	183	89.7%
	Low	0	0.0%
	High	21	10.3%
INR	<1.1	129	63.2%
	1.1 to 2.99	36	17.6%
	3 to 3.99	28	13.7%
	>4	11	5.4%

Figure 20: Distribution of RBS, Total bilirubin and INR among patients

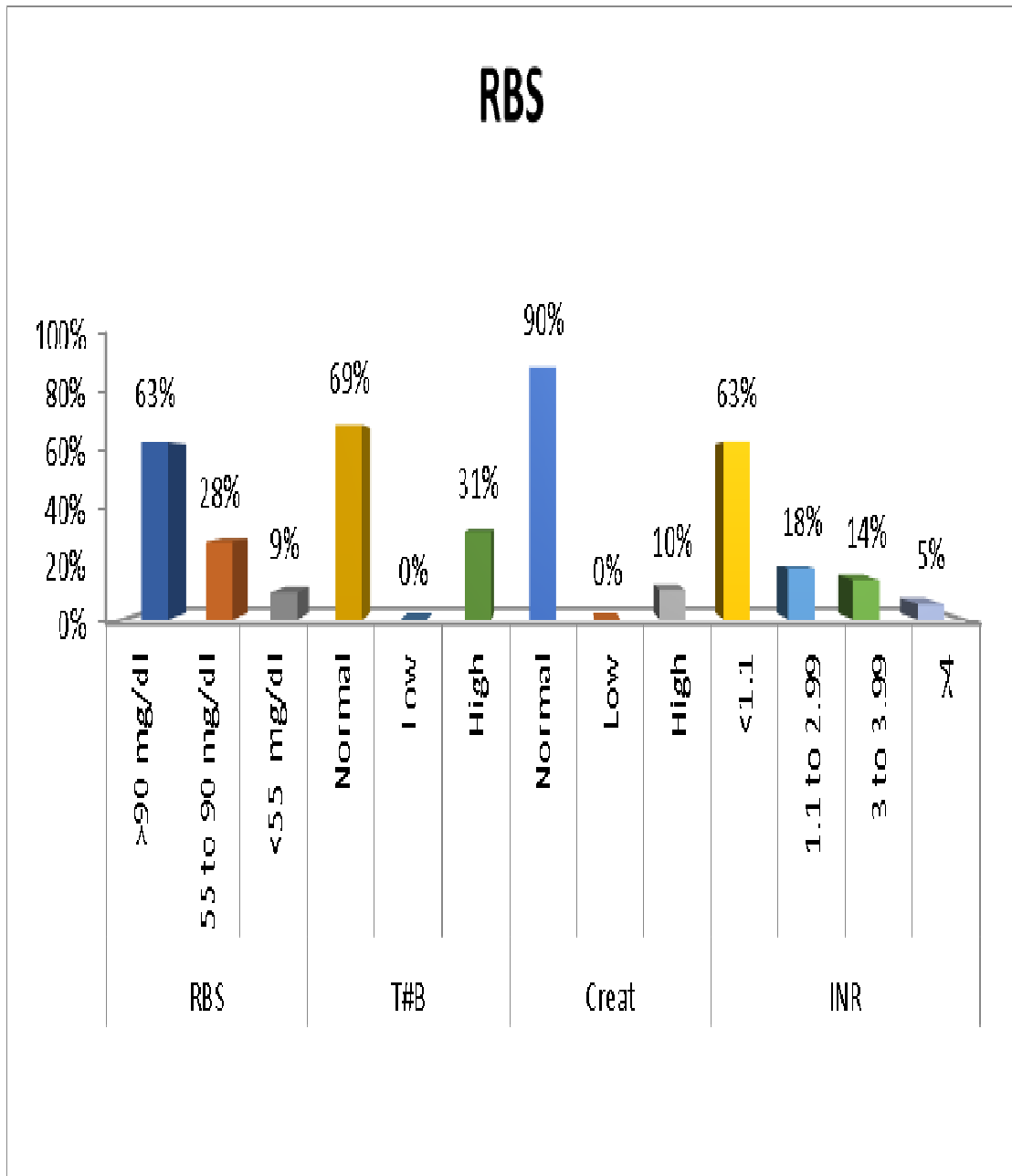


Table 17 and 18: Relation between Yellow phosphorus poisoning and Hepatic Transaminases

		YELLOW PHOSPHOROUS	
		No of Patients	% of Total
AST	Normal	44	39.6%
	2 nd day	11	9.9%
	3 rd day	16	14.4%
	4 th day	15	13.5%
	5 th day	17	15.3%
	6 th day	7	6.3%
	7 th day	1	0.9%
ALT	Normal	44	39.6%
	2 nd day	11	9.9%
	3 rd day	16	14.4%
	4 th day	15	13.5%
	5 th day	17	15.3%
	6 th day	7	6.3%
	7 th day	1	0.9%

Figure 21: Relation between Yellow phosphorus poisoning and AST

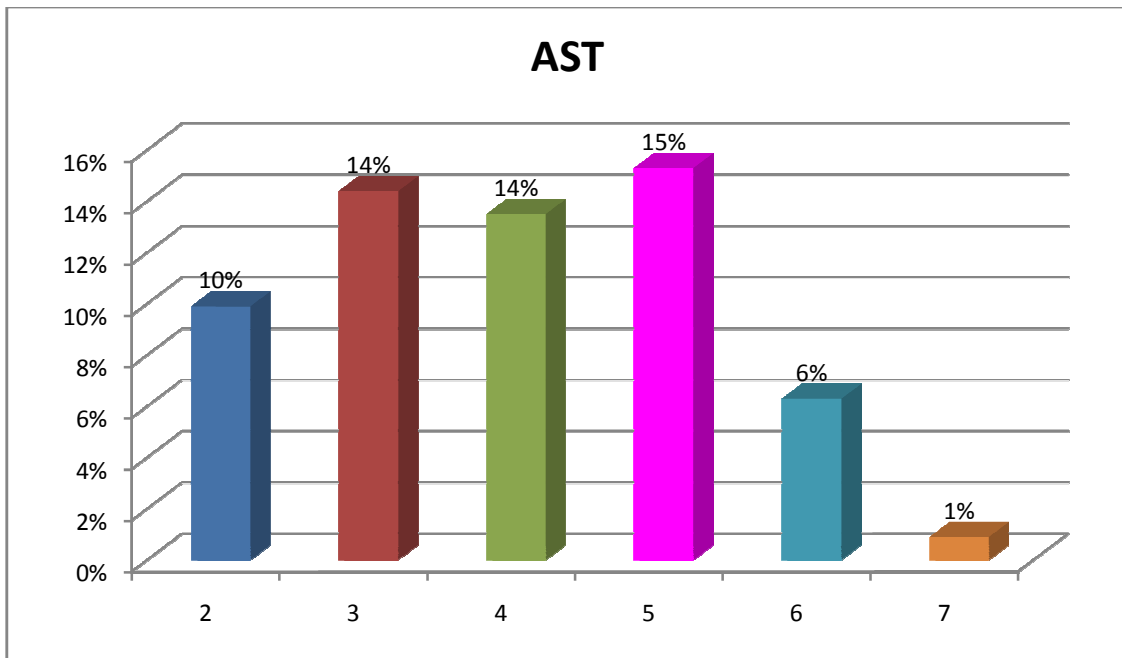


Figure 22: Relation between Yellow phosphorus poisoning and ALT

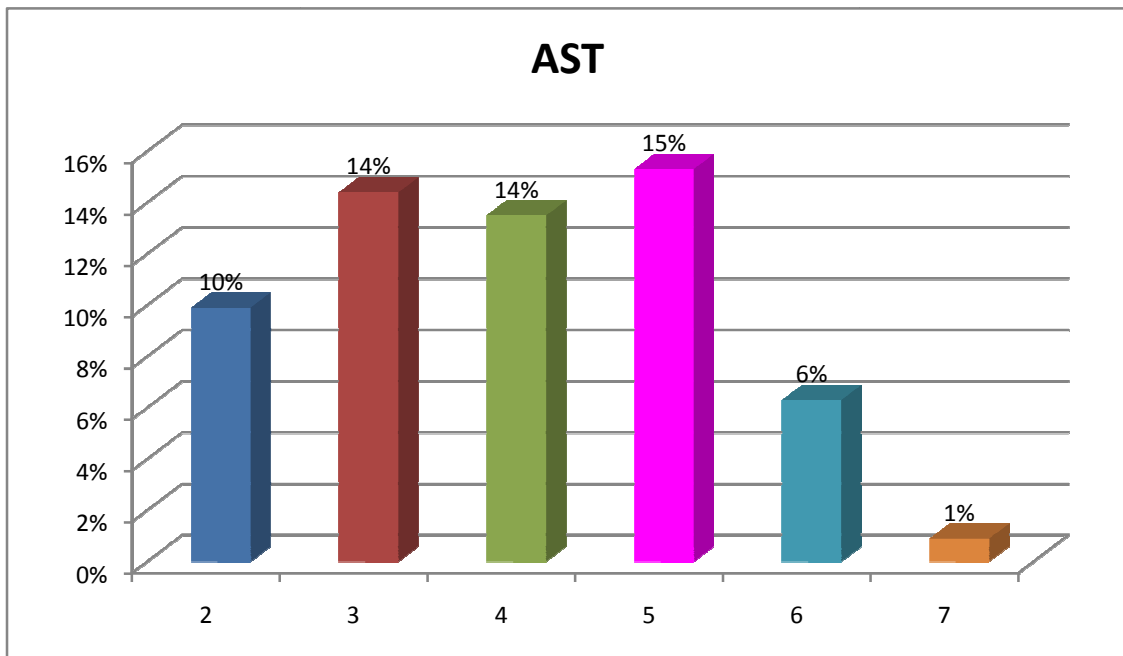


Table 19: Outcome of all the poisons

		Outcome			Total	
		Recovery without complications	Recovery with complications	Death		
Type	YELLOW PHOSPHOROUS	No of Patients	26	40	45	111
		% of Total	25.0%	75.5%	95.7%	54.4%
	SUPER WARFARIN	No of Patients	31	6	0	37
		% of Total	29.8%	11.3%	0.0%	18.1%
	ZINC PHOSPHIDE	No of Patients	47	7	2	56
		v	45.2%	13.2%	4.3%	27.5%
	Total	No of Patients	104	53	47	204
		% of Total	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=78.357** p<0.001

Figure 23: Outcome of all the poisons

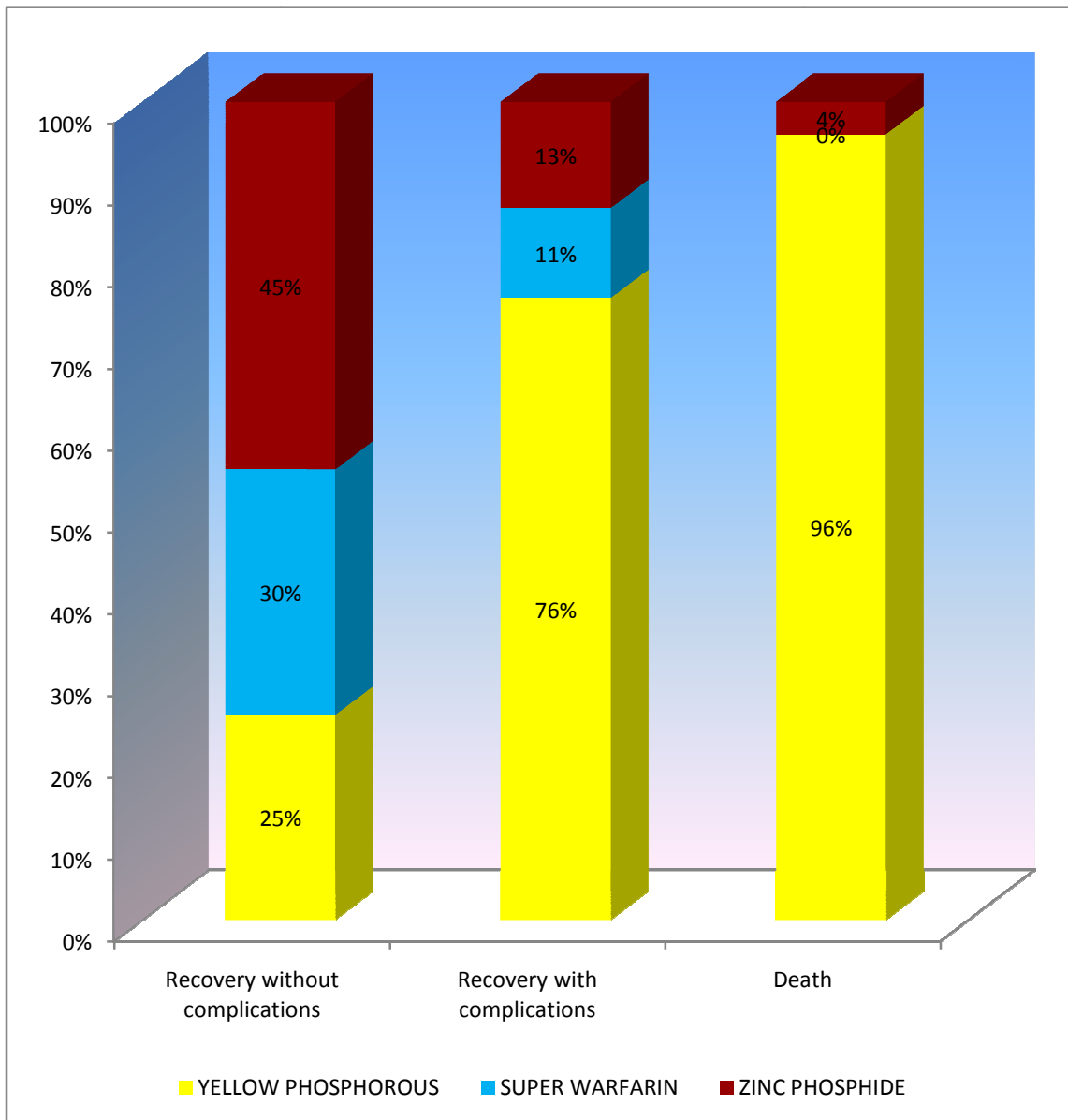


Table 20: Distribution of Complications

Complications	No of Patients	%
NO COMPLICATIONS	101	49.5
↑INR WITHOUT BLEEDING	13	6.4
↑INR WITH BLEEDING	7	3.4
ACUTE HEPATITIS	23	11.3
HEPATIC ENCEPALOPATHY	23	11.3
ACUTE KIDNEY INJURY	5	2.5
ARDS	27	13.2
HYPOGLYCEMIA	5	2.5
Total	204	100.0

Figure 24: Distribution of Complications

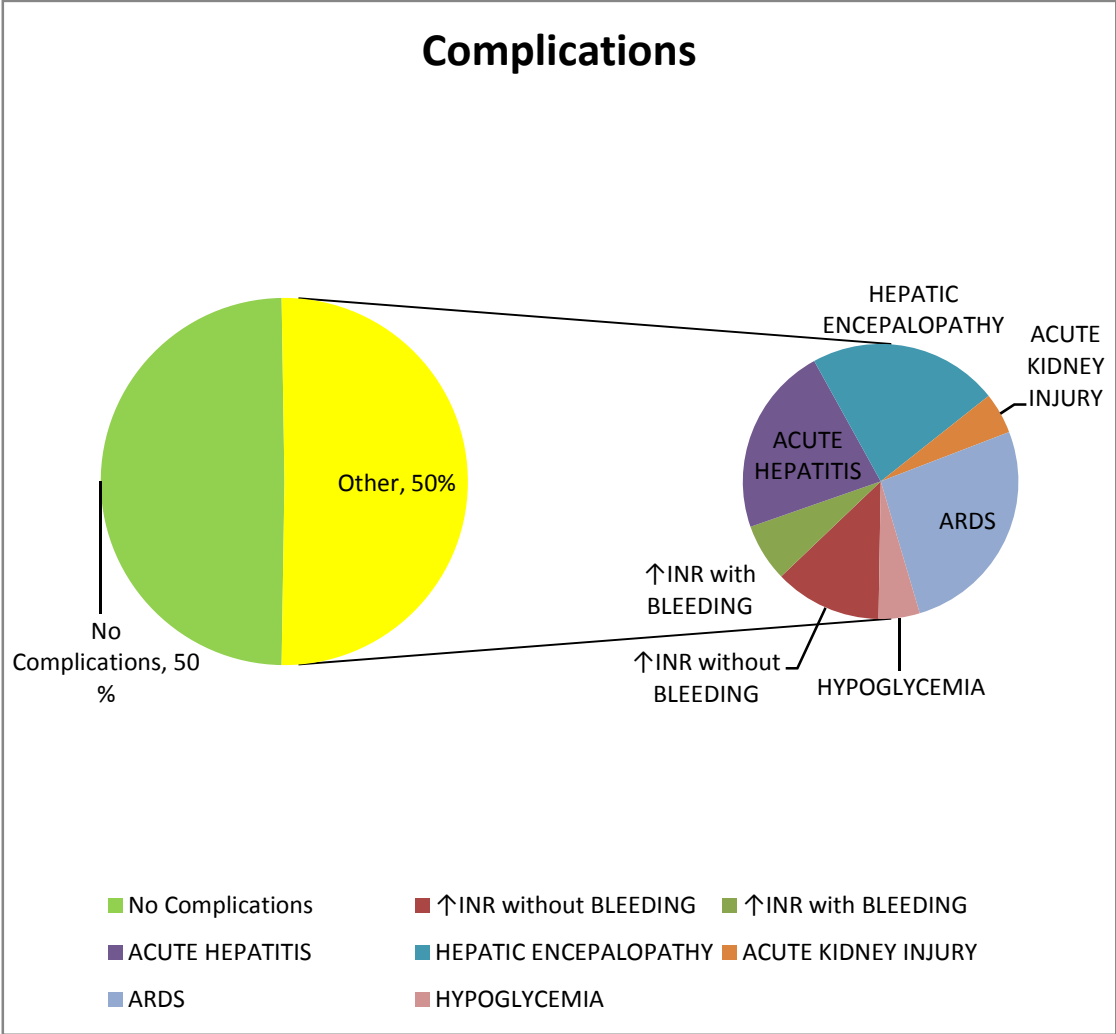


Table 21: Relation between Types of poison and Complications

Complication and Type of poison Crosstabulation						
			Type			Total
			Yellow Phosphorous	Super Warfarin	Zinc Phosphide	
Complic	No Complications	Count	24	30	47	101
		% within Type	21.6%	81.1%	83.9%	49.5%
	↑INR WITHOUT BLEEDING	Count	7	6	0	13
		% within Total	6.3%	16.2%	0.0%	6.4%
	↑INR WITH BLEEDING	Count	6	1	0	7
		% within Total	5.4%	2.7%	0.0%	3.4%
	ACUTE HEPATITIS	Count	23	0	0	23
		% within Total	20.7%	0.0%	0.0%	11.3%
	HEPATIC ENCEPALOPATHY	Count	23	0	0	23
		% within Total	20.7%	0.0%	0.0%	11.3%
	ACUTE KIDNEY	Count	3	0	2	5

	INJURY	% within Total	2.7%	0.0%	3.6%	2.5%
		Count	20	0	7	27
	ARDS	% within Total	18.0%	0.0%	12.5%	13.2%
		Count	5	0	0	5
	HYPOGLYCEMIA	% within Total	4.5%	0.0%	0.0%	2.5%
		Count	111	37	56	204
Total	% within Total	100.0%	100.0%	100.0%	100.0%	

Pearson Chi-Square=101.511** p<0.001

Figure 25: Relation between Types of poison and Complications

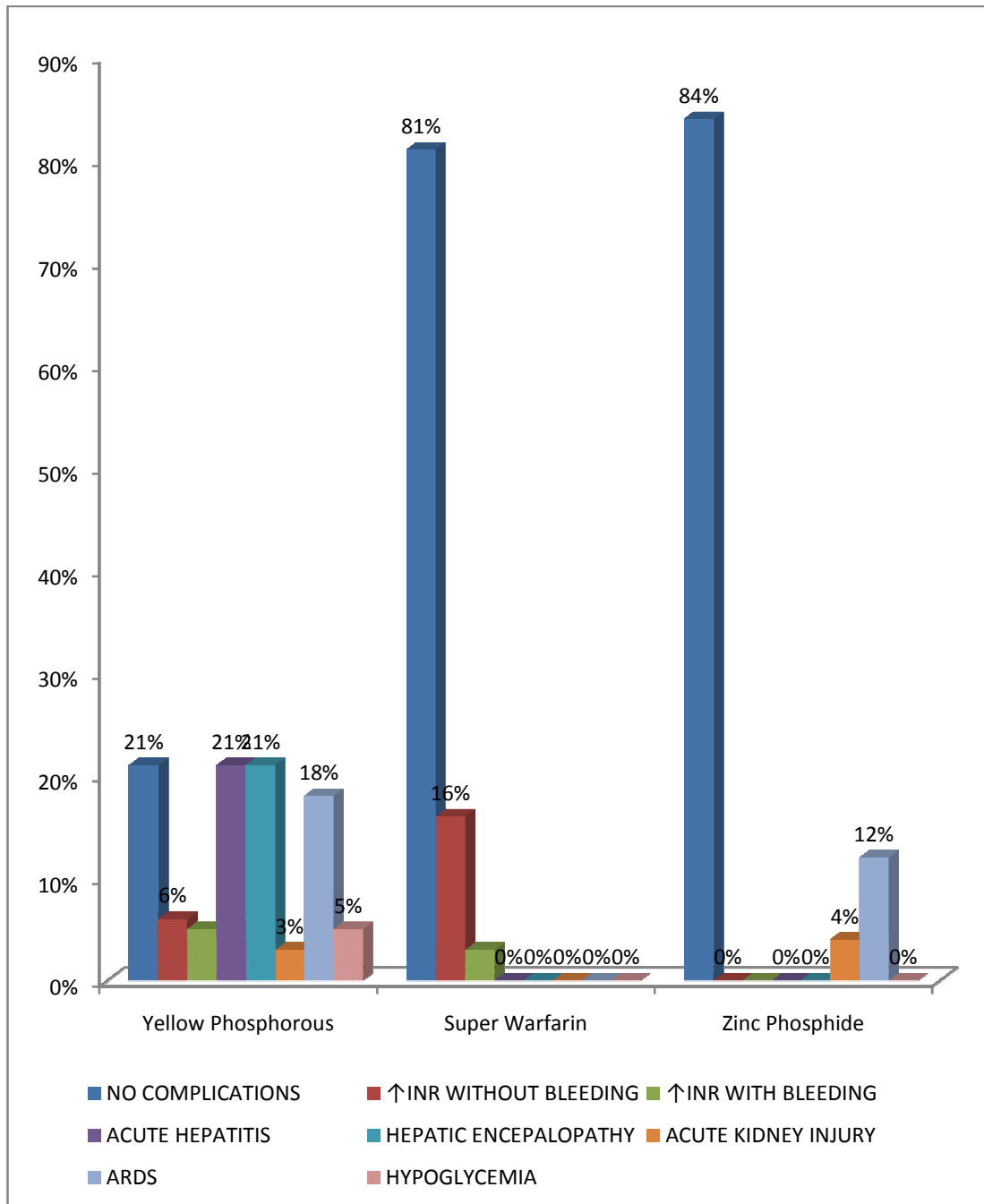


Table 22: MELD score and relation to outcome of yellow phosphorus poison

Descriptives								
MELD								
	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
No Complications	24	6.0000	.00000	.00000	6.0000	6.0000	6.00	6.00
Increasing INR without BLEEDING	7	19.1429	8.09174	3.05839	11.6592	26.6265	4.00	28.00
Increasing INR with BLEEDING INR	6	25.6667	6.37704	2.60342	18.9744	32.3590	20.00	36.00
ACUTE HEPATITIS	23	27.6957	4.89373	1.02041	25.5794	29.8119	14.00	35.00
HEPATIC ENCEPALOPATHY	23	34.5652	4.68873	.97767	32.5377	36.5928	26.00	40.00
ACUTE KIDNEY INJURY	3	21.0000	7.00000	4.04145	3.6110	38.3890	16.00	29.00
ARDS	20	15.3500	11.76648	2.63106	9.8431	20.8569	6.00	40.00
HYPOGLYCEMIA	5	20.2000	13.04607	5.83438	4.0012	36.3988	6.00	32.00
Total	111	21.0360	12.23848	1.16163	18.7340	23.3381	4.00	40.00

p<0.001

Figure 26: MELD score and relation to outcome of yellow phosphorus poison

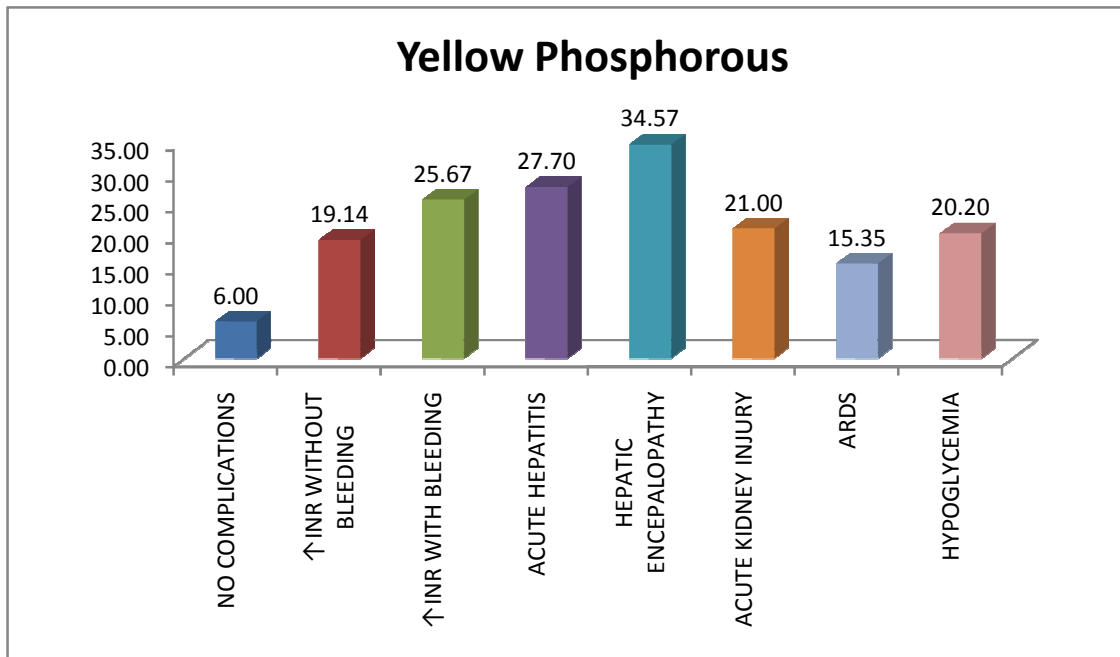


Table 23: Poison Severity Score Distribution

PSS	No of Patients	%
0	76	37.3
1	51	25.0
2	27	13.2
3	3	1.5
4	47	23.0
Total	204	100.0

Figure 27: Poison Severity Score Distribution

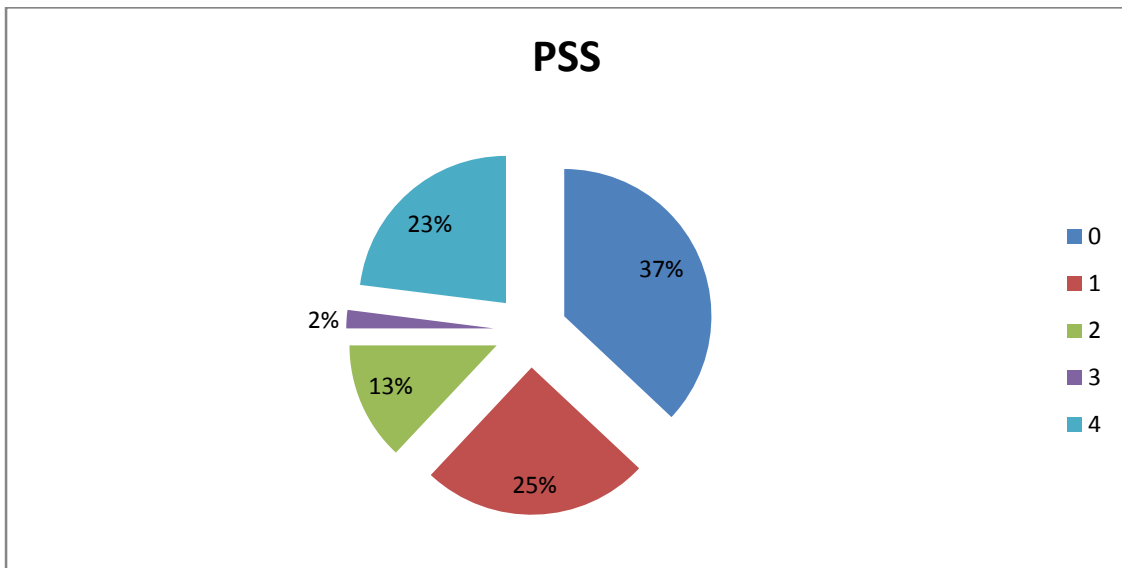


Table 24: Outcome of all Poisons.

Outcome	No of Patients	%
Recovery without complications	104	51.0
Recovery with complications	53	26.0
Death	47	23.0
Total	204	100.0

Figure 28: Outcome of all Poisons

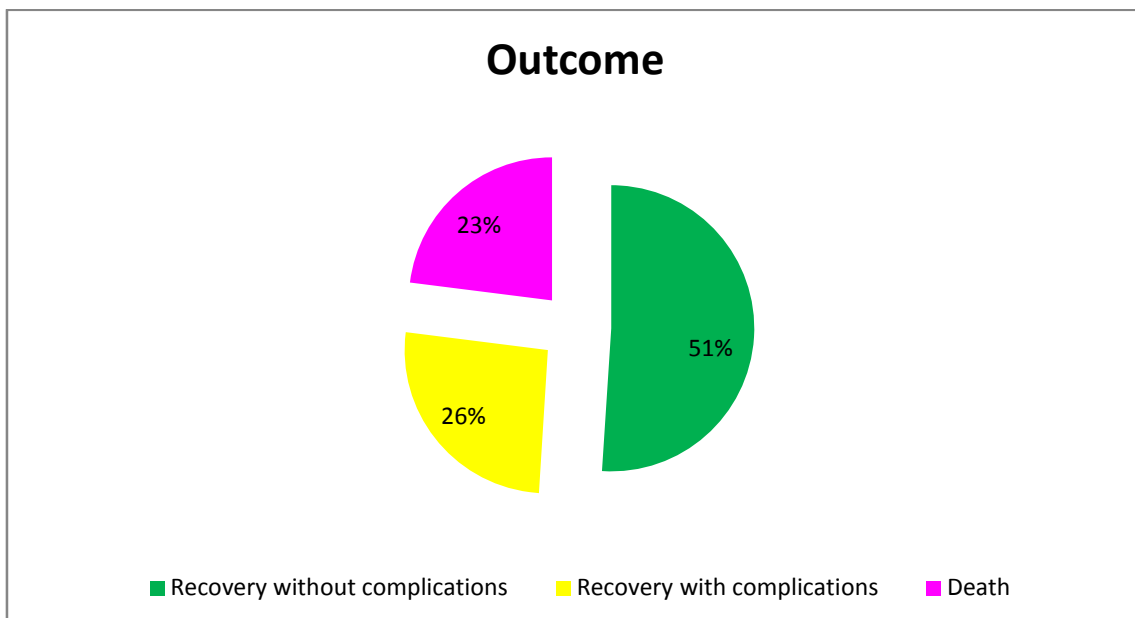
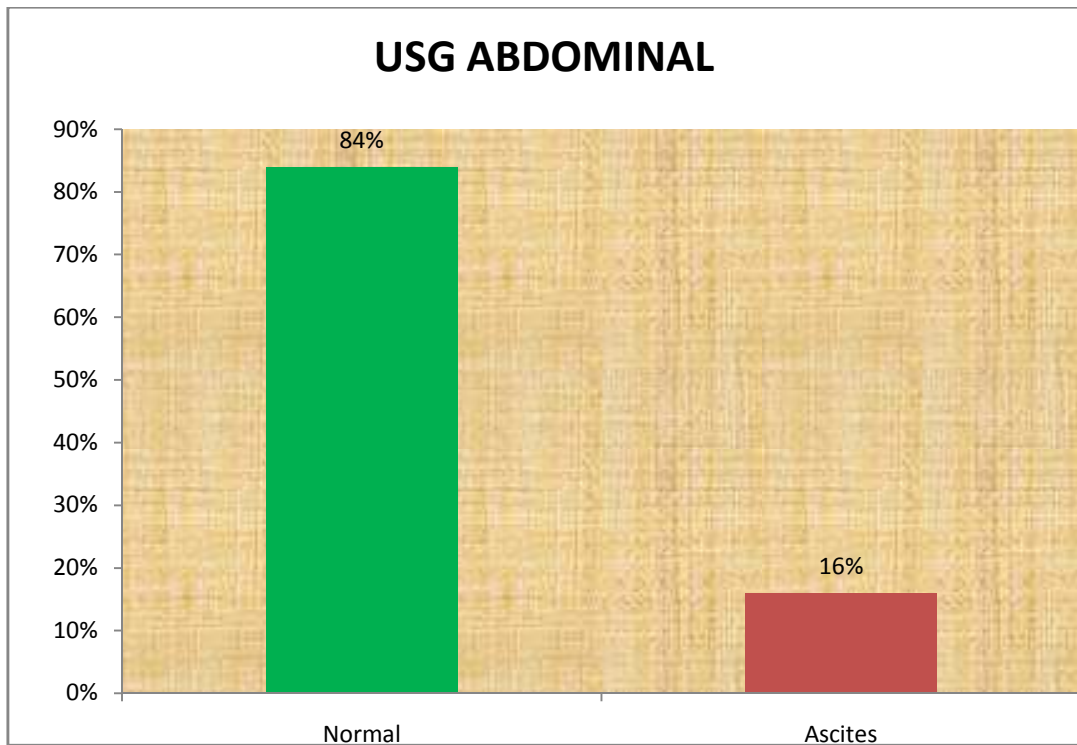


Table 25.USG Abdomen in Yellow Phosphorous Poison.

			YELLOW PHOSPHOROUS
USG ABD	Normal	Count	93
		% within Total	83.8%
	Ascites	Count	18
		% within Total	16.2%
Total		Count	111
		% within Total	100.0%

Figure 29: USG Abdomen in Yellow Phosphorous Poison.



DICUSSION

The total number of patients in our study was 204 who were selected after fulfilling the inclusion criteria.

-Out of 204 patients, Patients less than 18 years were 35 (17.2%), between 19 to 28 years were 98 (48%), between 29 to 39 were 43 (21.1%). 19 (9.3%) patients were between 40 to 49 years and 9 (4.4%) patients were beyond 50. More than 65% of patients fall below 29 years of age. As shown in Table 3 and Figure 7.

-In Karanth S, Nayyar V. et.al. study 61.3% of the population belonged age less than 30 years⁽²⁹⁾.

- *In our study 133 (55.2 %) patients were under the age of 29 years which is similar to karanth et al study.*

- Out of 204 patients, 95 (46.6 %) were males and 109 (53.4%) were females. A slight predominance of females over men in a ratio of 1: 1.4. As shown in Figure 8 and table 4.

- In Banerjee I, Tripathi SK, Roy AS et al study done over two and a half years showed a male to female ratio of 1: 1.3 with female predominance⁽³¹⁾.

- In Nalabothu M, Monigari N, Acharya R. et al study⁽³⁰⁾, Out of 97 patients, 56 (57.7%) were male and 41 (42.3%) were female which shows slight male prevalence in a ratio of 1: 1.36.

- In our study, males were 95 (46.6 %) and females were 109 (53.4%) with female incidence more than male which is in concordance with Banerjee et al study and is different from Nalabothu et al study.

- Out of 204 patients studied, 73 (35.8%) were unmarried and 131 (64.2%) were married. Rodenticide Poisoning seems to be common in married population. As shown in figure 9, table 5.

- Total of 49 (24%) patients who consumed poison got admitted within 6 hours of ingestion. 88 (43.1%) patients got admitted after 6 hours but before 24 hours of consumption. 67 (32.8%) patients got admitted after 24 hours of poison consumption. This is shown in Table 6 and figure 10.

- In Nalabothu M, Monigari N, Acharya R. et al study, out of 97 patients, 92 (94.8%) were suicidal and 5 (5.2 %) were accidental⁽³⁰⁾.

- Out of 204 patients, 203 patients consumed poison with suicidal intent. Only patient had an accidental exposure to the rodenticide. This finding is similar to the study done by Nalabothu et al. This is shown in Table 7 and figure 11.

- Total of 111 (54.4%) patients consume yellow phosphorus, 37 (18.1%) patients consumed super warfarin's and 56 (27.5%) out of 204 patients consumed zinc phosphide. This distribution is shown in Table 8 and Figure 12.

- Total of 104 (51%) patients consumed poison in dose of less than 5 grams ,60 (29.4%) patients have consumed in a dose of 5 to 10 grams. 40 (19.6%) consumed more than 10 grams. As shown in table 9 and figure 13.

- Out of 204 patients admitted, 86 (42.2%) had vomiting, 66 % (32.4%) had abdominal pain, 13 (6.4%) had bleeding manifestation, 39 (19.1%) had altered sensorium, 48 (23.5%) had jaundice, 20 (9.8%) had other symptoms like breathlessness at the time of admission. As shown in table 15 and figure 19.

- In a study done by Arun Kumar et al, out of 303 patients ,192 (63.3 %) had vomiting, 72 (23%) had abdominal pain and 95 (31.3%) patients were asymptomatic.

- *In our study clinical features are similar to findings seen in Arunkumar et al study.*

- Out of total 204 patients, 181 (88.7%) patients had normal BP at admission, 23 (11.3 %) patients were hypotensive. As shown in table 10 and figure 14.

- Out of 204 patients ,151 (79%) had normal pulse rate, 8(3.9 %) patients were in bradycardia and 45 (22.1%) patient had tachycardia. Shown in table 11 and figure 15.

- Out of 204 patients, 148 (72.5%) patients had GCS of 14 or 15 at presentation. 38 (18.6%) patients had GCS between 8 to 13. 18 (8.8%) patients had GCS less than 8 at presentation. As shown in table 12 and figure 16.

- Out of 204 patients admitted, 128 (63.1%) had RBS of > 90 mg/dl, 56 (27.6%) had RBS between 55 to 90 mg/dl, 19 (9.1%) had RBS < 55 mg/dl at the time of admission. As shown in table 16 and figure 20.

- Out of 204 patients, 63 (30.9%) high bilirubin and 141 (69.1%) had normal range of bilirubin during the course of treatment. As shown in table 16 and figure 20.

- Out of 204 patients, 183 (89.7%) had creatinine which is of normal range and 21 (10.3%) had high creatinine during the course of stay. As shown in table 16 and figure 20.

- Out of 204 patients, 129 (63.2%) had INR < 1.1, 36 (17.6%) had INR between 1.1 to 2.99, 28 (13.7%) had INR between 3 and 3.99, 11 (5.4%) had an INR 4 and beyond. INR was taken 72 hours after consumption of poison. As shown in table 16 and figure 20.

- In Karanth S, Nayyar V. et.al. study⁽²⁹⁾, Out of 334 patients consumed yellow phosphorous, 27 (8.8%) had INR < 1.7, 17 (5%) had INR between 1.7 and 2.2 and 52 (16%) had more than 2.2. In the same study 134

(40.11%) patients had acute hepatitis, 95 (28.4%) had hepatic encephalopathy, had 51 (15.3 %) had hypoglycemia.

- Out of 204 patients, 101(49.5%) had no complications, 23 (11.3%) had developed acute hepatitis during the course of treatment, 23 (11.3%) had developed hepatic encephalopathy. 27 (13.2 %) had developed ARDS, 13 (6.4%) had increased INR without bleeding, 7 (3.4%) had increased INR with bleeding manifestation, 5 (2.5%) developed AKI and 5 (2.5%) had hypoglycemia. As shown in table 20 and figure 24.

- In our study the occurrence of complications is different from what is seen in Karanth et al studies.

- *Relation between time to admission and clinical outcome in our study is significant as p value <0.001.*

Out of 49 patients who got admitted before 6 hours, 44 (42.3%) had no complications ,4 had recovered with complications and only 1 death.

Out of 88 patients who got admitted between 6 to 24 hours, 53 (51%) had recovered without complications, 23 (43.4%) recovered with complication, 12(25.5%) died.

- Out of 67 patients who got admitted beyond 24 hours, 7 (6.7%) had no complication, 26 (49.1%) recovered with complications and 34 (72.3%) died. As shown in table 13 and figure 17.

- It is evident from this comparison that patients admitted before 6 hours had low mortality rate and admission beyond 24 hours have high mortality rate.

-There was a significant correlation between the amount of poison ingestion to outcome of patients, as p value <0.001 .

Out of 104 patients who consumed <5 grams, 72 (69.2%) recovered without complication, 27 (50.9%) recovered with complications and 5 (10.6%) died.

Out of 60 patients who consumed between 5 to 10 grams, 11 (10.6%) recovered without complications, 21 (39.6%) patients recovered with complications, 28 (59.6%) died.

Out of 40 patients who consumed more than 10 grams, 21 (20.2%) recovered without complication, 5 (9.4%) recovered with complication and 14 (29.8%) died.

Patients who consumed less than 5 gram had low mortality rate.

-Out of 111 patients who consumed yellow phosphorus, 44 (39.6%) patient had no rise in AST/ALT during the course of stay, 11 (9.9%) had rise in day 2, 16 (14.4%) had rise on day 3, 15 (13.5%) had rise on day 4, 17 (15.3%) had rise on day 5, 7 (6.3%) had rise on day 6 and one (0.9%) had rise only on day 7. As shown in table 17, 18 and figure 21, 22.

Around 48 (43.2%) of all the patients consumed had a raise in AST/ALT from 3rd day to 5th day. Around 39.6% of all patients consumed yellow phosphorus had no raise in AST/ALT.

-The relationship between type of poison and outcome has been tabulated and relation was significant as p value <0.001 and the same is shown in table 19 and figure 23.

-Out of 111 patients consumed yellow phosphorus, 26 (25%) recovered without complications, 40 (75.5%) had recovered with complications, 45 (95.7%) of patients died.

-Out of 37 patients consumed super warfarin, 31 (29.8%) recovered without complications, 6 (11.3%) recovered with complications and zero deaths.

-Out of 56 patients consumed zinc phosphide, 47 (45.2%) recovered without complications, 7 (13.2%) recovered with complications and 2 (4.3%) patients died.

-Out of 204 patients, 104 (51%) had no complications, 53 (26%) had recovery with complications and 47 (23%) patients ended up being dead.

- *Superwarfarins had the best outcome compared to other rodenticides as it had zero mortality and majority had no complications. This finding is similar to Ying Yu et al⁽³⁵⁾ and Nalabothu et al⁽³⁰⁾ studies.*

-Type of poison was cross tabulated with complication of poisoning and the correlation is found to be significant as p value<0.001. This relation is shown in table 21 and figure 25.

- Out of 101 patients who had no complications during the course of stay in hospital, 26 (25%) were in Yellow phosphorus poisoning, 30 (81.1%) were in superwarfarins poisoning, 47 (83.9%) were in zinc phosphide poisoning.

- Out of 13 patients with raised INR without bleeding, 7 (6.3%) were due to yellow phosphorus ,6 (16.2%) were due to superwarfarins and no such complications in zinc phosphide.

- Out of 7 patients with raised INR and bleeding tendency, 6 (5.4%) were due to Yellow phosphorus and 1(2.7 %) due to superwarfarins.

- *Out of 23 patients with Acute hepatitis, all were due to Yellow phosphorus.*

- *Out of 23 patients with Hepatic encephalopathy all were due to Yellow phosphorus poisoning.*

- Out of 27 patients with ARDS, 20 (18%) was due to yellow phosphorus and 7 (12.5%) was due to zinc phosphide.

- Out of 5 patients with AKI , 3 were due to yellow phosphorus and 2 were due to zinc phosphide.

- *Out of 5 patients who had hypoglycemia , all were due to yellow phosphorus.*

-Poison severity score was calculated for rodenticides and is shown in figure 27 and table 23.

- 76 (37.3%) patients had zero score.
- 51 (25%) patients had score of 1.
- 27 (13.2%) patients had score of 2.
- 3 (1.5%) patients had score of 3.
- 47 (23.1%) patients had score of 4.

-Out of 111 patients who consumed yellow phosphorus, 93 (83.6%) had normal USG Abdomen, 18 (16.4 %) had ascites. As shown in table 25 and figure 29.

-MELD score was correlated with clinical outcome of yellow phosphorus poisoning which was statistically significant as evident by p value <0.001.

- Katoonizadeh A, Decaestecker J, Wilmer A, Aerts R, Verslype C, Vansteenbergen W, et al. study ⁽³⁶⁾, favor's use of MELD score to predict the outcome.

- In our study also an increase in MELD score is associated with increasing rate of complications.

Yellow Phosphorus:

In Karanth S, Nayyar V. et al study, where out of 334 patients, 134(4.11%) developed toxic hepatitis, 70 (20.95%) developed jaundice, 51 (15.27%) developed hypoglycemia ⁽²⁹⁾.

In our study, Acute hepatitis is seen in 23 (11.2%) out of 204 patients, 63 (30.8%) patients had jaundice and 19 (9.3%) patients had hypoglycemia. As shown in table 16, 21 and figure 20, 25.

In Nalabothu M et al study, out of 43 patients, 21 (48.8%) survived and 12 (27.9%) died and 10 (23.3 %) were discharged AMA ⁽³⁰⁾.

In Pande TK, Pandey S. et al study which was done on white phosphorus poisoning, reported a case fatality rate of 10 to 50% ⁽³³⁾.

In Fernandez and Canzares et al published a case series of 15 patients with yellow phosphorus where the mortality rate is 27% ⁽⁶⁾.

In our study Out of 111 patients consumed yellow phosphorus, 45 (40.5%) patients died and 66 (59.5%) persons survived which is higher than other similar studies by Nalabotu et al, Pande et al and Fernandez et al.

Zinc Phosphide:

In Nalabothu M et al study, Out of 28 patients who were admitted with zinc phosphide poisoning 16 (57.1%) recovered, 10 (35.7%) died and 2 (7.14%) ⁽³⁰⁾.

A study done in Turkey by Mehnet tahir, Gokdemi et al showed a mortality rate of 28.3 %.

In our study a total of 56 patients consumed zinc phosphide, out of which 47 (83.92%) recovered without complication, 7 (12.5%) recovered with complication and 2 (3.5%) died.

Our study differs from others as the mortality rate are lower compared to studies done on zinc phosphide poisoning as quoted earlier which may be due higher toxicity in locally available zinc phosphide in the region where the study was undertaken.

In ChughSN, Aggarwal HK, Mahajan SK. et al study which included 20 patients with zinc phosphide poisoning, all patients had symptoms of vomiting and

abdominal pain, and 80% had palpitations, 75% had dyspnea, 40% presented with hypotension. 5 (25%) patients died in this study⁽³²⁾.

In our study 42.2 % persons had vomiting, 32.4% had abdominal pain, 9.8 % had shortness of breath at presentation. 2(4.3%) patients died.

Not all patients had Abdominal pain and vomiting in our study. This may be due to consumption of low doses of poison by patients admitted in our study as most patients took < 5 grams. Mortality rate is low when compared to chugh et al studies.

Superwarfarins:

A study conducted in Taiwan by Hsin – Ying Yu et al in 2013 on 2 patients, only 20 % had complications and had zero mortality⁽³⁵⁾.

In Nalabothu M et al study titled “Clinical profile and outcomes of rodenticide poisoning in tertiary care hospital “, out of 26 patients there was no mortality documented⁽³⁰⁾.

In our study 37 patients who consumed Superwarfarins, 31 (83.7%) had no complications, 6 (16.3%) had prolongation of INR and there was no mortality. This finding in our study is similar to other studies done on superwarfarins.

CONCLUSION

-The literature has only a few studies comparing the various types of rodenticide poisons.

-Among all the patients admitted with rodenticide poisoning, most common poison was yellow phosphorus (54.4%) followed zinc phosphide (27.5%) and Superwarfarins (18.1%) in that order.

-Females were more common than men in a ratio of 1: 1.14.

-Poisoning were more common in married persons.

- More than half of the patients admitted with rodenticide poisoning were below 29 years of age (65%).

- Patients presented within 6 hours of consumption had lower mortality rate (2.1%) when compared to presentation beyond 24 hours (72.3%).

-Superwarfarins are the least poisonous of the three types in this study as they have zero mortality.

- Yellow phosphorous has worst prognosis of the three poisons in this study as it has the mortality rate of 40.5% among yellow phosphorous poisoning and the highest mortality rate (95.7%) in overall rodenticide poisoning.

-Zinc phosphide and Superwarfarins poisoning has a very low complication rate (16.1 %) compared to yellow phosphorous.

- Mortality rate is low for amount of toxin consumed < 5 grams compared to doses beyond 5 grams.

- The most common symptom at presentation are Vomiting (42.2%), Abdominal pain (32.4%) and are followed by jaundice, altered sensorium, shortness of breath and bleeding manifestation in that order.

- In our study acute hepatitis and hepatic encephalopathy were seen only in yellow phosphorous poisoning.

- MELD score can be used as a prognostic indicator in yellow phosphorous poisoning.

- In majority of patients (71.6%) had a rise in AST and ALT between day 3 and 5, among patients who had a rise in liver enzymes.

LIMITATIONS OF THE STUDY

-Long term effects of these poison could not be assessed.

-The poison packet was not brought in most occasions and the quantity of poison is a rough estimate given by the attenders or the patient.

-In terms of treatment, all cases of yellow phosphorus poisoning are given N acetyl cysteine on admission, since being a tertiary care Centre, most cases admitted are referral from other peripheral and private hospitals and are admitted beyond 24 hours of consumption. Comparison between early and late administration of N acetyl cysteine could not be done.

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**CLINICAL PROFILE OF RODENTICIDE POISONING AND ITS OUTCOME IN
A TERTIARY CARE CENTRE"**

Name : _____ **Age :_yrs** _____
Sex:(Male/Female) Occupation:(Unskilled/semiskilled/skilled) _____
Education: _____

Address : _____ **Referral:** _____
Marital Status : (Married/Unmarried/Spouse deceased) **Socio - Economic Status:** _____
Date and time of consumption: _____ **Poison Container** _____
Brought:(Y/N) _____
Quantity of poison consumed : _____
Time from consumption to Stomach wash: _____
Patient received in Intubated state:(Y/N) _____
No of Days (Admission to Discharge/Death): _____

Present History:

- H/O Vomiting
- H/O Hematemesis
- H/O Abdominal pain
- H/O Diarrhea
- H/O Malena
- H/O Chest pain
- H/O Seizure
- H/O Altered mental status
- H/O difficulty in breathing
- H/O Petechial rash
- H/O other bleeding Manifestation
- H/O Body pain
- H/O Reduced urine output
- H/O Fever
- Others

Past History:

- H/o Similar complaints/Self Harm in the past (Y/N)
- H/o Hypertension, Diabetes, Asthma, Epilepsy, CAD, CLD, TB, CKD, Thyroid disease

Personal History:

- Diet:(Vegetarian/Mixed)
- H/O Drug intake ()
- H/O Smoking
- H/O Alcohol consumption:(Y/N) ;(If yes : _g/week,_ years)

Menstrual history:

Examination General:

GCS: _/15

(Pallor, Icterus, Clubbing, Cyanosis, Pedal edema, Lymphadenopathy)

Vital Signs:

	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8
BP (mm Hg)								
PR(/min)								
RR(/min)								
Spo2								

Systemic Examination:

- CVS : S₁ S₂ +()
- RS : B/L AE +,(No Added Sounds.)
- P/A: Soft ,No organomegaly, Tenderness()
- CNS:(PERL +)

CBC	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8
WBC (10 ³ /μL)								
Hb (g/dL)								
Hct (%)								
RBC (10 ⁶ /μL)								
Platelet(10 ³ /μL)								
MCV (fl)								
MCH (pg)								
Lymphocyte(%)								
Neutrophil(%)								
RDW-SD(fl)								
RDW-CV(%)								
MPV(fl)								

ABG				
pH				
pCO ₂ (mmHg)				
cHCO ₃ ⁻ (mmol/L)				
pO ₂ (mmHg)				
sO ₂ (%)				

LFT	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8
T.Bilirubin(mg/dl)								
D.Bilirubin(mg/dl)								
SGOT(IU/L)								
SGPT(IU/L)								
ALP(IU/L)								
T.Protien(g/dl)								
Albumin(g/dl)								
PT(sec)								
INR								

PSS:

MELD:

ECG: Rhythm-Rate-

Axis-

ST-T changes –

Others –

X RAY-

Epithelial cells			
Pus cells			
Protien			
Sugar			

URINE R/E:

RBC			
Casts			

USG Abdomen:

	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8
RBS(mg/dl)								
Urea(mg/dl)								
Creatinine(mg/dl)								
Na ⁺ (meq/L)								
K ⁺ (meq/L)								
CK(IU/L)								
CK-MB(IU/L)								

Specific Treatment:

1.NAC:

	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8	Total Dose
Route									
Dose(mg)									

2.Blood Products:

	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8
FFP								
Whole Blood								

3.Vitamin K

	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8
Route								
Dose								

4.Renal Replacement Therapy:

5.Plamapheresis:

6.Others

**INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003**

EC Reg No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.Arvind Kumar.V.
1 Year PG in M.D. General Medicine
Institute of Internal Medicine
Madras Medical College
Chennai

Dear Dr.Arvind Kumar.V,

The Institutional Ethics Committee has considered your request and approved your study titled **"CLINICAL PROFILE OF RODENTICIDE POISONING AND ITS OUTCOME IN A TERTIARY CARE CENTRE "** - NO.14042018

The following members of Ethics Committee were present in the meeting held on **03.04.2018** conducted at Madras Medical College, Chennai 3

1. Prof.P.V. Jayachandran	Chairman
2. Prof.R.Jayarathi.MD.,FRCP(Glasg) Dear MMC,Ch-3	Member
3. Prof.Sadha Seshayyan,MD., Vice Principal,MMC,Ch-3	Member Secretary
4. Prof.N. Gopalakrishnan MD,Director,Inst.of Nephrology,MMC,Ch	Member
5. Prof.S. Mayilvahanan,MD, Director,Inst. of Geriatrics,MMC, Ch-3	Member
6. Prof.A.Pandiya Raj,Director, Inst. of O.R.S Surgery,MMC	: Member
7. Prof.Shanthy Gunasingh, Director, Inst of Social Obstetrics,KGH	: Member
8. Prof.Rema Chandramohan, Prof.of Paediatrics,ICH,Chennai	: Member
9. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3	: Member
10.Prof.K.Rama Devi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3	: Member
11.Prof.Pharathi Vidya Jayarathi,Director, Inst. of Pathology,MMC,Ch-3	: Member
12. Mr. S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Mrs.Arnold Savitna, MA.,MSW.,	: Social Scientist
14.Thiru K.Ranjith, Ch- 91	: Lay Person

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

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


Member Secretary - Ethics Committee

Urkund Analysis Result

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6

CERTIFICATE - II

This is to certify that this dissertation work titled “**CLINICAL PROFILE OF RODENTICIDE POISONING AND ITS OUTCOME IN A TERTIARY CARE CENTRE**” of the candidate **Dr. ARVIND KUMAR .V** with registration Number **201711003** for the award of M.D. in the branch of **GENERAL MEDICINE**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **2percentage** of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

INFORMATION SHEET

TITLE: “CLINICAL PROFILE OF RODENTICIDE POISONING IN A TERTIARY CARE CENTRE”

Investigators : **Dr.ARVIND KUMAR.V**

Name of the Participant

Age:

Sex:

Study Setting :Institute of Internal Medicine,MMC&RGGGH, Chennai – 3.

You are invited to take part in this study. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns. We are conducting a study on “*CLINICAL PROFILE OF RODENTICIDE POISONING AND ITS OUTCOME IN A TERTIARY CARE CENTRE*”. This study will not affect your treatment. Investigationstaken drugs course of stay in hospital will be compared with other eligible candidates fitting the inclusion criteria. The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled. The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator

Signature of participant

Dr. ARVIND KUMAR.V

Date:

Date:

ஆய்வு தகவல் தாள்

ஆய்வு தலைப்பு:

"ஸி லெவீயின் மருத்துவ விவரம் மற்றும் லுள்ளம் திசை மருத்துவமான சிகிச்சையும் அதன் வெளிப்பாடும்"

ஆய்வாளர் பெயர் : மரு. V.அலித் சூபா

ஆய்வு திசையும் : பொது மருத்துவப் பிரிவு

சென்னை மருத்துவக் கல்லூரி, சென்னை-3.

இந்த ஆய்வில் தங்களை பங்கேற்ற அழைக்கிறோம். இந்த தகவல் அறிக்கையில் கூறப்பட்டிருக்கும் தகவல்கள் தங்கள் இந்த ஆராய்ச்சியில் பங்கேற்குமாறு வேண்டி மாற்றியமைக்கப்பட்டது. இந்த ஆய்வில் பங்கேற்றவர்கள் உடனடி தகவல்கள் மூலம் உடனடி சந்தேகங்களை நீக்கி தயாரிப்பைக் கொடுக்கலாம்.

நீங்கள் இந்த ஆராய்ச்சியில் பங்கேற்ற தகவல்கள் விருப்பப்படுகின்றன. முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களுடைய பெயரோ அல்லது அடையாளங்களோ வெளியிடப்படும் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்ற தகவல்களைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியில் இருந்து பின் வரக்கூடிய மாற்றியமைப்பை தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்புப் பரிசீலனையின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவில் தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம்

தேதி:

பங்கேற்றாளர் கையொப்பம்/

இடது கட்டை விகல் பெயர்

தேதி:

INFORMED CONSENT FORM

Title of the study: “***CLINICAL PROFILE OF RODENTICIDE POISONING AND ITS OUTCOME IN A TERTIARY CARE CENTRE***”

Name of the Participant :

Name of the Principal (Investigator): Dr. **ARVIND KUMAR.V**

Name of the Institution : Institute of Internal Medicine,
Rajiv Gandhi Govt. General Hospital, Chennai.

Documentation of the informed consent

I _____ have read it has been read for me, the information in this form. I was free to ask any questions and they have been answered. I am over 18 years of age and exercising my free power of choice, hereby give my consent to be included as a participant in the study.

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained in detail to me.
3. I have been explained about the nature of the study.
4. My rights and responsibilities have been explained to me by the investigator.
5. I agree to cooperate with the investigator and I will inform her immediately if I suffer from unusual symptoms.
6. I have not participated in any research study at any time.
7. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.

8. I hereby give permission to the investigators to release the information obtained from me as a result of participation in this study to the regulatory authorities, government agencies, and Institutional Ethics Committee. I understand that they are publicly presented.
9. My identity will be kept confidential if my data are publicly presented.
10. I am aware that if I have any question during this study, I should contact the investigator.

Participant's Initials: _____

For adult participants:

Name and signature / thumb impression of the participant (or legal representative if participant incompetent)

Name _____ Signature _____

Date _____

Name and Signature of impartial witness (required for illiterate patients):

Name _____ Signature _____

Date _____

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

Name _____ Signature _____

Date _____

ஆய்வு ஒப்புதல் படிவம்

ஆய்வு தலைப்பு

"எதி வெர்லியின் மருத்துவ விவரம் மற்றும் மூன்றாம் திசை மருத்துவமனை சிகிச்சையும் அதன் வெளிப்பாடும்"

பெயர் :

வயது :

பால் :

தேதி:

வெளிநோயாளி எண்:

ஆராய்ச்சி நோக்கம் எண்:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்துகொண்டு நான் எனது சம்பந்தத்தை தெரிவிக்கிறேன்.

நான் ஆராய்ச்சியாளருடன் ஒத்துழைப்பென் என்றும், எனக்கு ஏற்படக்கூடிய ஆசாதான நிகழ்வுகள் பற்றியும் உடனடியாக ஆராய்ச்சியாளரிடம் தெரிவிப்பென் என்று உறுதி கூறுகிறேன். இந்த ஆய்விலிருந்து எப்போது வேண்டுமானாலும் எக்காரணமும் கூறாமல் என்னை விடுவிர்த்துக் கொள்ளலாம் என்பதை அறியேன்.

என்னிடம் இருந்து பெறப்படும் தகவல்களை அரசு, மனநலநிறுவன அறிவிப்புகள் ஆகியவற்றின் பரிந்துரைகளை ஆராய்ச்சியாளருக்கு அனுப்பி அளிக்கிறேன். என்னுடைய சிகிச்சைக் கட்டுமான பார்வையிட உரிமை உண்டு. என்னுடைய தகவல்களின் அடையாளம் இரகசியமாக வைக்கப்படும் என்பதை அறியேன்.

இந்த ஆராய்ச்சியின் பங்கேற்க தன்னிச்சையாக முழு மனதுடன் சம்பந்திக்கிறேன்.

பங்கேற்பாளரின் கையொப்பம் / சேலை

பங்கேற்பாளர் பெயர்

இடம்:

தேதி:

ஆய்வாளர் கையொப்பம்

ஆய்வாளர் பெயர்

இடம்:

தேதி:

1.MARITAL STATUS

UM - UNMARRIED

M - MARRIED

2.TIME to ADMISSION

A - < 6 hours

B – 6 to 24 hours

C - > 24 hours

3.TYPE

YP – YELLOW PHOSPHOROUS

SW – SUPER WARFARIN

ZP – ZINC PHOSPHIDE

4.AMOUNT

A - <5 grams

B – 5 to 10 grams

C – 11 to 25 grams

D - > 25 grams

5.CLINICAL FEATURES

[Vomiting, AbdominalPain, Bleeding, Alteredsensorium, Others like
Breathlessness]

Y – YES

N – NO

6. BP,PR, UREA, CREATININE, WBC, TOTAL/DIRECT BILIRUBIN, BT,

CT, PT, aPTT

N – NORMAL

H - HIGH

L – LOW

7. P/A

N – NORMAL

A – ASCITES

B – TENDERNESS

8.CNS

N – NORMAL

A – FLAPPING TREMOR

B - UNCONSCIOUS

9.GCS

A – 14 and 15

B – 8 to 13

C - < 8

10.MAJOR COMPLICATIONS

N – NO COMPLICATIONS

A - ↑INR without BLEEDING

B - ↑INR with BLEEDING

C – ACUTE HEPATITIS

D – HEPATIC ENCEPALOPATHY

E – ACUTE KIDNEY INJURY

F – ARDS

G – HYPOGLYCEMIA

11.OUTCOME

A – RECOVERY WITHOUT COMPLICATIONS

B - RECOVERYWITH COMPLICATIONS

C – DEATH

12.RBS

1 - >90 mg/dl

2 – 55 to 90mg/dl

3 - <55 mg/dl

13.INR at day 3

A - <1.1

B - 1.1 to 2.99

C – 3 to 3.99

D - >4

14.AST and ALT

N – NORMAL

2,3,4,5,6 – DAYS AFTER CONSUMPTION IT STARTED RAISING

