A Dissertation on

A STUDY ON CLINICAL PROFILE OF RODENTICIDE POISONING AT GOVERNMENT STANLEY HOSPITAL, CHENNAI – 600 001.

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600032.

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for the Award of the Degree of

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
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CERTIFICATE

This is to certify that **Dr. G.ARUN KUMAR,** Post -Graduate Student (MAY 2010 TO APRIL 2013) in the Department of General Medicine STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on "**A STUDY ON CLINICAL PROFILE OF RODENTICIDE POISONING AT GOVERNMENT STANLEY HOSPITAL, CHENNAI – 600001"** under my guidance and supervision in partial fulfillment of the regulations laid down by the Tamilnadu Dr.M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in April 2013.

Dr.R.S.MURALIDHARAN, M.D;
Associate Professor (IMCU),
Department of Medicine,
Govt. Stanley Medical
College & Hospital,
Chennai – 600001.

Dr.P.VASANTHI, M.D
Professor,
Department of Medicine,
Govt. Stanley Medical
College & Hospital,
Chennai – 600001.

Dr.MAGESH KUMAR, M.D;
Professor and HOD
Department of Medicine,
Govt. Stanley Medical
College & Hospital,
Chennai – 600001.

Prof.Dr.S.GEETHALAKSHMI M.D, PhD;
Dean
Govt.Stanley Medical College & Hospital,
Chennai – 600001.

DECLARATION

I **Dr.G.ARUN KUMAR** declare that I carried out this work on "A STUDY ON CLINICAL PROFILE OF RODENTICIDE POISONING AT GOVERNMENT STANLEY HOSPITAL, CHENNAI - 600001" at the Toxicology unit of IMCU and Medical wards of Government Stanley Hospital during the period November 2011 to November 2012. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

This is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M. D. Degree examination in General Medicine.

DR.G.ARUNKUMAR

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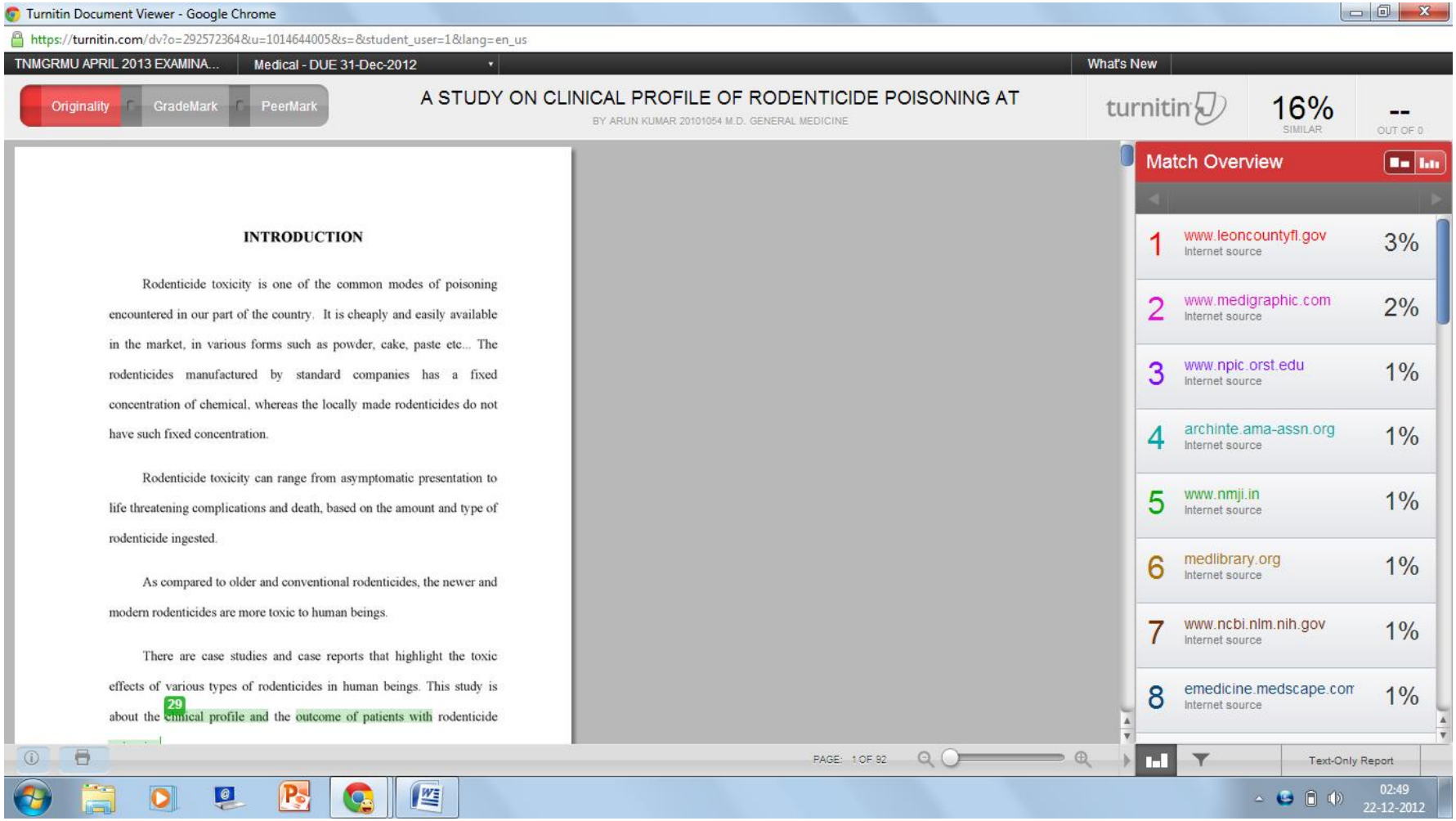
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INTRODUCTION
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Rodenticide toxicity can range from asymptomatic presentation to life threatening complications and death, based on the amount and type of rodenticide ingested.
As compared to older and conventional rodenticides, the newer and modern rodenticides are more toxic to human beings.
There are case studies and case reports that highlight the toxic effects of various types of rodenticides in human beings. This study is about the clinical profile and the outcome of patients with rodenticide
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PAGE: 1 OF 92
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CONTENTS

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There are case studies and case reports that highlight the toxic effects of various types of rodenticides in human beings. This study is about the clinical profile and the outcome of patients with rodenticide poisoning.

REVIEW OF LITERATURE

A rodenticide is any commercially available product that is designed to kill rats, mice, squirrels and other small rodents. Rodenticides includes various compounds ranging from highly toxic compounds requiring just a single dose ingestion like zinc phosphide, yellow phosphorus, sodium monofluroacetate, fluroacetamide, arsenic, thallium to less toxic compounds requiring repeated ingestion over a period of time like warfarins, and scilliroside.

Figure - 1 Commercially available Rodenticides

Local made **Company made**

Since the rodenticides are agents that are specifically designed to kill mammals, its toxic effects are very similar both against rodents and human beings. As the rodents have developed resistance to the existing rodenticides there is a necessity for development of newer and more toxic rodenticides. As the rodents are resistant to warfarin baits, the

development of superwarfarins has posed a higher risk to human beings [1, 2]. Rodenticide toxicity in humans occurs either as a result of accidental ingestion or as a result of suicidal intent.

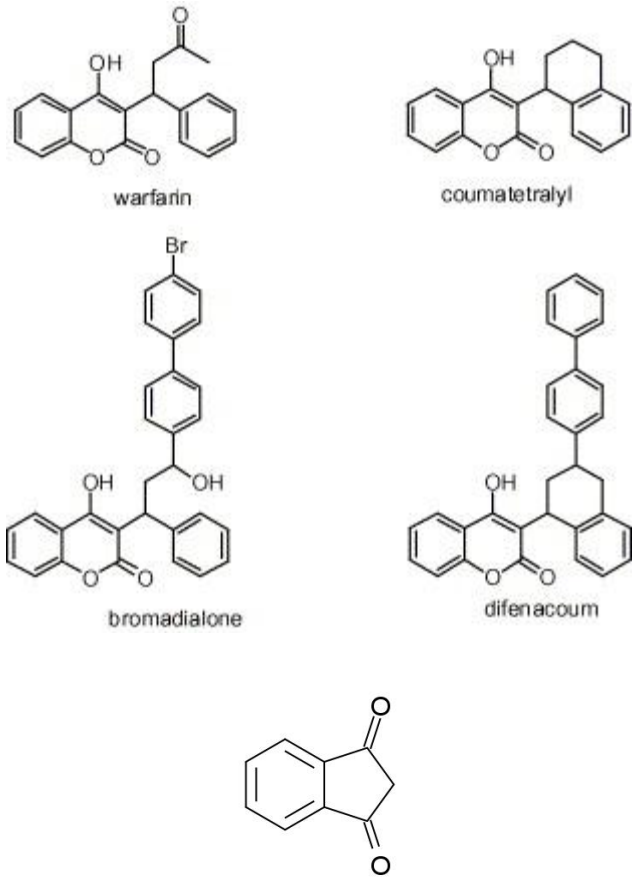
Table - 1CLASSIFICATION OF RODENTICIDES

S.NO	CLASS	EXAMPLE
1	Coumarins	First generation : Warfarin, Coumachlor, Coumafuryl, Coumatetralyl, Dicoumarol Second generation : Brodifacoum, Bromadiolone, Difenacoum, Difethialone Flocoumafen
2	Indandione	Chlorophacinone, Diphacinone, Pindone, Valone
3	Inorganic	Zinc phosphide, Arsenous oxide, Yellow phosphorus, Potassium arsenite, Sodium arsenite, Thallium sulphate
4	Organochlorides	Gamma-HCH, HCH, Lindane
5	Organophosphorus	Phosacetim
6	Organofluorides	Fluoroacetamide, Sodium fluoroacetate
7	Pyrimidinamine	Crimidine
8	Urea	Pyrinuron
9	Thiourea	Promurit, Thiosemicarbazide
10	Carbanilate	3-Pyridylmethyl 4- nitro carbanilate
11	Botanical	Scilliroside, Strychnine
12	Unclassified	Bromethalin, Chloralose, tetramine Alpha-chlorohydrin, Cholecalciferol, Flupropadine, Hydrogen cyanide, Norbormide, Silatrane,

ANTICOAGULANT RODENTICIDES

Anticoagulant rodenticides mainly comprises of Coumarins (4-hydroxycoumarin) and 1,3-Indandiones.Warfarin, a 1st generation coumarin is an active ingredient present in most of the commercially available rodenticides. Due to their shorter half-life they are required in a much higher concentration (<0.1%) than 2nd generation coumarins (0.001% - 0.005%) to exert their desired anticoagulant effect [3].

Figure – 2 Structure of Coumarins and Indandiones



1,3Indandione

To achieve a particular anticoagulant effect, 1st generation compounds require multiple dosing when compared to second generation compounds which require just a single dose. As the second generation compounds are highly toxic they are commonly referred as **"Super Warfarins"** [4]. These compounds are easily absorbed from the gastrointestinal tract and rarely across the skin.

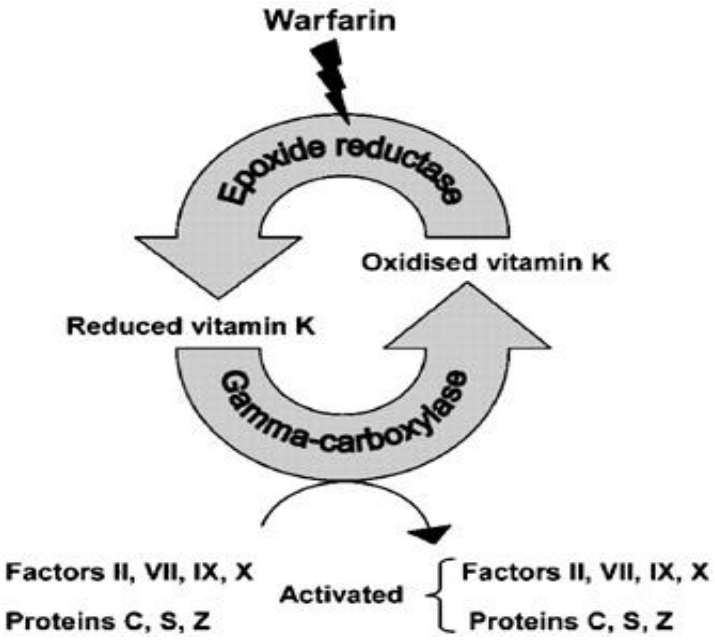
Bromadiolone and Brodifacoum are commercially available in cake and powder form. It is commonly available at a concentration of 0.005 % wax cake (Bromadiolone). However locally manufactured powders do not have a fixed concentration of the active ingredient present in them.

Figure - 3 Bromadiolone in cake and powder form

Mechanism of action:

Vitamin K dependent clotting factors (2, 7, 9, and 10) are the main targets of these anticoagulant rodenticides. These compounds inhibit vitamin K1 -2,3 epoxide reductase, thereby preventing vitamin K to get converted to its active state. Warfarin mainly targets prothrombin and factor X. Hence the measurement of prothrombin time is used to assess the severity of warfarin toxicity.

Figure – 4 Warfarin - Site of action



The prolonged duration of action of the 2nd generation compounds (superwarfarins) is due to the following reasons [5]:

1. It has a high affinity for vitamin K1-2,3-epoxide reductase,

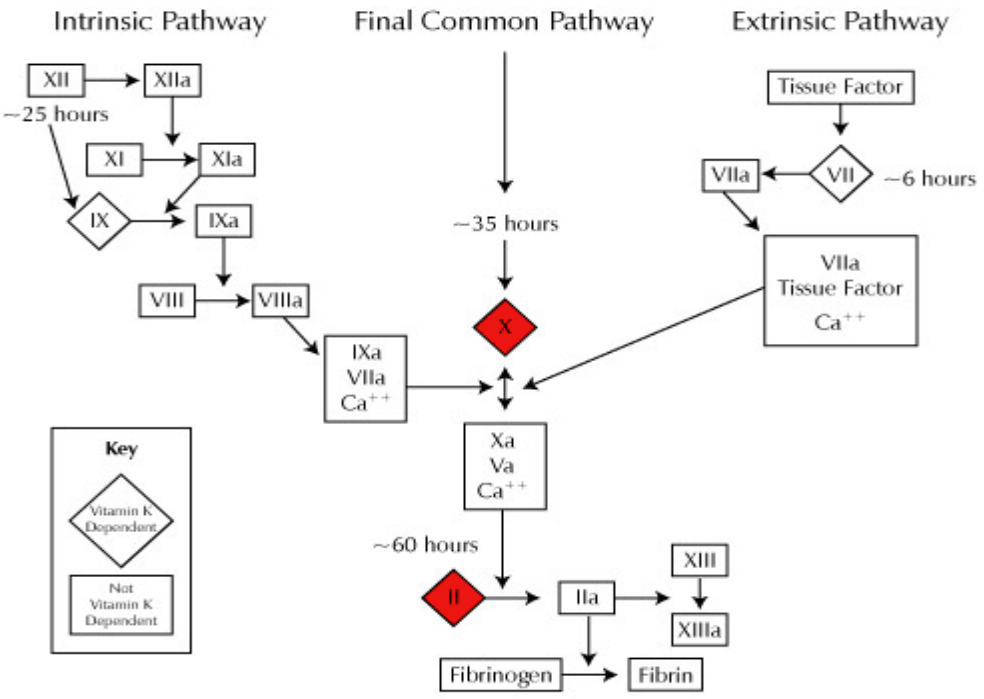
2. It accumulates more in the liver,

3. It targets the Vitamin K – Epoxide cycle at multiple levels, and

4. It has a prolonged half-life.

These agents also increase the capillary permeability and thereby causing haemorrhage inside our body. Haemorrhage due to warfarin toxicity occurs few days after poisoning as the clotting factors 2, 7, 9, and 10 have a longer half-life [1, 2]. But the second generation compounds can cause lethal haemorrhage earlier even with low doses [1].

Figure – 5 Coagulation cascade



Coagulation cascade and vitamin K–dependent clotting factor half-lives.
Shaded clotting factors are critical warfarin targets.

After the ingestion of toxic doses of warfarin or superwarfarin, the prolongation of prothrombin time can occur within a day, but the time for maximum prolongation may take upto 3 – 4 days [1, 6, 7]. At low doses, the only manifestation may be prolonged prothrombin time instead of haemorrhage.

Toxicity:

The toxic dose for superwarfarins in humans has not been established. But, as low as 1 milligram of brodifacoum, a second generation coumarin has produced significant toxicity in adults [1]. Larger single doses with suicidal intent have resulted in significant symptoms due to the longer half-life of superwarfarins [2].

Clinical features:

Haemorrhagic manifestations like epistaxis, gum bleed, upper GI bleed, subconjunctival haemorrhage and petechiae are commonly seen[1, 2, 8, 10]. In case of severe poisoning, shock and even death can occur.

Some indandiones exhibit signs of cardiopulmonary and neurologic insult in rodents causing death before haemorrhage occurs. Such actions occur only in rodents and have not yet been reported in human beings.

Management:

1. Gastric Lavage:

Gastric lavage is done using a nasogastric tube if the patient presents within 1 hour of ingestion. Lavage performed after one hour of ingestion has no proven benefits.

2. Activated charcoal:

Activated charcoal effectively absorbs the ingested rodenticide if given within 1 hour of ingestion [4]. It is administered at a dose of 25 to 100 grams mixed with 400-800 ml of water.

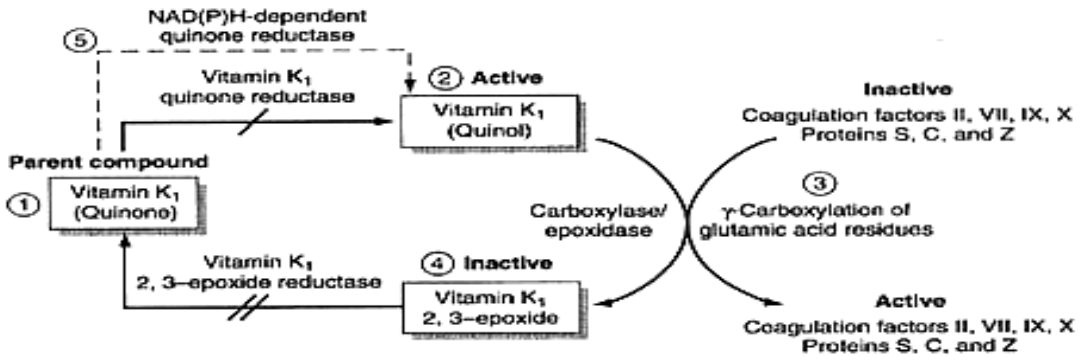
3. Vitamin K1:

There is a decrease in plasma prothrombin levels within 24-48 hours of poisoning and this may persist for at least one to three weeks. Due to this reduced plasma prothrombin concentration there is an increase in prothrombin time [1, 6, 7]. Patients who present earlier after ingestion (< 24 hrs) will have a normal prothrombin time. But in many patients who had a normal prothrombin time at 24 hours had a prolonged prothrombin time at 48 hours [7].

In cases of accidental ingestions involving a very small amount, medical management is usually not required, but those patients must be observed for any bleeding manifestations.

In cases of suicidal ingestion of unknown or relatively large amounts, phytonadione (vitamin Kl) is administered orally to protect the patient against the anticoagulant effect of these rodenticides. Phytonadione administration has no potential adverse effect on the patient.

Figure –6 Action of Vitamin K1



In cases where larger amount has been ingested, phytonadione therapy should be started along with monitoring of prothrombin time serially at 24 and 48 hours. Patients with increased prothrombin time or bleeding manifestations should be treated only with vitamin K1 and not with vitamin K3 or vitamin K4. For adults and children over 12 years the dose of vitamin K1 is fifteen to twenty-five mg.

If the prothrombin time is significantly prolonged, phytonadione should be administered intramuscularly at a dose of 5 – 10 mg. Upto 125 mg of phytonadione may be needed to treat patients with brodifacoum poisoning who had persistently elevated prothrombin time despite regular treatment [8, 9, 11]. Phytonadione dose must be repeated if the prothrombin time has not decreased from the original value since 24 hours.

Patients who had consumed a very large quantity of superwarfarin compounds will have decreased or low level of prothrombin activity for a very long period and hence those patients may require treatment for as long as three to four months [8, 9].

If the patient bleeds as a result of anticoagulant poisoning, phytonadione must be administered intravenously at a higher dose (10 mg). Phytonadione is diluted in normal saline or dextrose containing fluids and administered as intravenous infusion. The rate of infusion should not be too rapid. The response is usually seen within 3-6 hours. In cases of superwarfarin toxicity subsequent dosages should be adjusted based on response to therapy [8, 9, 11].

In cases of severe bleeding fresh blood or fresh frozen plasma should be supplemented along with phytonadione and other supportive measures. Prothrombin time and haemoglobin levels are checked every six to twelve hours to monitor the effectiveness of the treatment.

INORGANIC RODENTICIDES

METAL PHOSPHIDE:

There are different types of metal phosphides used in the manufacturing of rodenticides. Single dose poisoning can be lethal when ingested in sufficient amounts.

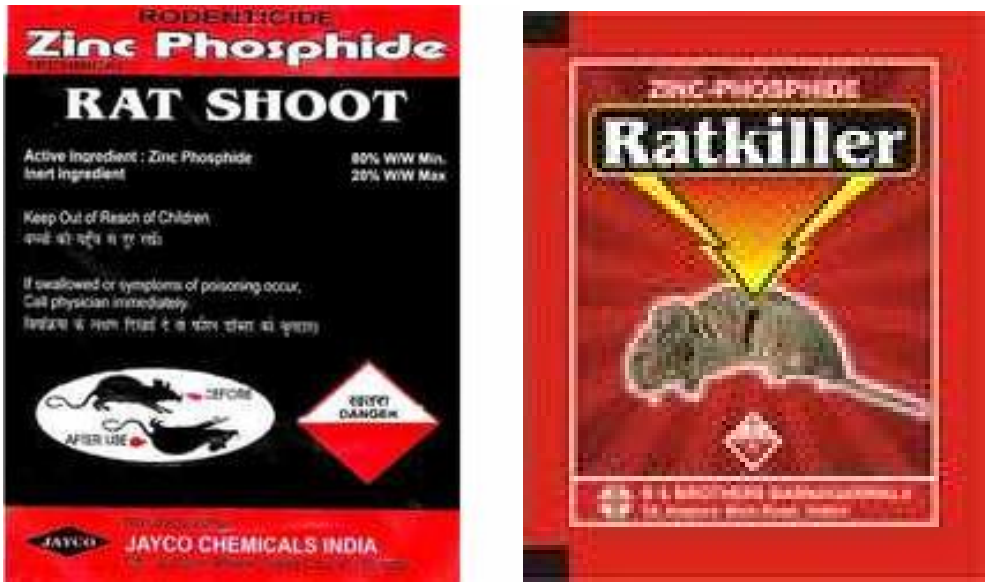
Table – 2 Physical appearance and lethal dose of various metal phosphides

METAL PHOSPHIDE	PHYSICAL APPEARANCE	LETHAL DOSE
Aluminium	Yellow or dark grey crystals	20 mg/kg
Zinc	Grey tetragonal crystals/grey black powder	4–5 g (Toxic dose)
Magnesium	Yellow-green crystals	10.4 mg/kg
Calcium	Red-brown crystals/grey lumps	8.7 mg/kg

Of the above mentioned metal phosphides, only zinc phosphide is used in baits. Other phosphides are used as fumigant rodenticide.

Zinc phosphide is a grey-black coloured compound available in powdered form. It has garlic like odour [12]. Its toxicity is primarily due to its hydrolysed product called phosphine, a colourless and flammable gas [12, 13, 14].

Figure – 7 Commercially available zinc phosphide powder



Zinc phosphide is used as baits at a concentration of 0.75 – 2 %. In the rodenticide formulation along with 75 % of zinc phosphide, 25 % of antimony potassium tartrate is added as an emetic. This is added to induce vomiting if it is accidentally ingested by human beings.

Zinc phosphide requires acidic PH to get hydrolysed and liberate phosphine, whereas other metal phosphides hydrolyse at a neutral pH [15].

The toxic dose of aluminium phosphide for humans is considered to be 150 – 500 mg. Serum phosphine levels of more than 1.6 milligram / dl are considered to have a high mortality rate.

Mechanism of action:

The exact mechanism of action is not clear but the following mechanisms have been hypothesized

1. It inhibits Cytochrome C oxidase
2. It inhibits oxidative respiration
3. It blocks the protein and enzyme synthesis[16]and thereby blocks mitochondrial respiration
4. It produces of free radicals that inhibits the catalase and peroxidase and also causes lipid peroxidation
5. It has a corrosive effect on the exposed tissues
6. Toxicity due to zinc, magnesium or aluminium [17, 18, 19]
7. Phosphine also exerts an anticholinesterase activity [18]
8. Impairment of glycogenolysis and gluconeogenesis[20, 21]

Clinical features:

Zinc phosphide is much less corrosive to skin and mucous membranes, but on inhalation it can cause pulmonary oedema. The emetic effect of zinc and antimony potassium tartrate may provide protection; however, patients develop excitement, chills, chest tightness, breathlessness and even pulmonary oedema.

At toxic dose it can cause hepatic failure, renal failure, tetany, and arrhythmias which can be fatal [21, 22].

TREATMENT

The treatment of metal phosphide poisoning is mainly supportive. Securing of airway and controlling the seizures (if present) should be given priority over gastrointestinal decontamination.

Metal phosphides react with the acidic contents in the stomach and liberate phosphine gas. This toxic gas is present in the vomitus, lavage fluid, and the faeces of the patient and hence care should be taken to avoid inhalation of the toxic gas.

Test for phosphine gas [23]:

The gastric lavage fluid (5 ml) is diluted with 15 ml of water and heated in a container for fifteen to twenty minutes at 50 degree Celsius. The mouth of the container should have two filter papers coated with 0.1 N silver nitrate and 0.1 N lead acetate.

If phosphine gas is present, it will turn the silver nitrate paper black due to the formation of metallic silver and the other paper will not have a change in colour. If both the papers turn black, it signifies the presence of hydrogen sulphide.

1. GASTRIC LAVAGE:

Gastrointestinal decontamination is effective in the patients who present to the hospital within 1 hour of ingestion. Lavage performed after 1 hour of ingestion has no proven benefits.

2. SUPPORTIVE MANAGEMENT:

Vital signs must be monitored periodically. Blood sugar, electrolytes and pH are constantly monitored and treated accordingly with

appropriate IV fluids. Patients can develop acidosis, which should be treated promptly by appropriate IV fluids.

3. OXYGEN:

Low concentration oxygen is given by nasal mask. Pulmonary oedema is managed with intermittent or continuous positive pressure ventilation. Oxygen should not be used at a higher concentration for prolonged periods, because it may add on to the lung injury.

4. MAGNESIUM SULPHATE:

Patients with zinc or aluminium phosphide toxicity, when treated with magnesium sulphate have lesser complications and more favourable outcome [21, 22, 25]. Although the exact mechanism is not clearly understood, it may be due to the special property that magnesium stabilizes the cell membrane and prevents the occurrence of life threatening arrhythmias.

Moreover intracellular magnesium is used as a cofactor in the synthesis of glutathione and other antioxidants [26]. Hence intravenous

magnesium sulphate may be used in the setting of acute metal phosphide poisoning.

Dose of magnesium sulphate

Three grams IV infusion is given over a period of 3 hours, followed by six grams per day for the next three to five days [22].

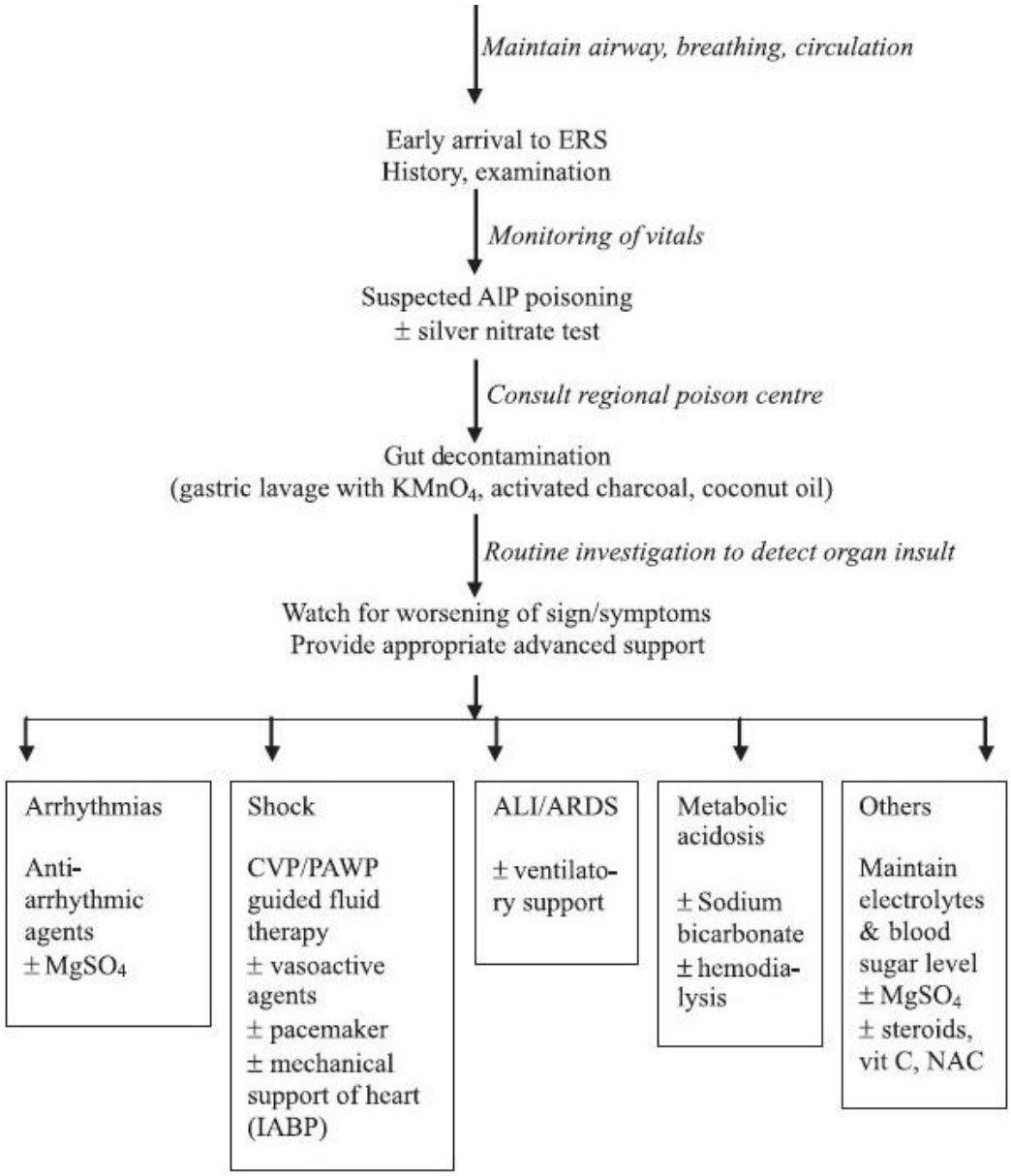
Extracorporeal haemodialysis is done if acute kidney injury occurs.

Serum ALP, LDH, Transaminases, prothrombin time, and bilirubin are monitored for hepatic involvement. Vitamin K1 is given if prothrombin level declines.

N – Acetyl cysteine [27] and coconut oil has been used with some success in the treatment of metal phosphide poisoning [28].

The following protocol can be used in the emergency management of phosphine or metal phosphide poisoning [29].

Figure – 8 Protocol for management of Metal phosphide poisoning



Note: CVP = central venous pressure, PAWP = pulmonary artery wedge pressure, IABP = intra-aortic balloon pump, ECLS = extra corporeal life support, ALI/ARDS = acute lung injury/acute respiratory distress syndrome, NAC = n-acetylcysteine

YELLOW PHOSPHORUS

Yellow phosphorus is a highly toxic inorganic element that is commonly used in crackers, ammunition, fertilizers and rodenticide. It has a garlic like odour [30] and also called as “White Phosphorus”

As there is an emerging resistance to conventional rodenticides, yellow phosphorus is now being used as rodenticide in form of a paste. Since it comes in a paste formulation there is an increased chance of accidental poisoning particularly among the children [31].

Figure – 9 Commercially available yellow phosphorus

TOXICITY:

Intoxication occurs with suicidal or accidental ingestion. It is easily absorbed from the gastrointestinal tract and from other mucosal surfaces and even through skin [30]. It is evenly distributed and concentrated in many tissues in our body, particularly the liver [32, 33] and the peak level is reached after 2 to 3 hours of ingestion. The toxic dose is found to be 15

mg and more than 50 mg is considered as a fatal dose (1 mg / kg on the average) [34].

MECHANISM OF ACTION:

It is a protoplasmic toxin causing multiorgan failure particularly cardiac, hepatic, renal toxicity. Its toxic effect is mainly due to the alterations that occur in the ribosomal function leading to altered protein synthesis [35]. It also causes dysregulation of blood sugar & glycogen deposits [36].

It also affects the lipoprotein synthesis & secretion of tri-glycerides, causing fatty degeneration of multiple organs [34, 37, 38].

CLINICAL FEATURES:

Patients with yellow phosphorus intoxication pass through 3 stages.

Table – 3 Stages of Yellow Phosphorus poisoning

S.no	Stage	Time since poisoning
1	Stage of General symptoms	0 – 24 hours
2	Asymptomatic stage	24 – 72 hours
3	Advanced stage	> 72 hours

Stage of general symptoms:

This stage is predominantly characterised by gastrointestinal symptoms

1. Abdominal pain,
2. Nausea & vomiting,
3. loose stools and
4. Fever.

Apart from these clinical symptoms, the patients do not have any abnormalities that can be detected by means of lab investigations.

Asymptomatic stage:

The patient seems to be well preserved and has no symptoms during this stage. But there will a mild elevation of bilirubin, hepatic transaminase (SGOT, SGPT) with demonstrable histological changes suggestive of toxic hepatitis. This is a stage where the patient may be discharged from the hospital as the patient is symptom free.

Advanced stage:

This stage occurs after 3 days of poisoning and lasts until recovery or death of the patient [34, 36, 37, 39, 40].

Table –4 Clinical features in Advanced stage

S.no	System	Clinical features
1	Cardiovascular	Hypotension, Tachycardia, Arrhythmias, Cardiogenic shock
2	Gastrointestinal	Acute fulminant hepatitis, Hepatic encephalopathy, Coagulopathy. (liver histology shows steatohepatitis and necrosis)
3	Central nervous system	Irritability, Confusion, Hallucinations, Psychosis, Coma
4	Renal	Acute tubular necrosis, Anuric renal failure

Depending on the amount of yellow phosphorus ingested, some patients recover from the initial phase of insult spontaneously. The reason for spontaneous recovery is unknown [35]. Some patients develop oliguria and acute tubular necrosis and eventually land up in renal failure.

MANAGEMENT:

1. **SKIN DECONTAMINATION** As yellow phosphorus can be absorbed across the skin; all the particles must be cleanly washed off with water.

2. **SUPPORTIVE MANAGEMENT:** Airway securing and seizure control must be done prior to gastric lavage and catharsis.

3. **GASTRIC LAVAGE AND CATHARSIS:** Gastric lavage with 0.01-0.1% KMO_4 (potassium permanganate) or 0.2-0.4% $CuSO_4$(copper sulphate) solution is done to convert the phosphorus to relatively harmless oxides which should then be followed by administration of activated charcoal adsorbent and 30 minutes later by a saline cathartic.

Fat favours additional absorption of phosphorus, hence patients must avoid fat in diet for 3-4 days or longer. Mineral oil dissolves phosphorus and prevents its absorption and hence it can be taken orally [41]. It is usually given at a dose of 1.5 ml per Kg body weight. There is no specific antidote for yellow phosphorus.

Close monitoring of hepatic and renal function must be done and managed accordingly. Liver transplantation has been successfully done in suitable candidates for acute hepatic failure [42].

Yellow phosphorus ingestion carries a grave prognosis unless treatment is started early. Fernandez and Canizares in a case series of 15 patients have reported a mortality rate of 27%, confirming that yellow phosphorus is extremely lethal when ingested [40].The mortality rate may even be as high as 50% [1].

Liver transplantation should be considered in suitable patients. Patients for liver transplantation are selected as per “Criteria of King's College, London”.

Table-- 5Criteria for Liver Transplantation in Acute Liver Failure

Criteria of King's College, London
Acetaminophen cases Arterial pH <7.3, or INR >6.5 and serum creatinine >3.4 mg/dL Non-acetaminophen cases INR >6.5, or Any three of the following: Age <10 years or >40 years Duration of jaundice before encephalopathy >7 days Etiology: non-A, non-B hepatitis; halothane hepatitis; idiosyncratic drug reaction; indeterminate Serum bilirubin >17.6 mg/dL INR >3.5 (PT >50 seconds)

THALLIUM SULPHATE

Besides used in the treatment of TB, syphilis and gonorrhoea in the past, it was also used as a rodenticide. But now, it is banned in US because of many human deaths due to poisoning [43]. Because it is odourless and tasteless it is still being used as a rodenticide and ant killer in many developing countries.

Thallium sulphate is readily absorbed from the gastrointestinal system and across the skin. It has a high volume of distribution (3.6-5.6 L/kg). Most intravascular thallium is present in RBC. Half-life in humans is about 1.9 days. Its LD50 in humans is 10 - 15 mg/kg. [44].

MECHANISM OF ACTION

Table – 6 Mechanism of action of Thallium [45]

1	Ribosomal inhibition
2	Disruption of potassium-dependent processes
3	Riboflavin sequestration
4	Interference with the cysteine residues
5	Injury to the Myelin sheath

TOXICITY

Its lethal dose is considered to be fifteen to twenty mg per kg body weight. But even lesser doses have caused death. It has a varied clinical manifestations due to its 3-phase toxicokinetics.

Table - 7 Three phase toxicokinetics

Phase 1	Intravascular distribution	< 4 hours	Distributed to organs having high perfusion (kidney, liver, muscle)
Phase 2	CNS distribution	4-48 hours	Distribution into the CNS
Phase 3	Elimination	> 24 hours	Elimination process starts

Thallium is excreted mainly through faeces (51.4%) and urine (26.4%).Because of prolonged elimination half-life of 3-30 days; it also acts as a cumulative poison.

The best method for diagnosis is estimation of twenty four hours urinary thallium excretion, which is normally less than 10 mcg/L. Values more than this are corroborative with thallium poisoning [44, 46].

CLINICAL FEATURES

Early symptoms that occur after acute toxicity are predominantly gastrointestinal symptoms like

Abdominal pain,

Nausea & vomiting,

Loose stools,

Stomatitis,

Increased salivation,

Ileus.

Hepatic involvement is evidenced by increased transaminases. Other symptoms which can occur late are headache, muscle weakness, lethargy, paresthesias, tremor, ataxia and ptosis [44, 47].

Myoclonic movements, convulsions, delirium, and coma occur in case of severe poisoning. Fever is considered as a poor prognostic sign of CNS involvement. Alopecia can occur after two weeks or more and is therefore not helpful in diagnosing at an earlier stage [44, 48].

Cardiovascular effects include early hypotension, due to toxic cardiomyopathy. Ventricular arrhythmias can occur. Hypertension can

occur as a late manifestation due to vasoconstriction. It can also lead to Acute Respiratory Distress Syndrome [49].

Death occurs primarily due to respiratory paralysis or cardiovascular collapse.

TREATMENT

1. Gastric Lavage:

Gastrointestinal decontamination is effective in patients if done within one hour of ingestion. Lavage performed after one hour of ingestion has no proven benefits.

2. Activated charcoal:

Activated charcoal effectively absorbs the thallium if given within one hour of ingestion [8]. It is administered at a dose of 25 to 100 grams mixed with 400-800 ml of water. Multiple doses of activated charcoal can increase the elimination thallium [46].

3. Supportive management:

Electrolyte and dextrose should be administered by IV infusion to increase diuresis, thereby enhancing urinary excretion of thallium.

Patients must be carefully monitored for development of arrhythmias.

4. Haemodialysis and haemoperfusion:

It has been effective in patients with severe poisoning by decreasing the total body content of thallium.

5. Potassium ferric ferrocyanide:

Also known as Prussian blue, it increases thallium excretion via faeces.

STRYCHNINE

It is a naturally occurring alkaloid commonly known as nux vomica. It is rarely used in the manufacture of rodenticides.

MECHANISM OF ACTION

It selectively blocks the post synaptic inhibitive neurons and thereby causing an excitatory state among the cells of central nervous system, mainly the spinal cord and medulla. Respiratory arrest either primarily or secondary to convulsion is the common mode of death.

Strychnine is rapidly absorbed from the gastrointestinal system. It is rapidly cleared off the blood stream due its low plasma protein binding property. It is metabolised by the liver and excreted by the kidneys. Lethal dose of strychnine is found to be fifty to hundred mg [50].

TREATMENT

1. Early gastrointestinal decontamination with activated charcoal and potassium permanganate.

2. There is no specific antidote for strychnine poisoning.

3. Anticonvulsants are used for controlling seizures.

4. Acidification of urine can be done to increase the urinary excretion of strychnine.

CRIMIDINE

It is a synthetic rodenticide [2-chloro-4-(dimethylamino)-6-methylpyrimidine]. It is a highly toxic compound with a lethal dose of 5 mg/kg. Within thirty minutes of ingestion the physical signs and symptoms starts to appear.

Clinical features include restlessness, involuntary movements, seizures, muscle spasm. If respiratory muscles are involved, patient will develop respiratory arrest, cyanosis and eventually death [51].

TREATMENT

1. Seizure control with anticonvulsants. Along with anticonvulsants, pyridoxine can also be used to control seizures.

2. Early gastric lavage with activated charcoal.

3. Administration of sodium bicarbonate along with IV fluids to enhance excretion of absorbed toxin and to combat metabolic acidosis that can occur as a result of convulsions.

SODIUM FLUOROACETATE AND FLUOROACETAMIDE

It is easily absorbed from the gastrointestinal system,

Mechanism of action

In the liver it is converted into flurocitrate which is a potent inhibitor of tricarboxylic acid cycle and thereby inhibiting the cellular inhibition

Toxic features

The organs most commonly affected are the heart, brain, and the kidneys which are manifested as arrhythmias, convulsions [52], metabolic acidosis and electrolyte imbalance.

TREATMENT:

1. **Gastric lavage**:

It is effective only when it is done within one hour of poisoning. However if the patient is having active seizure, it must be controlled first.

2. **Seizure control:**

For organochloride compounds, diazepam is still preferred over lorazepam as there is no strong evidence reported in the literature. It is always advisable to secure the airway and start on mechanical ventilation to prevent aspiration and related complications.

3. Hypocalcaemia:

For hypocalcaemia10% calcium gluconate solution is given as IV at a dose of 10 ml (100 mg/ml). ECG must be monitored for arrhythmias and, if present, it should be treated with appropriate antiarrhythmic drug

SCILLIROSIDE

It is a naturally available compound that is used in manufacturing botanical rodenticides. It is poorly absorbed across the gastrointestinal tract. This is usually less toxic to humans when compared to other rodenticides.

MECHANISM OF ACTION

Its toxic properties are mainly due to cardiac glycosides. It produces alteration in the conduction properties of heart and may produce arrhythmias.

TREATMENT

Scilliroside is less likely to cause toxicity unless it is consumed at large dosage because of its intense emetic effect. Only supportive and symptomatic treatment is given. It includes

1. Gastrointestinal decontamination,
2. Activated charcoal,
3. Cardiac monitoring,
4. Other supportive measures.

CHOLECALCIFEROL

Cholecalciferol (vitamin D3) exerts its toxic effects due to hypercalcemia. Cholecalciferol intoxication produces an elevated serum level of calcium, chiefly the unbound fraction.

Clinical features

Signs and symptoms of hypercalcemia are fatigue, weakness, headache, and nausea. Features that suggest renal involvement include polyuria, polydipsia, proteinuria, and azotemia.

TREATMENT

Treatment of cholecalciferol toxicity is mainly focussed on

1. To limit gastrointestinal absorption,
2. Enhance excretion,
3. To counteract the hypercalcemic effect.

1. Gastric Lavage:

Gastric is usually effective when done within one hour of poisoning. Lavage performed after 1 hour of ingestion has no proven benefits.

2. Activated charcoal:

Activated charcoal is given at a dose of 25-100 g in 300-800 mL repeated two to four hours.

3. IV fluids

Normal saline or dextrose solutions are used to enhance its excretion. Blood calcium levels should be constantly monitored. Urinalysis is done to assess the extent of renal injury.

4. Furosemide

20-40 mg IV or 40-120 mg orally shall be used to enhance diuresis.

5. Predinisone and other glucocorticoids are used to decrease the elevated blood calcium levels at a dose of one mg per kg per day.

6. Calcitonin can be used at a dose of 4 IU/ Kg twelfth hourly for two to five days.

AIMS AND OBJECTIVES

1. To study the clinical profile of patients admitted with rodenticide poisoning at IMCU, Govt. Stanley hospital, Chennai.

2. To assess the morbidity, mortality and clinical outcome of those patients.

MATERIALS AND METHODS

PLACE OF STUDY:

This study has been carried out at the toxicology unit of Intensive Medical Care Unit and Medical wards of Govt. Stanley hospital, Chennai.

STUDY PERIOD:

One year (From November 2011 to November 2012).

STUDY DESIGN:

This is a Retrospective and Prospective observational study.

ETHICAL COMMITTEE APPROVAL:

The ethical committee approval was obtained for this study.

INCLUSION CRITERIA:

1. Any patient admitted with rodenticide intake.

2. Age of the patients > 13 years

EXCLUSION CRITERIA:

1. Mixed poisons,

2. Chronic liver disease,

3. Alcohol intake within 24 hours before admission

4. Patients on drugs like anticoagulants, antiplatelets,

5. Patients with bleeding disorders,

6. Acute diarrhoeal disease,

7. Patients with known coronary artery disease

CONSENT:

The study group thus identified by the above criteria (inclusion and exclusion criteria) was first instructed about the nature of the study. Willing participants were taken up for this study after getting a written / informed consent from these patients or their relatives in the local vernacular language.

STUDY SUBJECTS:

All the patients who fulfilled the inclusion criteria above 13 years of age and both genders were included in this study. The included patients were subjected to detailed history taking, complete physical examination and the relevant laboratory investigations.

The details of the type and the amount of rodenticide ingested were collected from the patients or their relatives.

DEFINITIONS AND REFERENCE LAB VALUES USED IN THE PRESENT STUDY:

Acute hepatitis[53]: Acute onset of inflammation of liver which is evident clinically as low-grade fever, anorexia, nausea, vomiting, fatigue, malaise, dark urine, jaundice, hepatomegaly with or without splenomegaly or lymphadenopathy. It is diagnosed by either an increase in serum bilirubin along with elevated transaminases (SGOT & SGPT) or by means of liver biopsy.

Acute liver failure [53]: Acute liver failure (or fulminant hepatic failure) originally was defined by an interval of eight weeks or less between the onset of illness and appearance of encephalopathy

Hepatic encephalopathy[53]: Reversible neurologic and psychiatric manifestations usually found in patients with chronic liver disease and portal hypertension, but also seen in patients with acute liver failure.

Myocarditis: Signs of myocardial inflammation evidenced by echocardiogram and elevated cardiac enzymes (Troponin I and T).

ARDS (Acute respiratory distress syndrome)[54]: It is a clinical syndrome of sudden onset severe dyspnoea, hypoxemia, and diffuse pulmonary infiltrates leading to respiratory failure. It is diagnosed by arterial PaO2/FIO2 > 200 mmHg, presence of bilateral alveolar or interstitial infiltrates in chest X – ray and PCWP less than 18 mmHg or no clinical evidence of increased left atrial pressure

AKI (Acute Kidney Injury)[54]: An increase in the blood urea nitrogen concentration and/or an increase in the plasma or serum creatinine concentration, often associated with a reduction in urine volume.

Hypoglycaemia [54]: Glucose levels <55 mg/dl

RFT

Blood sugar – 90-140 mg/dl

Urea – 20-40 mg/dl

Serum Creatinine - 0.5–1.2 mg/dl(males), 0.4 – 1.1 mg/dl (females)

Sodium – 135 – 145 mEq/l

Potassium – 3.5 – 5.1 mEq/l

Bicarbonate – 22- 28 mEq/l

Chloride – 98 - 108 mEq/l

LFT[53]: Serum Total bilirubin - 1.0 and 1.5 mg/dl

Serum indirect bilirubin - 0.8 and 1.2 mg/dl

Serum Transaminase<30 U/L for men and <19 U/L for women.

Bleeding time (BT): 3 – 11 minutes

Clotting time (CT): 4-10 mins

Prothrombin time (PT)11 – 13 seconds

International normalized ratio (INR) 0.8 – 1.1

Activated Partial thromboplastin time (aPTT) 30 – 40 seconds

LIMITATIONS OF THE STUDY:

1. Long term toxic effects of the rodenticide ingested are not included in the study.

2. Locally made rodenticide preparations don't have standard concentration of active ingredients and hence the outcome in such patients could not be correlated to the amount of rodenticide ingested.

3. Quantification of amount ingested is done approximately according to the patients and their relatives' history.

RESULTS AND OBSERVATION

The total number of patients included in the study was 421. But only 303 patient's data was finally included for the analysis. The remaining 118 patients did not give consent, absconded or were discharged prematurely before the completion of investigations and were excluded from the study. The remaining 303 patients' data were taken for final analysis.

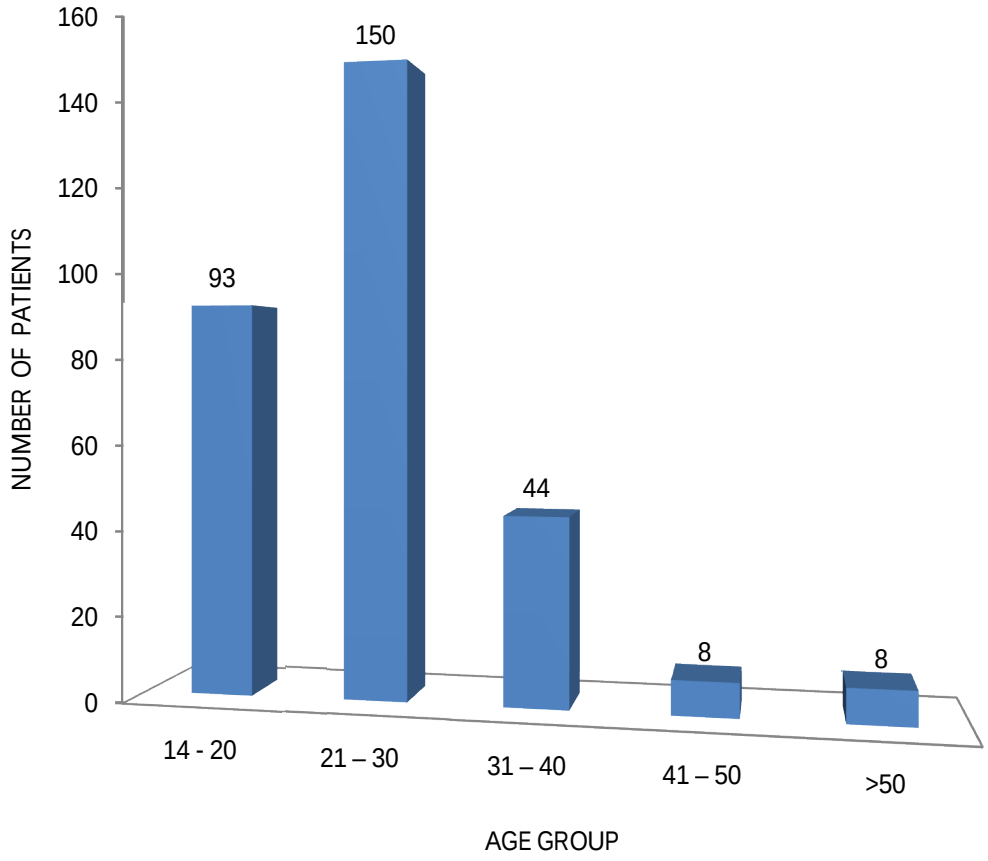
AGE AND SEX DISTRIBUTION

Table - 8 Age distribution

Age group (years)	No.of patients	%
14 - 20	93	30.7 %
21 – 30	150	49.5 %
31 – 40	44	14.5 %
41 – 50	8	2.6 %
>50	8	2.6 %
Total	303	100 %

Out of 303 patients studied most of the patients were below 30 years of age. 93 (30.7%) patients were between 14-20 years, 150 (49.5%) patients were between 21-30 years, 44(14.5%) patients were between 31-40 years, 8 (2.6%) were between 41-50 years, 8(2.6 %) patients were more than years of age.

Figure - 10 Age distribution



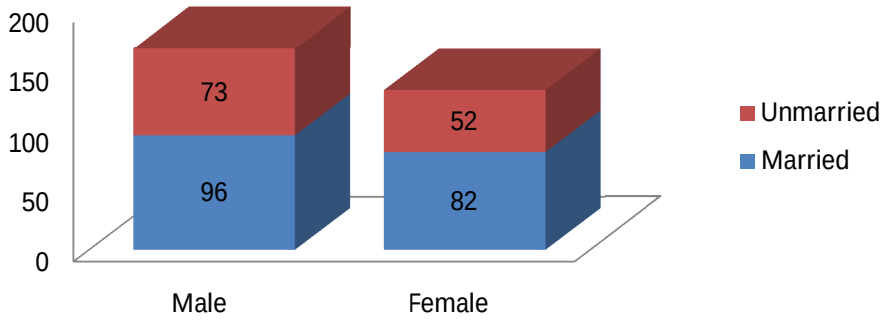
SEX AND MARITAL STATUS DISTRIBUTION

Table –9 Sex and Marital status distribution

Gender	Married		Unmarried		No. of patients	
	N	%	N	%	N	%
Male	96	56.8	73	43.2	169	100
Female	82	61.2	52	38.8	134	100
Total	178	58.7	125	41.3	303	100

Out of the 303 patients studied178 (58.7%) were married and 125 (41.3%) were unmarried. 169(55.8 %) were males and 134 (44.2 %) were females. Out of 169 males 96 (56.8%) were married and 73 (43.2%) were unmarried. Out of 134 females 82 (61.2%) were married and 52 (38.8%) were unmarried.

Figure - 11



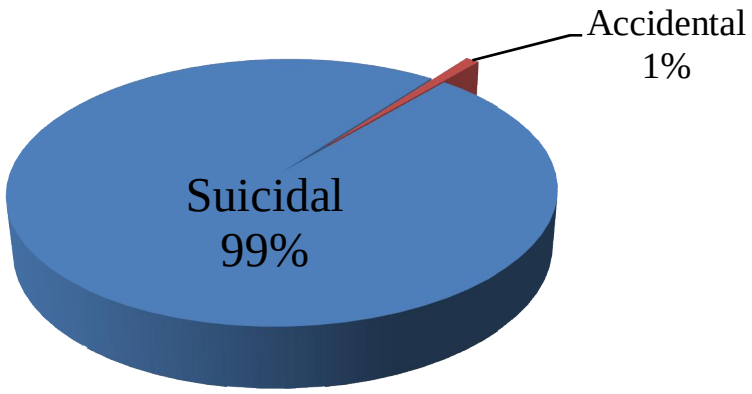
MODE OF POISONING

Table –10 Mode of poisoning

Mode of poisoning	No.of patients	%
Suicidal	300	99.0 %
Accidental	3	1.0 %
Total	303	100 %

Out of the 303 patients studied, 300 (99%) patients consumed rodenticide for committing suicide, 3 (1%) patients consumed rodenticide accidentally.

Figure 12- Mode of poisoning



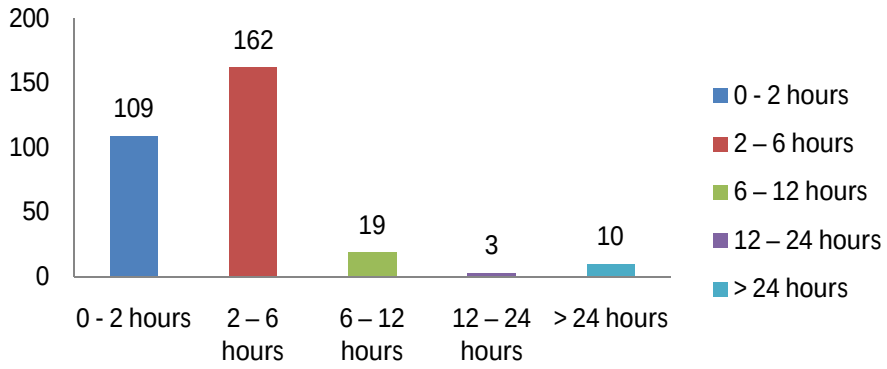
POISONING TO HOSPITAL PRESENTATION - TIME INTERVAL

Table –11 Time delay to present to hospital

Poisoning to hospital presentation - Time interval	No. of patients	%
0 - 2 hours	112	37.0 %
2 – 6 hours	163	53.8%
6 – 12 hours	19	6.2%
12 – 24 hours	3	1.0%
> 24 hours	6	2%
Total	303	100%

Out of the 303 patients studied 112 (37%) patients reached the hospital within 2 hours of consumption of rodenticide, 163 (53.8%) patients reached at 2 – 6 hours, 19(6.3%) patients reached at 6 – 12 hours, 3 (1%) patients reached at 12 – 24 hours, and 6 (2%) patients took more than 24 hours to reach the hospital.

Figure - 13 Time delay to present to hospital



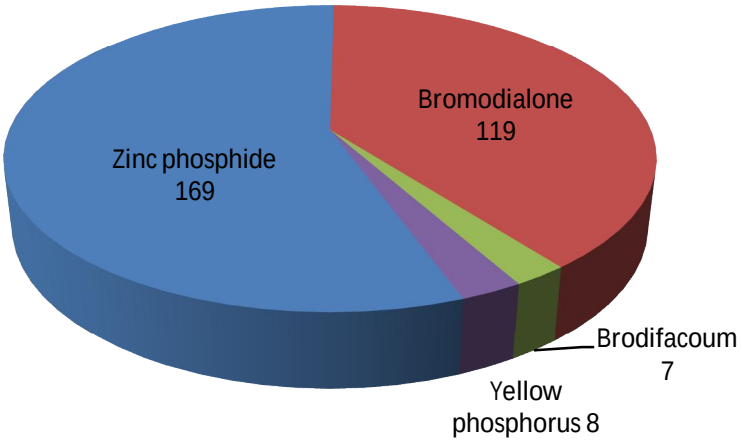
TYPE OF RODENTICIDE CONSUMED

Table –12 Type of Rodenticide consumed

Rodenticide type	No. of patients	%
Zinc phosphide	169	55.8%
Bromadiolone	119	39.3%
Brodifacoum	7	2.3%
Yellow phosphorus	8	2.6%
Total	303	100%

Out of the 303 patients studied 169 (55.8 %) consumed Zinc phosphide, 119 (39.3%) consumed Bromadiolone, 7 (2.3%) consumed Brodifacoum, 8 (2.6 %) consumed yellow phosphorus.

Figure - 14 Type of Rodenticide consumed



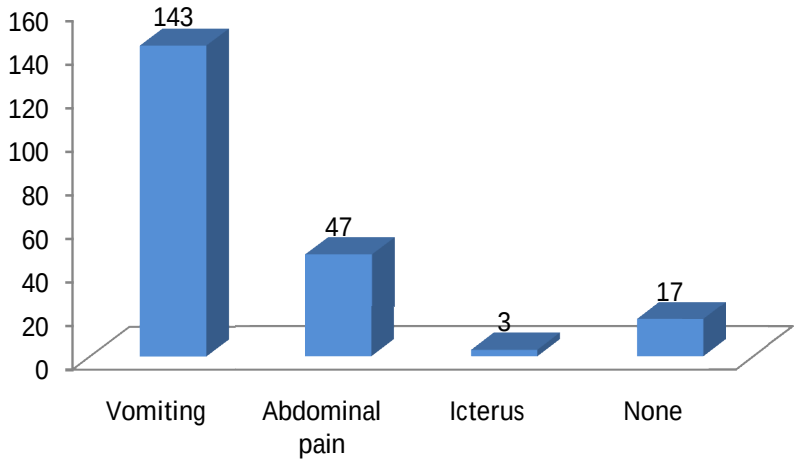
CLINICAL FEATURES AT PRESENTATION - ZINC PHOSPHIDE

Table –13 Clinical features of Zinc phosphide poisoning

Clinical feature	No. of patients	%
Vomiting	143	84.6%
Abdominal pain	47	27.8%
Icterus	3	1.8%
None	17	10.1 %
Total	169	100 %

Out of 169 patients who consumed zinc phosphide, 143 (84.6%) patients had vomiting, 47 (27.8%) had abdominal pain and 3 (1.8%) patients had icterus at presentation. Some of the patients had combination of these symptoms. 17(10.1%) patients had no complaints at presentation.

Figure - 15 Clinical features of Zinc phosphide poisoning



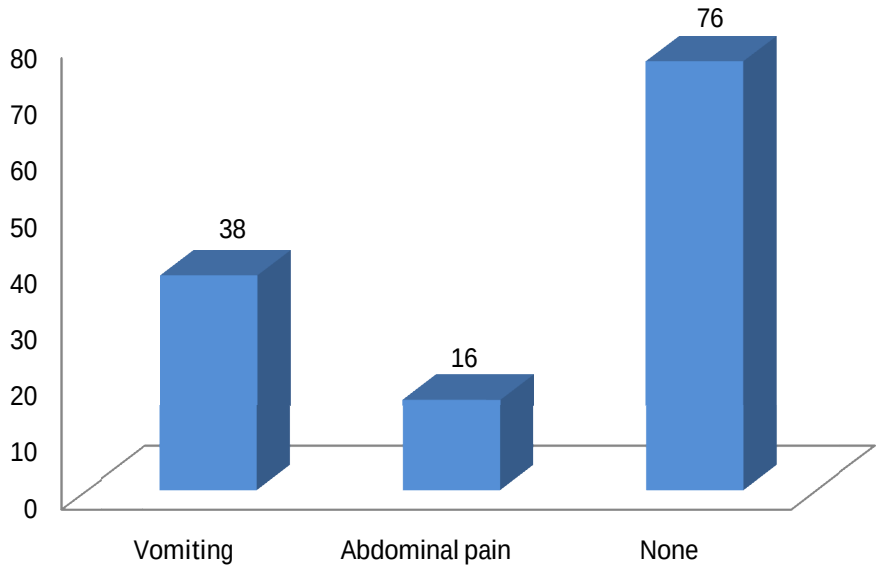
CLINICAL FEATURES AT PRESENTATION - BROMADIOLONE

Table –14 Clinical features of Bromadiolone poisoning

Clinical Features	No. of patients	%
Vomiting	38	31.9%
Abdominal pain	16	13.4%
None	76	63.9%
Total	119	100%

Out of 119 patients who consumed bromadiolone 38 (31.9%) patients had vomiting, 16 (13.4%) patients had abdominal pain and 76 (63.9%) had no complaints at presentation. Some of the patients had combination of these symptoms.

Figure - 16 Clinical features of Bromadiolone poisoning



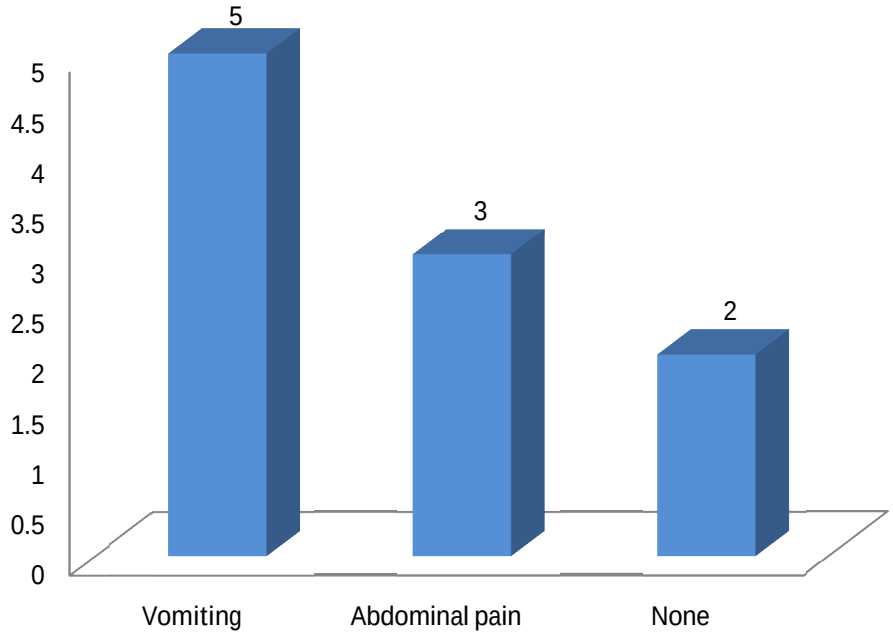
CLINICAL FEATURES AT PRESENTATION - BRODIFACOUM

Table –15 Clinical features of Brodifacoum poisoning

Clinical feature	N	%
Vomiting	5	71.4%
Abdominal pain	3	42.9%
None	2	28.6%
Total	7	100%

Out of 7 patients who consumed brodifacoum 5 (71.4%) had vomiting and 3 (42.9%) had abdominal pain. Some of the patients had combination of these symptoms. 2 (28.6%) patients had no complaints at presentation.

Figure - 17 Clinical features of Brodifacoum poisoning



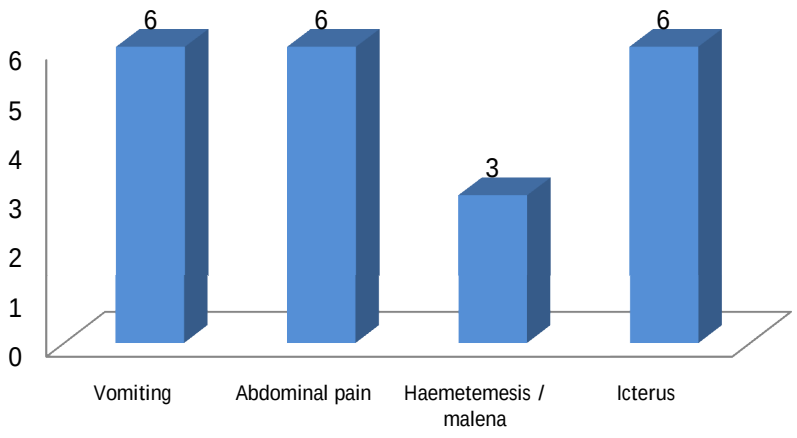
CLINICAL FEATURES AT PRESENTATION - YELLOW PHOSPHORUS

Table –16 Clinical features of Yellow phosphorus poisoning

Clinical feature	No. of patients	%
Vomiting	6	75%
Abdominal pain	6	75%
Haemetemesis / malena	3	37.5%
Icterus	6	75%
Total	8	100%

Out of the 8 patients who consumed yellow phosphorus, 6 (75%) patients had vomiting, 6 (75%) patients had abdominal pain, 3 (37.5%) patients had haemetemesis / malena, and 6 (75%) patients had icterus at presentation.

Figure - 18 Clinical features of Yellow phosphorus poisoning



VITAL SIGNS AT PRESENTATION

Table –17 Blood Pressure

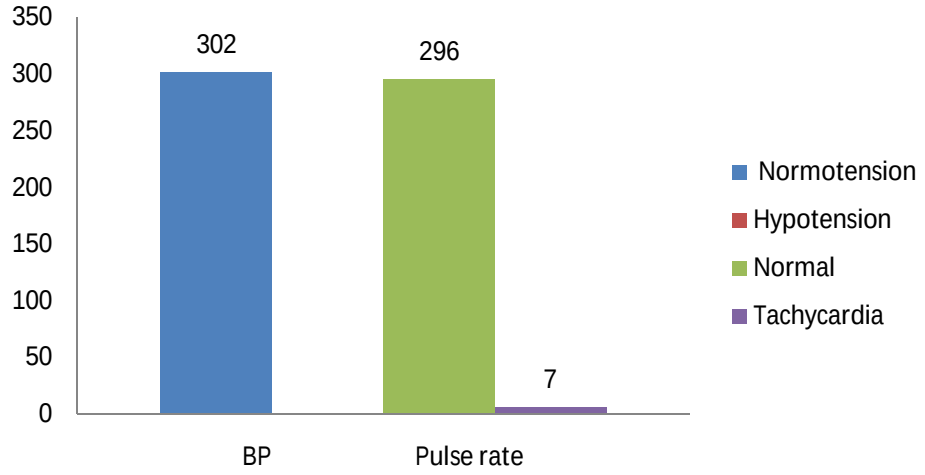
BP	No.of patients	%
Normotension	302	99.7%
Hypotension	1	0.3%
Hypertension	0	0%
Total	303	100%

Table –18 Pulse rate

Pulse rate	No.of patients	%
Normal	296	97.7%
Bradycardia	0	0%
Tachycardia	7	2.3%
Total	303	100%

Out of the 303 patients studied 302 (99.7%) patients had normal BP and 1 (0.3%) patient had hypotension and none had hypertension. Out of the 303 patients studied 296 (97.7%) had normal pulse rate, 7 (2.3%) had tachycardia and none had bradycardia.

Figure - 19 Vital signs



BLOOD SUGAR AT ADMISSION

Table -19 Blood sugar at admission

Rodenticide type	>90mg%		55 – 90mg%		< 55mg%		Total	
	N	%	N	%	N	%	N	%
Zinc Phosphide	93	55.0	67	39.6	9	5.3	169	100.0
Bromadiolone	110	92.4	9	7.6	0	0.0	119	100.0
Brodifacoum	6	85.7	1	14.3	0	0.0	7	100.0
Yellow Phosphorus	7	87.5	1	12.5	0	0.0	8	100.0
Total	216	71.3	78	25.7	9	3.0	303	100.0

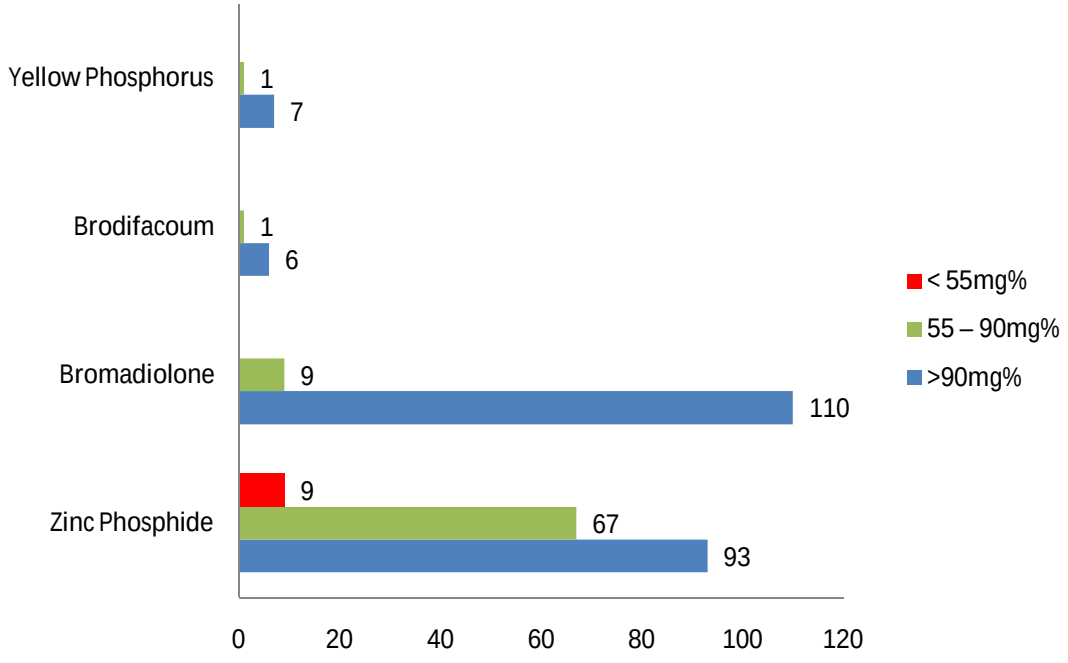
Out of 169 patients who consumed zinc phosphide 93 (55%) patients had blood sugar > 90mg /dl, 67 (39.6%) patients had blood sugar of 55 – 90 mg /dl, and 9 (5.3%) patients had blood sugar less than 55mg / dl.

Out of 119 patients who consumed bromadiolone 110 (92.4%) patients had blood sugar > 90mg / dl, 9 (7.6%) patients had blood sugar of 55 – 90 mg / dl, and none of the patients had blood sugar less than 55mg / dl.

Out of 7 patients who consumed brodifacoum 6 (85.7 %) patients had blood sugar > 90mg / dl, 1 (14.3%) patient had blood sugar of 55 – 90 mg / dl, and none of the patients had blood sugar less than 55mg / dl.

Out of 8 patients who consumed yellow phosphorus, 7 (87.5%) patients had blood sugar > 90mg / dl, 1 (12.5%) patient had blood sugar of 55 – 90 mg / dl, and none of the patients had blood sugar less than 55 mg / dl.

Figure - 20 Blood sugar at admission



INR - 48 HOURS AFTER ADMISSION

Table –20 INR - 48 hours after admission

	INR at 48 hours after admission										Total	
	<1.1		1.1 - 2		2.1 – 3		3.1 - 4		>4			
	N	%	N	%	N	%	N	%	N	%	N	%
Zinc Phosphide	163	96.4	4	2.4	0	0.0	0	0.0	2	1.2	169	100.0
Bromadiolone	48	40.3	7	5.9	35	29.4	23	19.3	6	5.0	119	100.0
Brodifacoum	2	28.6	0	0.0	4	57.1	1	14.3	0	0.0	7	100.0
Yellow Phosphorus	0	0.0	4	50.0	2	25.0	2	25.0	0	0.0	8	100.0
Total	213	70.3	15	5.0	41	13.5	26	8.6	8	2.6	303	100.0

Out of 169 patients who consumed zinc phosphide 163 (96.4%) patients had an INR <1.1, 4 (2.4%) patients had an INR 1.1 - 2, and2 (1.2%) patients had an INR >4.Out of 8 patients who consumed yellow phosphorus, 4 (50%) patients had an INR 1.1 - 2, 2 (25%) patients had an INR 2.1 – 3, and 4 (50%) patients had an INR 3.1 – 4.

Out of 119 patients who consumed bromadiolone 48 (40.3%) patients had an INR <1.1, 7 (5.9%) patients had an INR 1.1 - 2, 35 (29.4%) patients had an INR 2.1 – 3, 23 (19.3%) patients had an INR 3.1 – 4, and 6 (5%) patients had an INR >4.

Out of 7 patients who consumed brodifacoum 2 (28.6%) patients had an INR <1.1, 4 (57.1%) patients had an INR 2.1 – 3, 1 (14.3%) patient had an INR 3.1 – 4.

LIVER FUNCTION TEST

Table –21 Liver Function Test

Rodenticide type	Normal LFT		Abnormal LFT		Total	
	N	%	N	%	N	%
Zinc Phosphide	164	97.0	5	3.0	169	100%
Bromadiolone	119	100.0	0	0.0	119	100%
Brodifacoum	7	100.0	0	0.0	7	100%
Yellow Phosphorus	0	0.0	8	100.0	8	100%
Total	290	95.7	13	4.3	303	100%

Out of 169 patients who consumed zinc phosphide 164 (97%) patients had normal LFT, and 5 (3%) patients had abnormal LFT.

Out of 119 patients who consumed bromadiolone, all 119 (100%) patients had normal LFT and none of the patients had abnormal LFT.

Out of 7 patients who consumed brodifacoum, all 7 (100%) patients had normal LFT, and none of the patients had abnormal LFT.

Out of 8 patients who consumed yellow phosphorus, all 8 (100%) patients had abnormal LFT and none of the patients had normal LFT.

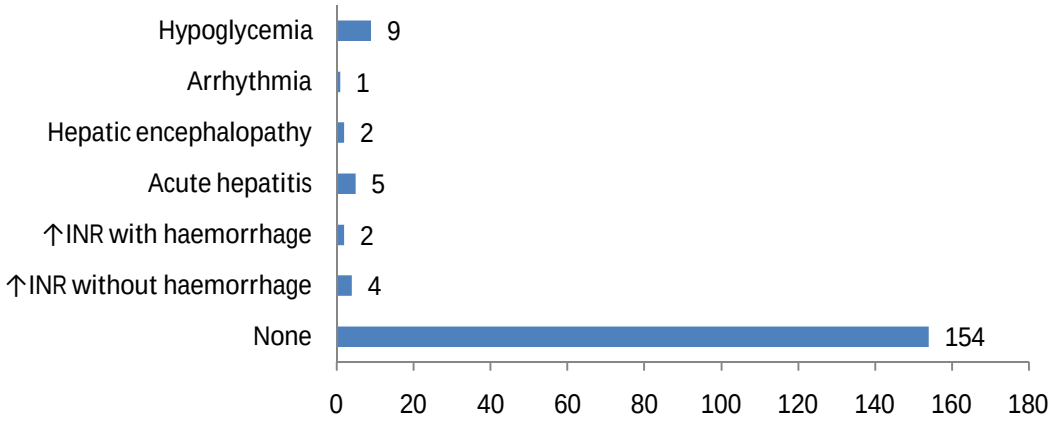
COMPLICATIONS OF ZINC PHOSPHIDE POISONING

Table -24 Complications of Zinc phosphide poisoning

Complication	No.	%
None	154	91.1 %
↑INR without haemorrhage	4	2.3 %
↑INR with haemorrhage	2	1.18 %
Acute hepatitis	5	2.95 %
Hepatic encephalopathy	2	1.18 %
Arrhythmia	1	0.6 %
Hypoglycaemia	9	5.3%
Total	169	100%

Out of the 169 patients who consumed zinc phosphide, 4 (2.3%) patients had increased INR without any haemorrhagic manifestation, 2 (1.18%) patients had increased INR with haemorrhagic manifestation, 5 (2.95%) patients had acute hepatitis, 2 (1.18%) patients had hepatic encephalopathy, 1 (0.6%) patient had arrhythmia, and 9 (5.3%) patients had hypoglycaemia.

Figure - 21 Complications of Zinc phosphide poisoning



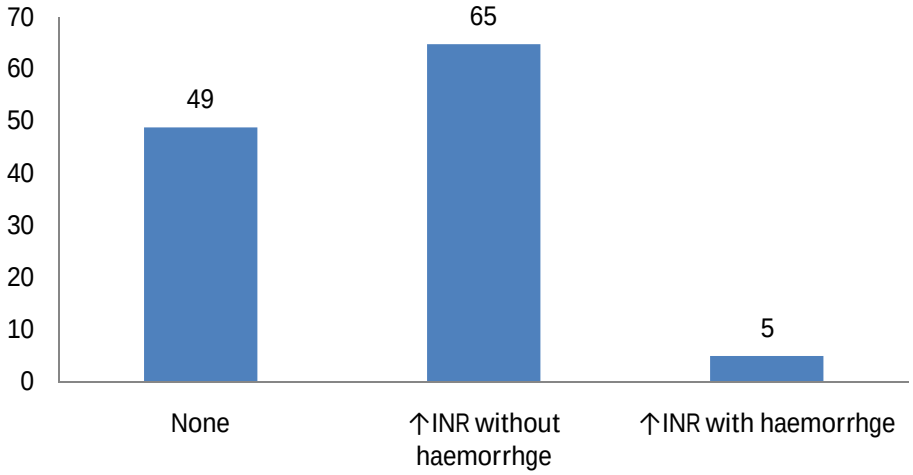
COMPLICATIONS OF BROMADIOLONE POISONING

Table - 23Complications of Bromadiolone poisoning

Complications	No	%
None	49	41.2 %
↑INR without haemorrhage	65	54.6 %
↑INR with haemorrhage	5	4.2 %
Total	119	100 %

Out of the 169 patients who consumed bromadiolone, 65 (54.6%) patients had increased INR without any haemorrhagic manifestation, 5 (4.2%) patients had increased INR with haemorrhagic manifestation, and 49 (41.2 %) patients did not develop any complications.

Figure - 22 Complications of Bromadiolone poisoning



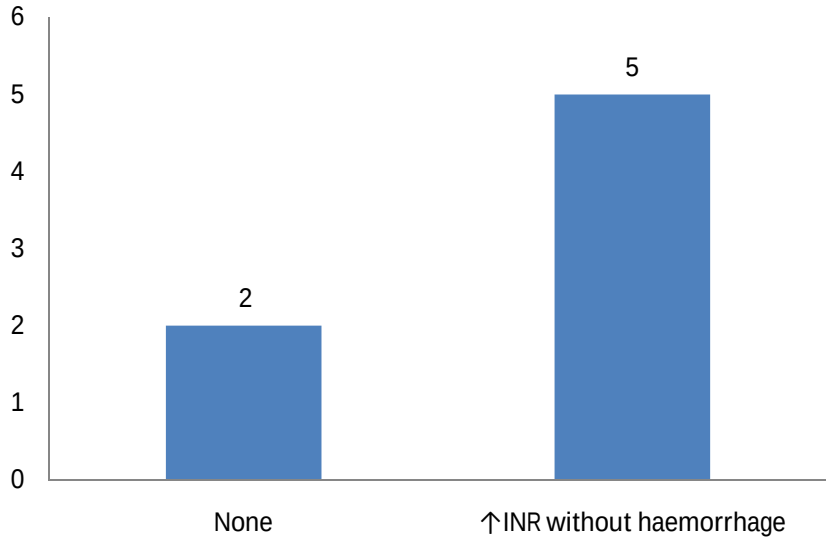
COMPLICATIONS OF BRODIFACOUM POISONING

Table –24 Complications of Brodifacoum poisoning

Complications	No	%
None	2	28.6 %
↑INR without haemorrhage	5	71.4 %
Total	7	100 %

Out of the 7 patients who consumed brodifacoum, 5 (71.4%) patients had increased INR without any haemorrhagic manifestation, and 2 (28.6 %) patients did not develop any complications.

Figure - 23 Complications of Brodifacoum poisoning



COMPLICATIONS OF YELLOW PHOSPHORUS POISONING

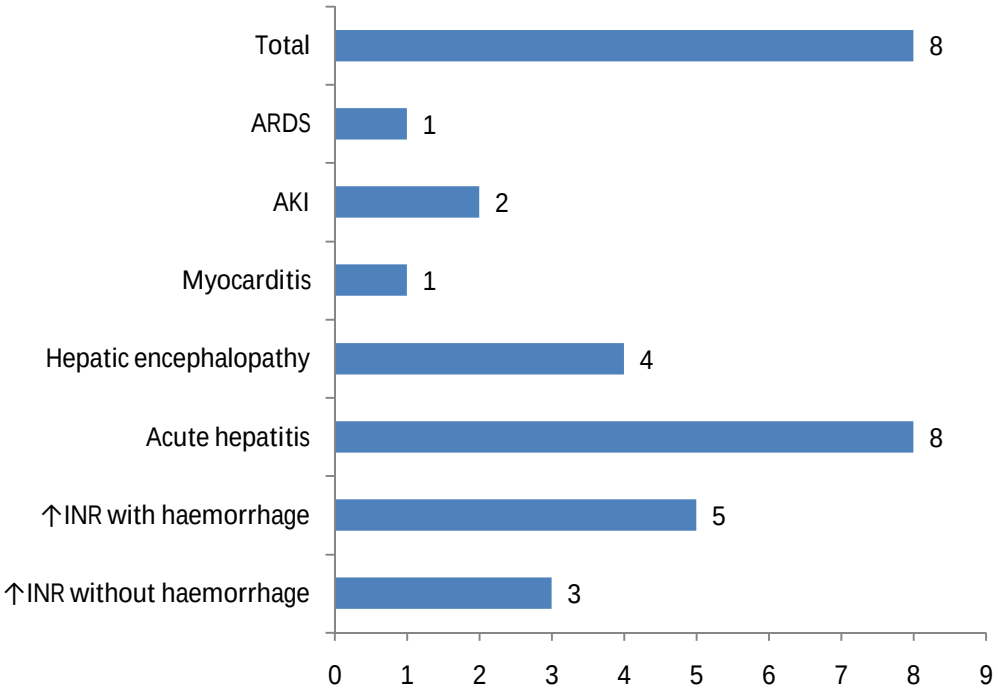
Table –25 Complications of Yellow phosphorus poisoning

Complications	No. of patients	%
↑INR without haemorrhage	3	37.5
↑INR with haemorrhage	5	62.5
Acute hepatitis	8	100
Hepatic encephalopathy	4	50
Myocarditis	1	12.5
AKI	2	25
ARDS	1	12.5
Total	8	100

Out of the 8 patients who consumed Yellow phosphorus, 3 (37.5%) patients had increased INR without any haemorrhagic manifestation, 5 (62.5%) patients had increased INR with haemorrhagic manifestation, 8 (100%) patients had acute hepatitis, 4 (50%) patients had hepatic

encephalopathy, 1 (12.5 %) patient had myocarditis, 2 (25%) patients had AKI (Acute Kidney Injury), and 1 (12.5%) patient had ARDS (Adult Respiratory Distress Syndrome).

Figure - 24 Complications of Yellow phosphorus poisoning



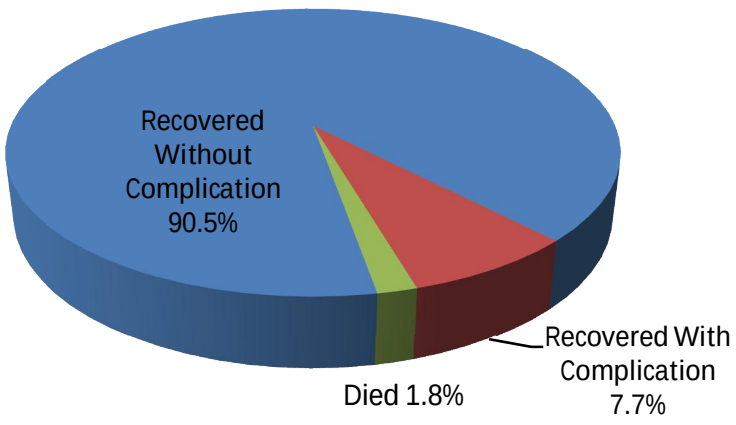
OUTCOME OF THE PATIENTS

Table - 26

Rodenticide type	OUTCOME						Total	
	Recovered Without Complication		Recovered With Complication		Died			
	N	%	N	%	N	%	N	%
Zinc Phosphide	153	90.5	13	7.7	3	1.8	169	100
Bromadiolone	49	41.2	70	58.8	0	0	119	100
Brodifacoum	2	28.6	5	71.4	0	0	7	100
Yellow Phosphorus	0	0	4	50	4	50	8	100
Total	204	67.3	92	30.4	7	2.3	303	100

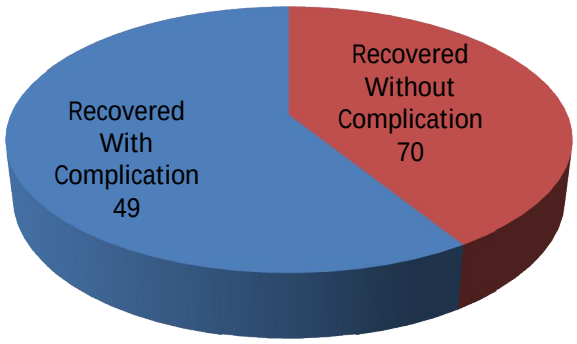
Out of 169 patients who consumed zinc phosphide 153 (90.5%) patients recovered without complication, 13 (7.7%) patients recovered with complication, and 3 (1.8%) patients died.

Figure - 25 Outcome of Zinc phosphide poisoning



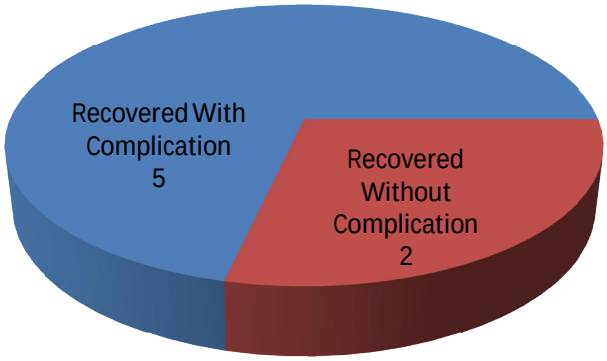
Out of 119 patients who consumed bromadiolone, 49 (41.2%) patients recovered without complication, 70 (58.8%) patients recovered with complication, and none of the patients died.

Figure - 26 Outcome of Bromadiolone poisoning



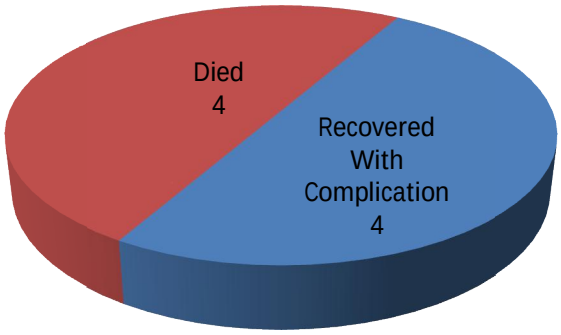
Out of 7 patients who consumed brodifacoum, 2 (28.6%) patients recovered without complication, 5 (71.4%) patients recovered with complication, and none of the patients died.

Figure - 27 Outcome of Brodifacoum poisoning



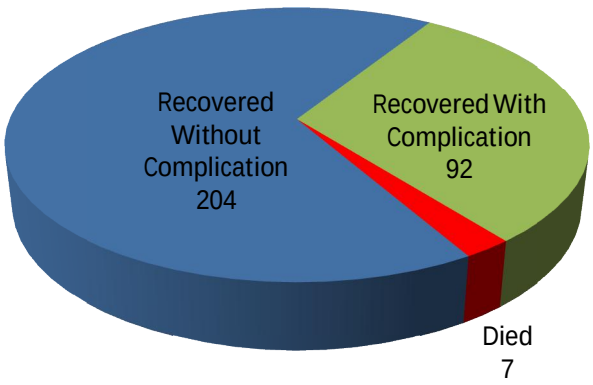
Out of 8 patients who consumed yellow phosphorus, 4 (50%) patients recovered with complication, 4 (50%) patients died. 96.4% (163) and none of the patients recovered without complication.

Figure - 28 Outcome of Yellow phosphorus poisoning



Out of the total 303 patients studied phosphide 204 (67.3%) patients recovered without complication, 92 (30.4%) patients recovered with complication, and 7 (2.3%) patients died.

Figure - 29 Total outcome of all the patients



COMPLICATIONS

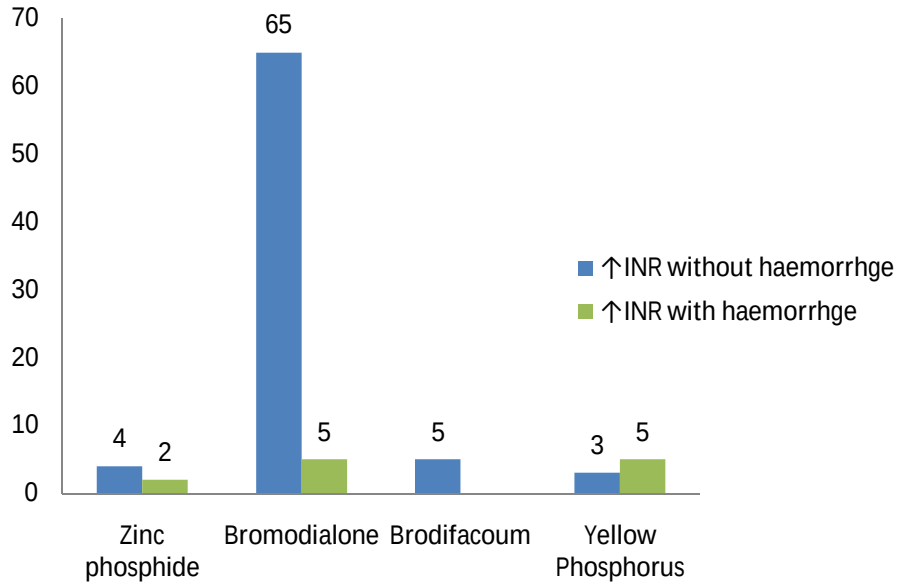
Table - 27

Complication	Zinc phosphide		Bromadiolone		Brodifacoum		Yellow Phosphorus		Total	
	N	%	N	%	N	%	N	%	N	%
↑ INR without haemorrhage	4	5.2	65	84.4	5	6.5	3	3.9	77	100
↑ INR with haemorrhage	2	16.6	5	41.7	0	0	5	41.7	12	100
Acute hepatitis	5	38.5	0	0	0	0	8	61.5	13	100
Hepatic encephalopathy	2	33.3	0	0	0	0	4	66.7	6	100
Myocarditis	0	0	0	0	0	0	1	100	1	100
Arrhythmia	1	100	0	0	0	0	0	0	1	100
AKI	0	0	0	0	0	0	2	100	2	100
Hypoglycaemia	9	100	0	0	0	0	0	0	9	100
ARDS	0	0	0	0	0	0	1	100	1	100
Total	23	18.8	70	57.4	5	4.1	24	19.7	122	100

Out of 77 patients who had increased INR without haemorrhage, 4 (5.2%) were due to Zinc Phosphide, 65 (84.4%) were due to Bromadiolone, 5 (6.5%) were due to Brodifacoum, 3 (3.9%) were due to Yellow Phosphorus.

Out of 12 patients who had increased INR with haemorrhage, 4 (16.6%) were due to Zinc Phosphide, 5 (41.7%) were due to Bromadiolone, 5 (41.7%) were due to Yellow Phosphorus.

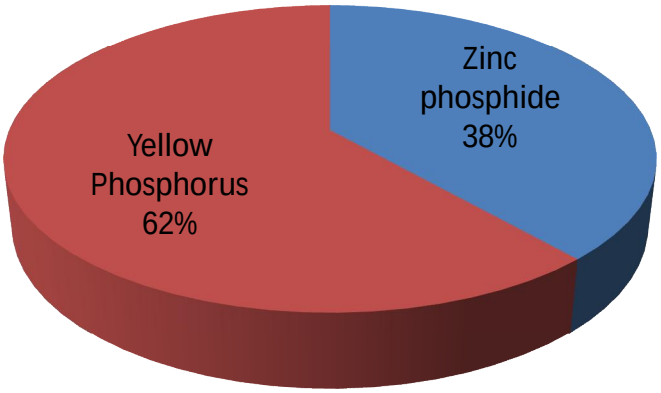
Figure - 30 INR changes in all the poisoning



- Myocarditis occurred in 1 patient which was due to Yellow Phosphorus.
- Arrhythmia occurred in 1 patient which was due to Zinc phosphide.
- ARDS occurred in 1 patient which was due to Yellow Phosphorus.
- Hypoglycaemia occurred in 9 patients which were due to Zinc phosphide.
- Acute Kidney Injury (AKI) occurred in 2 patients which were due to Yellow Phosphorus.

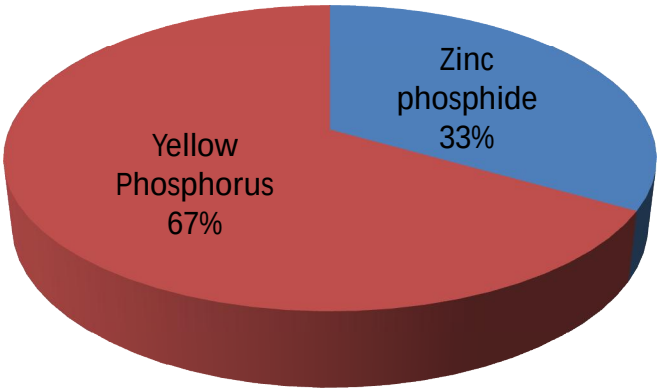
Out of 13 patients who had Acute Hepatitis, 5 (38.5%) were due to Zinc Phosphide, 8 (61.5%) were due to Yellow Phosphorus.

Figure - 31 Acute hepatitis



Out of 6 patients who had increased Hepatic encephalopathy, 2 (33.3%) were due to Zinc Phosphide, 4 (66.7%) were due to Yellow Phosphorus.

Figure - 32 Hepatic encephalopathy



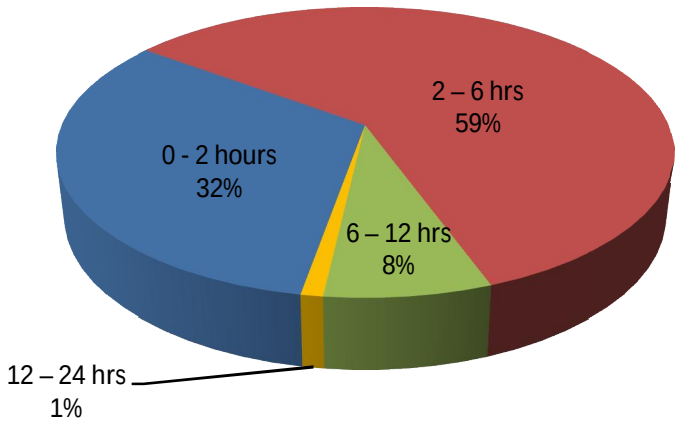
COMPARISION OF TIME DELAY IN PRESENTATION TO HOSPITAL AND THEIR OUTCOME

Table - 28

Duration	Time delay to present to hospital in hours										Total	
	0 - 2 hours		2 – 6 hrs		6 – 12 hrs		12 – 24 hrs		> 24 hrs			
	N	%	N	%	N	%	N	%	N	%	N	%
Recovered Without Complication	66	32.3	121	59.3	15	7.3	2	1	0	0	204	100
Recovered With Complication	42	45.6	41	44.5	4	4.3	1	1	4	4.3	92	100
Died	4	57.1	1	14.3	0	0	0	0	2	28.6	7	100
Total	112	37	163	53.8	19	6.2	3	1	6	2	303	100

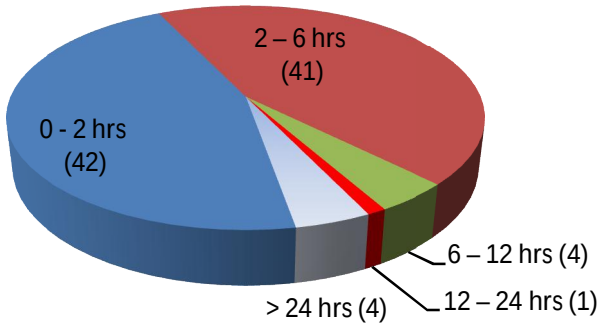
Out of the 204 patients who recovered without any complications, 66 (32.3%) patients presented to hospital within 2 hours of rodenticide ingestion, 121 (59.3%) patients presented at 2 – 6 hours, 15 (7.3%) patients presented at 6 – 12 hours, 2 (1%) patients presented at 12 – 24 hours, and none of the patients presented later than 24 hours after ingestion.

Figure - 33 Presentation - Time delay in patients who Recovered Without Complication



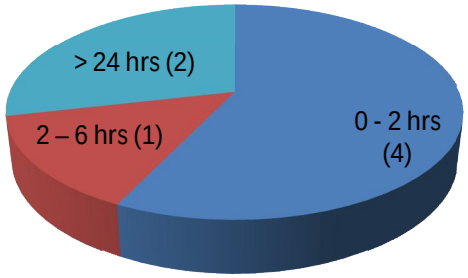
Out of the 92 patients who recovered with some complications, 42 (45.6%) patients presented to hospital within 2 hours of rodenticide ingestion, 41 (44.5%) patients presented at 2 – 6 hours, 4 (4.3%) patients presented at 6 – 12 hours, 1 (1%) patient presented at 12 – 24 hours, and 4 (4.3%) patients presented later than 24 hours after ingestion

Figure - 34 Presentation Time delay in patients who Recovered With Complications



Out of the 7 patients who died, 4 (57.1%) patient presented within 2 hours of rodenticide ingestion, 1 (14.3%) patient presented at 2 – 6 hours and 2 (28.6%) patients presented later than 24 hours after ingestion.

Figure -35 Presentation - Time delay in patients who died



DISCUSSION

Rodenticide toxicity is a commonly encountered condition in an emergency room setting. Most of the rodenticide poisoned patients recover well. Only a few patients develop significant toxic effects and even die. This varied clinical presentation depends on the type and quantity of the rodenticide consumed and particularly their make (whether locally prepared or a branded one). Instead of active ingredients, most of the locally made preparations contain a large amount of inert substances.

This study analyses the clinical presentation and outcome of various types of rodenticide toxicity encountered in our centre. Patients who were referred from other hospitals for the treatment of complications due to rodenticide toxicity are also included in this study.

Most patients [300 (99%)] consumed rodenticide with a suicidal intent.

80.2% (243) patients were below 30 years of age, signifying that suicidal tendencies are common among this age group.

Rodenticide consumption was found to be more common among the married (59 %) as compared to unmarried (41 %). This may be probably due to their marital stress.

275(90.8%) patients reached the hospital within 6 hours of poisoning, 21(7.2%) patients reached between 6 – 24 hours, and 6 (2 %) patients reached after 24 hours. Of the 6 (2%) patients who took more than 24 hours to reach the hospital, 2 patients succumbed and 4 patients recovered with complications. This clearly signifies that early and appropriate medical care has a strong effect on the outcome of the patients.

169 (55.8%) patients consumed zinc phosphide. Most of the patients in this group consumed locally made preparation as it was very cheap and easily available. 126(41.6%) patients consumed anticoagulant rodenticide which was available in form of powder and cake. 8(2.6 %) patients consumed yellow phosphorus which was available in form of a paste.

84.6 % of patients with zinc phosphide poisoning presented with vomiting which explains the highly emetic property of zinc phosphide and the potassium antimony tartrate which is added with zinc phosphide formulations to promote emesis.

Most of the patients who consumed bromadiolone or brodifacoum had no symptoms. Only a few patients had vomiting, abdominal pain at presentation.

Patients with yellow phosphorus poisoning were admitted with vomiting (75%), haemetemesis & malena (37.5%), icterus (75%). Of the total 7 patients with yellow phosphorus poisoning, 3 patients were referred from other hospitals for further management.

There were no demonstrable effects on the blood pressure and pulse rate of the patients except for hypotension in 1 patient and tachycardia in 7 patients.

3% of zinc phosphide poisoning and 100% of yellow phosphorus poisoning had an elevated serum total bilirubin along with elevated transaminases (ALT > AST) indicating significant hepatic involvement in those patient.

Zinc phosphide poisoning

In Shyam P. Lohani et al study titled “An Epidemiological Study on Acute Zinc Phosphide Poisoning in Nepal” conducted in Nepal from 1997 to 2002 analysed 178 patients on the clinical presentation and outcome after zinc phosphide ingestion. The results revealed that 79%

were asymptomatic, 18% had minor symptoms like abdominal pain, vomiting, dizziness, 2% had major symptoms and 1% died [55].

Patial RK et al case study revealed two patients with acute zinc phosphide poisoning developed severe hypoglycaemia and finally succumbed [56].

A case report by Frangides CY and Pneumatikos IA reveals persistent severe hypoglycaemia in patients with acute zinc phosphide poisoning [57].

In our study 154 (91%) patients who consumed zinc phosphide recovered without any complications. This may be due to highly emetic effect of zinc and potassium antimony tartrate or due to low concentration of the active ingredient present in the locally made preparations.

Out of 169 patients who consumed zinc phosphide 67 (39.6%) patients had blood sugar of 55 – 90 mg / dl, and 9 (5.3%) patients had blood sugar less than 55mg / dl. The blood sugar of all the patients improved with administration of dextrose solution and the repeated blood sugars until discharge of the patients were normal. This signifies that there is a period of transient hypoglycaemia due to zinc phosphide toxicity.

Out of 9 (5.3%) patients had documented hypoglycaemia, 1 patient died due to acute liver failure, and the remaining 8 patients recovered with no other complications.

Of the 5 (2.95%) patients who developed acute hepatitis 2 patients progressed to acute liver failure and succumbed.

One patient developed ventricular tachycardia and died.

Anticoagulant Rodenticide poisoning

Ingels M et al study on unintentional warfarin poisoning on 595 patients revealed no major coagulopathy and concluded that acute unintentional superwarfarin ingestions of less than1 box can be managed without gastric lavage or prophylactic vitamin K. and laboratory testing for coagulopathy should be done for patients with clinically evident bleeding abnormalities [58].

Chua JD and Friedenberg WR study on superwarfarin poisoning in 11 patients revealed no mortality. Coagulation abnormalities in all the patients were successfully reverted with vitamin k1 therapy [59].

An 8 year consolidated data from the Annual report of the American Association of Poison Control Centres, National Data

Collection System (1988 - 1995) on superwarfarin poisoning by LitovitzTLSchmitz revealed that there was a total of 79025 cases of superwarfarin exposure or poisoning reported over a period of 8 years in America. Out of which only 8 deaths were reported and only 67 patients (0.08%) had major complications [60 - 67].

In our study 70 (59.7%) patients with bromadiolone poisoning and 5(71.4%) patients with brodifacoum poisoning had an increased INR after 48 hours. The remaining patients had no coagulation abnormalities or other complications. Although the superwarfarin poisoning causes an elevated INR, 49(41.2%) patients with bromadiolone poisoning and 2(28.6 %) patients with brodifacoum poisoning had normal INR in this study. This could be explained by early gastrointestinal decontamination or inadequate amounts ingested. There were no deaths in this group of poisoning.

In our study most of the patients with superwarfarin poisoning did not develop major complications.

Yellow phosphorus poisoning

Oscar Santos et al in their case series titled “Acute liver failure due to white phosphorus ingestion” reported 3 patients with yellow

phosphorus poisoning, out of which 2 patients died and 1 patient recovered from acute fulminant hepatic failure [68].

S Karanth and V Nayyar reported 2 patients with yellow phosphorus poisoning with acute fulminant hepatitis. One of them succumbed to death and other patient was discharged at 2 weeks following signs of complete recovery [69].

Anupama Mauskar et al reported a case of accidental yellow phosphorus poisoning in a 3 year old girl who initially was asymptomatic but got admitted to hospital after 8 hours for vomiting, and later developed acute fulminant liver failure and recovered after 3 weeks of treatment [70].

Fernandez and Canizares in a case series of 15 patients have reported a mortality rate of 27%, confirming that yellow phosphorus is extremely lethal when ingested [40].

In our study all the patients who consumed yellow phosphorus developed acute hepatitis. Out of which 4 (50%) patients progressed to acute liver failure and succumbed. Other complications that were observed in this study were myocarditis in 1 (12.5%) patient, AKI in 2 (25 %) patients, ARDS in 1 (12.5%) patient.

All the patients with yellow phosphorus poisoning developed acute hepatitis and they were treated with dextrose containing fluids. Hence hypoglycaemia was not documented in this group. However there was one patient with low blood sugar.

All the patients with yellow phosphorus poisoning in our study were residents of salem and its suburban areas. This is probably due to easy availability of yellow phosphorus paste which is commonly used as rodenticide in paddy fields of salem. Those patients were referred to our hospital for tertiary care from the primary care hospitals in and around salem.

The time delay taken for the first medical contact is considered as the poisoning to hospital presentation - time interval. 3 (75%) patients who died due to yellow phosphorus poisoning consumed more than 10 grams of yellow phosphorus paste but presented to hospital within 2 hours of poisoning. Out of 4 patients who recovered with complications, 2 (50%) patients presented to hospital later than 24 hours of poisoning.

The above observation suggests the highly mortality in yellow phosphorus poisoning is dependent on the amount of yellow phosphorus ingested rather than the time delay at presentation.

CONCLUSION

Rodenticide poisoning, though commonly remarked as ‘rat never dies’; can be fatal to humans when the specific ingredient is consumed in a lethal dose.

Although there are ample studies about individual types of rodenticide, to our best of knowledge, we could not find any other study in the literature that compares the various types. Having said that, we must say the comparative study has yielded few striking facts.

- ✓ Zinc phosphide is the most prevalent poison in our study, but their mortality and morbidity rate is very low
- ✓ Super warfarin poisoning, the second common rodenticide in ourstudy, produced significant derangement in coagulation parameters, but failed to manifest clinically. (In short, it is rather benign)
- ✓ Yellow phosphorous, although leastcommon in our study, had a significant impact on the patient’s outcome. The mortality rate is a whopping 50% among the consumed.

Rodenticide poisoning, though encountered every day, had so many nuances and intricacies, which became apparent to us after analysing their clinical profile.

Indeed, it is a very satisfying experience in a common poison!

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PROFOMA

Name	
Age /sex:	
Occupation:	
Address with contact no:	
IP No:	
Date of admission:	
Date of discharge/ death	

Date and time of consumption	
Amount of consumption	
Type of rodenticide	

Vomiting		Altered sensorium	
Abdominal pain		Muscle twitching	
Haemetemesis		Specific odour	
Malena		Breathlessness	
Jaundice		Others	

Sensorium		CVS	
Icterus		RS	
Pedal edema		P/A	
Pallor		CNS	
BP		PR	

INVESTIGATIONS

TC	DC	ESR	HB	PCV	MCV	MCH	MCHC	PLT

Sugar	Urea	Cr	Na	K	Hco3	Cl

TB	DB	SGOT	SGPT	SAP	Total proteins	Albumin

PT	APTT	INR	BT	CT

Viral serology	USG abdomen

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A study on Clinical Profile of rodenticide poisoning at Intensive Medical Care Unit, Government Stanley Hospital, Chennai

Principal Investigator : Dr. G.Arunkumar

Designation : PG in MD (Gen. Med.)

Department : Department of General Medicine
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 11.06.2012 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

05/10/12

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MASTER CHART - 1

S.NO	IP.NO	AGE	SEX	MARITAL STATUS	DURATION	RODENTENCIDE			Clinical feature at presentation					VITALS		SYSTEMIC EXAM	OUTCOME	COMPLICATION
						TYPE	AMT	FORM	Vom	Abd pain	Bleeding	Icterus	Others	BP	PR			
1	37223	25	F	M	1	BD	4	2	A	A	A	A	N	N	N	N	Rec / com +	1
2	37320	19	F	UM	1	BD	3	2	A	A	A	A	N	N	N	N	Rec / com +	1
3	37346	30	M	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
4	37368	18	M	UM	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
5	37429	23	M	M	1	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
6	37601	19	M	UM	2	BD	2	1	A	A	A	A	N	N	N	N	Rec / com +	1
7	37793	13	M	UM	3	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
8	37803	45	M	M	1	BD	2	1	A	A	A	A	N	N	N	N	Rec / com +	1
9	37896	25	F	M	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com +	1
10	38107	32	M	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
11	38153	30	M	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
12	38212	21	M	UM	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
13	38383	17	F	UM	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
14	38411	36	M	M	1	BD	2	2	A	A	A	A	N	N	N	N	Rec / com +	1
15	38806	29	F	M	1	BD	4	1	P	P	A	A	N	N	N	N	Rec / com +	1
16	38940	24	M	M	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com +	1
17	39106	18	F	UM	1	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
18	40147	39	F	M	1	BD	2	1	A	A	A	A	N	N	N	N	Rec / com +	1
19	40342	20	F	UM	1	BD	2	1	A	A	A	A	N	N	N	N	Rec / com +	1
20	40589	20	M	UM	1	BD	4	1	P	A	A	A	N	N	N	N	Rec / com +	1
21	40643	28	F	M	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
22	40725	25	F	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
23	41216	23	M	UM	1	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
24	41375	24	F	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
25	41480	28	M	M	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
26	41531	21	M	UM	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com +	8

MASTER CHART - 1

27	41976	35	M	M	2	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
28	42422	28	M	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com +	8
29	42440	47	F	M	1	BD	2	2	A	A	A	A	N	N	N	N	Rec / com +	1
30	42942	19	F	UM	1	BD	3	1	P	A	A	A	N	N	N	N	Rec / com +	1
31	43393	20	M	UM	1	BD	1	1	A	A	A	A	N	N	N	N	Rec / com +	1
32	43869	25	F	UM	2	BD	3	2	P	A	A	A	N	N	N	N	Rec / com +	1
33	44014	52	F	M	1	ZP	4	1	P	A	A	A	N	N	T	N	DIED	6
34	44085	66	M	M	1	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com +	0
35	49295	27	F	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
36	44309	18	F	M	2	ZP	3	1	P	P	A	A	N	N	N	N	Rec / com -	0
37	44313	20	F	UM	2	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
38	44500	20	M	UM	1	BD	2	1	A	A	A	A	N	N	N	N	Rec / com +	1
39	44653	23	M	M	1	ZP	2	2	P	A	A	A	N	N	N	N	Rec / com +	1
40	45029	21	M	UM	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
41	45686	21	M	UM	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
42	45692	17	F	UM	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
43	45803	34	M	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
44	45848	27	F	M	2	ZP	1	2	A	A	A	A	N	N	N	N	Rec / com -	0
45	45880	43	M	M	4	BD	4	1	A	A	A	A	N	N	N	N	Rec / com +	1
46	46014	29	M	M	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com +	8
47	46294	60	M	M	2	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com +	8
48	46452	16	M	UM	2	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
49	46572	24	F	M	1	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
50	46574	22	F	UM	3	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
51	698	24	M	M	2	BD	2	1	A	A	A	A	N	N	N	N	Rec / com +	1
52	736	42	M	M	2	BD	3	2	P	A	A	A	N	N	N	N	Rec / com +	1
53	856	15	F	UM	1	ZP	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
54	934	24	M	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
55	943	24	M	M	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0

MASTER CHART - 1

56	1099	18	M	UM	1	YP	4	3	P	A	P	P	N	N	T	N	DIED	2,3,4,7
57	1210	22	M	UM	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com +	8
58	1468	20	F	UM	1	BD	1	2	A	A	A	A	N	N	N	N	Rec / com +	1
59	1545	18	F	UM	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com +	1
60	1636	21	F	UM	2	ZP	3	1	P	P	A	A	N	N	N	N	Rec / com -	0
61	2076	28	F	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
62	2340	18	F	UM	2	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
63	2369	18	F	UM	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
64	2693	31	M	M	2	BD	4	2	P	A	A	A	N	N	N	N	Rec / com +	1
65	2730	21	M	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
66	3354	30	M	M	1	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
67	3607	14	F	UM	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
68	3628	19	M	UM	2	BD	3	2	P	A	A	A	N	N	N	N	Rec / com +	1
69	3944	16	F	UM	2	ZP	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
70	4002	34	M	M	5	YP	2	3	A	A	A	P	N	N	N	N	Rec / com +	2,3
71	4919	60	F	M	1	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
72	5212	17	F	UM	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
73	5347	25	M	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
74	5556	24	M	M	2	BD	4	2	A	A	A	A	N	N	N	N	Rec / com +	2
75	5594	33	F	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
76	5670	22	M	UM	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
77	6172	26	F	M	1	BF	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
78	6375	28	M	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
79	6465	29	F	M	1	BD	4	1	P	P	A	A	N	N	N	N	Rec / com +	1
80	6565	20	F	UM	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
81	6870	24	F	M	5	ZP	4	1	P	A	A	A	N	N	N	N	DIED	2,3,4
82	7624	32	M	M	1	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
83	8316	26	F	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
84	8678	26	F	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0

MASTER CHART - 1

85	8710	23	F	M	1	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
86	8784	20	F	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
87	8796	19	F	UM	1	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
88	9103	24	F	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
89	9332	23	F	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
90	9582	30	F	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
91	9765	31	M	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
92	9992	26	M	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
93	10148	16	F	UM	1	BD	3	1	P	A	A	A	N	N	N	N	Rec / com +	1
94	10538	27	F	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
95	11519	17	F	UM	2	ZP	1	1	P	P	A	A	N	N	N	N	Rec / com -	0
96	11536	30	F	M	2	BD	3	1	A	A	A	A	N	N	N	N	Rec / com +	1
97	11569	16	F	UM	3	ZP	3	1	P	P	A	A	N	N	N	N	Rec / com -	0
98	11591	30	F	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
99	12791	23	M	UM	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
100	14976	34	M	M	1	BD	2	2	P	P	A	A	N	N	N	N	Rec / com -	0
101	15248	26	M	M	1	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
102	15336	32	F	M	2	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
103	15800	28	M	M	2	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
104	16259	25	F	M	1	YP	4	3	P	P	P	P	N	N	T	N	DIED	2,3,4,7
105	16366	39	F	M	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
106	16458	40	M	M	2	ZP	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
107	16468	36	M	M	1	BD	4	1	P	A	A	A	N	N	N	N	Rec / com +	1
108	17217	44	M	M	2	BD	3	1	P	A	A	A	N	N	N	N	Rec / com +	1
109	17323	26	M	M	5	ZP	4	1	P	P	A	P	N	N	N	N	Rec / com +	1,3
110	17327	21	F	UM	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
111	17400	17	M	UM	2	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
112	17654	25	F	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
113	17708	25	M	M	1	BD	2	1	A	A	A	A	N	N	N	N	Rec / com +	1

MASTER CHART - 1

114	18137	35	M	M	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
115	18146	22	F	UM	1	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
116	18201	21	M	UM	1	BD	3	1	A	A	A	A	N	N	N	N	Rec / com +	1
117	18444	17	F	UM	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
118	18475	50	M	M	1	BD	2	2	A	A	A	A	N	N	N	N	Rec / com -	0
119	18675	18	F	UM	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
120	18707	16	F	UM	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
121	18809	21	F	UM	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
122	18980	19	M	UM	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
123	19366	25	F	M	3	BD	3	2	P	P	A	A	N	N	N	N	Rec / com +	1
124	19419	19	F	UM	1	BD	3	1	P	P	A	A	N	N	N	N	Rec / com +	1
125	19578	29	M	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
126	19764	18	M	UM	1	YP	4	3	P	P	P	P	1	N	T	1	DIED	2,3,4
127	21051	27	F	M	2	BF	4	1	P	P	A	A	N	N	N	N	Rec / com +	1
128	21194	22	M	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
129	21203	20	F	UM	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
130	21395	20	M	UM	1	BF	2	1	P	P	A	A	N	N	N	N	Rec / com +	1
131	21407	24	M	M	4	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
132	21793	33	F	M	2	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
133	22119	34	F	M	2	BD	1	2	A	A	A	A	N	N	N	N	Rec / com -	0
134	22325	18	M	UM	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
135	22425	25	F	M	4	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
136	22503	35	F	M	1	ZP	1	1	P	P	A	A	N	N	N	N	Rec / com -	0
137	22493	22	F	UM	3	BD	4	2	A	A	A	A	N	N	N	N	Rec / com +	1
138	22524	20	M	UM	1	BD	3	2	P	P	A	A	N	N	N	N	Rec / com -	0
139	22543	23	M	UM	3	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
140	22865	17	F	UM	2	ZP	3	1	P	P	A	A	N	N	N	N	Rec / com -	0
141	22862	17	F	UM	1	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
142	24217	25	M	M	1	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0

MASTER CHART - 1

143	24674	24	M	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
144	24804	24	M	M	5	ZP	4	1	P	A	A	P	N	N	N	N	Rec / com +	1,3
145	23145	40	M	M	2	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
146	23969	14	F	UM	1	BD	3	1	P	A	A	A	N	N	N	N	Rec / com +	1
147	23999	18	M	UM	2	ZP	3	1	P	P	A	A	N	N	N	N	Rec / com -	0
148	24692	19	M	UM	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
149	24821	19	M	UM	1	BD	3	2	A	A	A	A	N	N	N	N	Rec / com +	1
150	24924	31	F	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
151	24972	30	M	M	2	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
152	24994	37	M	M	2	BD	3	1	P	A	A	A	N	N	N	N	Rec / com +	1
153	25395	21	F	M	1	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
154	25406	18	M	UM	2	BD	4	1	A	A	A	A	N	N	N	N	Rec / com +	1
155	25436	19	F	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
156	25694	19	M	UM	1	ZP	1	1	P	P	A	A	N	N	N	N	Rec / com -	0
157	25737	32	M	UM	1	BD	3	2	P	A	A	A	N	N	N	N	Rec / com +	1
158	25740	24	M	UM	3	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
159	25914	17	M	UM	2	BD	3	2	P	A	A	A	N	N	N	N	Rec / com +	1
160	25917	28	M	M	1	BD	3	1	P	A	A	A	N	N	N	N	Rec / com +	1
161	26111	20	F	UM	2	ZP	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
162	26765	21	M	UM	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
163	26401	32	M	M	1	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com -	0
164	26463	21	M	M	1	BD	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
165	26523	21	M	M	1	BD	1	2	A	A	A	A	N	N	N	N	Rec / com -	0
166	26574	19	F	M	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
167	26985	15	F	UM	2	BD	4	1	P	P	A	A	N	N	N	N	Rec / com +	1
168	27066	23	M	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
169	27474	16	F	UM	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
170	27481	24	M	UM	2	ZP	4	1	P	P	A	A	N	N	N	N	Rec / com -	0
171	27617	15	F	UM	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0

MASTER CHART - 1

172	28249	16	M	UM	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
173	28359	25	M	M	2	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com +	8
174	28550	29	M	M	1	YP	2	3	P	P	A	A	N	N	N	N	Rec / com +	1,3
175	28873	22	F	UM	2	BD	3	1	A	A	A	A	N	N	N	N	Rec / com +	1
176	28970	20	F	M	1	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
177	29168	18	F	M	1	BD	3	1	P	P	A	A	N	N	N	N	Rec / com +	1
178	29321	35	F	M	3	BD	4	1	A	A	A	A	N	N	N	N	Rec / com +	1
179	29685	20	F	UM	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
180	29801	21	F	M	2	BD	3	2	A	A	A	A	N	N	N	N	Rec / com +	1
181	29947	13	F	UM	1	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
182	29975	23	M	M	1	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
183	30161	20	M	UM	3	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
184	30307	30	M	UM	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
185	30352	29	F	M	2	BD	3	2	A	A	A	A	N	N	N	N	Rec / com +	1
186	30367	31	M	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
187	30501	24	F	M	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
188	30514	43	M	M	3	ZP	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
189	30681	37	M	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
190	30760	23	F	M	3	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
191	31134	26	F	M	3	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com +	1,3
192	31423	21	F	M	1	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
193	31447	30	F	M	2	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
194	31596	30	M	M	3	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
195	31875	25	M	M	1	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
196	32348	20	M	UM	2	ZP	4	1	P	P	A	A	N	N	N	N	Rec / com -	0
197	32568	22	M	UM	2	BD	3	2	A	A	A	A	N	N	N	N	Rec / com +	1
198	32644	23	M	UM	3	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
199	32829	17	F	UM	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
200	32906	17	F	UM	2	BD	1	1	A	P	A	A	N	N	N	N	Rec / com -	0

MASTER CHART - 1

201	32925	25	F	M	3	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
202	33055	22	F	M	1	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
203	33318	27	F	M	2	BD	3	1	A	A	A	A	N	N	N	N	Rec / com +	1
204	33520	24	M	UM	2	ZP	3	1	P	P	A	A	N	N	N	N	Rec / com -	0
205	33630	23	M	UM	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
206	33764	28	F	M	1	BD	2	2	A	A	A	A	N	N	N	N	Rec / com +	1
207	33846	53	M	M	1	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com -	0
208	34158	32	M	M	1	ZP	4	1	P	P	A	A	N	N	N	N	Rec / com -	0
209	34196	27	M	M	2	BD	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
210	34486	29	M	M	2	BD	3	2	A	A	A	A	N	N	N	N	Rec / com -	0
211	34596	34	F	M	1	ZP	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
212	34609	22	M	UM	1	BD	3	1	P	P	A	A	N	N	N	N	Rec / com +	2
213	34614	18	F	M	1	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
214	34674	31	M	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
215	34761	36	M	M	1	BD	4	2	A	P	A	A	N	N	N	N	Rec / com +	1
216	34897	20	M	UM	2	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com -	0
217	35020	25	M	UM	2	ZP	3	1	P	P	A	A	N	N	N	N	Rec / com -	0
218	35080	25	M	M	2	BD	4	2	P	A	A	A	N	N	N	N	Rec / com +	2
219	35118	37	M	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
220	35189	25	M	UM	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
221	35340	32	M	M	2	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com -	0
222	35413	27	F	M	2	BD	4	2	A	A	A	A	N	N	N	N	Rec / com +	1
223	35490	50	M	M	2	BD	4	1	P	P	A	A	N	N	N	N	Rec / com +	1
224	35515	35	F	M	2	BD	4	1	P	A	A	A	N	N	N	N	Rec / com +	1
225	35985	22	F	M	2	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
226	35995	28	M	M	2	ZP	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
227	36483	39	M	M	2	BF	3	2	A	A	A	A	N	N	N	N	Rec / com +	1
228	36740	35	M	M	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
229	36809	23	F	M	3	BF	1	1	P	A	A	A	N	N	N	N	Rec / com -	0

MASTER CHART - 1

230	36980	20	M	UM	1	YP	2	3	A	P	A	A	N	N	N	N	Rec / com +	1,3
231	37211	38	M	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
232	37337	24	F	M	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
233	37367	26	M	UM	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
234	37706	21	M	UM	2	ZP	4	1	P	P	A	A	N	N	N	N	Rec / com -	0
235	37737	20	M	UM	2	BD	4	1	P	A	A	A	N	N	N	N	Rec / com +	1
236	37728	21	F	UM	2	BD	3	1	P	A	A	A	N	N	N	N	Rec / com +	1
237	38047	26	F	M	1	ZP	4	1	P	P	A	A	N	N	N	N	Rec / com -	0
238	38101	17	M	UM	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
239	38139	19	M	UM	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
240	38393	18	M	UM	5	YP	3	3	P	P	A	P	1,2	N	T	N	Rec / com +	1,3
241	38462	19	F	M	2	BD	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
242	38672	27	M	M	2	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com -	0
243	38959	24	M	UM	3	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
244	38969	35	M	M	2	BD	3	2	P	A	A	A	N	N	N	N	Rec / com +	1
245	39027	26	M	UM	1	ZP	1	1	P	P	A	A	N	N	N	N	Rec / com -	0
246	39348	27	M	M	1	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
247	39420	27	F	M	1	BD	2	2	A	A	A	A	N	N	N	N	Rec / com +	1
248	39419	17	M	UM	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
249	39810	20	F	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
250	39912	26	M	M	1	BD	2	2	A	A	A	A	N	N	N	N	Rec / com +	2
251	40034	35	F	M	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
252	40050	24	F	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
253	40352	22	F	M	1	BD	3	2	A	A	A	A	N	N	N	N	Rec / com +	2
254	40486	25	F	M	2	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com -	0
255	40583	32	F	M	1	ZP	3	1	P	P	A	A	N	N	N	N	Rec / com -	0
256	40857	24	F	M	2	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
257	40879	29	F	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
258	41064	31	M	M	1	ZP	4	1	P	P	A	A	N	N	N	N	Rec / com -	0

MASTER CHART - 1

259	41235	19	F	M	1	BD	2	2	A	A	A	A	N	N	N	N	Rec / com +	1
260	41317	28	M	M	1	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
261	41377	18	M	UM	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
262	41878	20	F	M	3	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
263	42198	36	F	M	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
264	42305	15	F	UM	1	BD	4	1	P	P	A	A	N	N	N	N	Rec / com +	1
265	42469	14	F	UM	2	BD	3	1	P	A	A	A	N	N	N	N	Rec / com +	1
266	42472	23	M	UM	1	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
267	42496	22	M	UM	2	BF	2	2	A	A	A	A	N	N	N	N	Rec / com +	1
268	42610	63	M	M	1	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
269	42809	23	M	UM	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
270	42995	26	M	M	1	ZP	4	1	P	P	A	A	N	N	N	N	Rec / com +	8
271	43003	17	F	UM	1	BF	2	1	P	P	A	A	N	N	N	N	Rec / com +	1
272	43687	23	M	UM	3	BD	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
273	43120	27	M	M	2	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
274	43119	15	M	UM	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
275	43130	22	M	UM	2	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com -	0
276	43337	30	M	M	2	BD	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
277	43476	18	M	UM	2	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com -	0
278	43939	23	F	M	1	BD	2	2	P	A	A	A	N	N	N	N	Rec / com -	0
279	44067	37	M	M	2	ZP	1	1	P	A	A	A	N	N	N	N	Rec / com -	0
280	45057	16	F	UM	1	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
281	49248	18	M	UM	1	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com -	0
282	49343	23	M	UM	2	ZP	4	1	P	P	A	P	N	L	T	N	DIED	2,3,4,8
283	49426	18	F	UM	2	ZP	4	1	P	P	A	A	N	N	N	N	Rec / com -	0
284	49447	84	M	M	2	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
285	49945	15	F	UM	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
286	50029	28	F	M	1	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
287	50078	20	M	UM	5	YP	4	3	P	P	A	P	2	N	T	2	DIED	2,3,4,5,9

MASTER CHART - 1

288	50131	19	F	M	2	ZP	2	1	P	A	A	A	N	N	N	N	Rec / com -	0
289	50149	26	M	M	2	ZP	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
290	50365	16	F	UM	2	BD	2	1	A	A	A	A	N	N	N	N	Rec / com +	1
291	50552	26	M	UM	2	BD	2	2	A	A	A	A	N	N	N	N	Rec / com -	0
292	50555	24	M	M	2	ZP	4	1	P	A	A	A	N	N	N	N	Rec / com +	8
293	50970	22	M	UM	2	BD	3	1	P	A	A	A	N	N	N	N	Rec / com +	1
294	51120	27	M	M	1	ZP	1	1	A	P	A	A	N	N	N	N	Rec / com -	0
295	51131	27	M	M	1	ZP	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
296	51148	27	M	M	2	BD	4	1	P	A	A	A	N	N	N	N	Rec / com +	1
297	51303	33	M	M	1	BD	3	2	P	A	A	A	N	N	N	N	Rec / com +	1
298	52132	20	M	UM	1	ZP	3	1	P	A	A	A	N	N	N	N	Rec / com -	0
299	52393	19	M	UM	1	BD	2	1	A	A	A	A	N	N	N	N	Rec / com -	0
300	52475	28	M	M	2	ZP	2	1	P	P	A	A	N	N	N	N	Rec / com -	0
301	52473	80	M	M	1	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
302	52695	23	F	M	1	BD	1	1	A	A	A	A	N	N	N	N	Rec / com -	0
303	53603	23	M	UM	1	BD	3	2	P	A	A	A	N	N	N	N	Rec / com +	1

MASTER CHART - 2 (INVESTIGATIONS)

S.NO	IP.NO	RFT				LFT							CBC	COAGULATION PROFILE					VIRAL SEROLOGY	USG ABDOMEN
		Sugar	Ur	Cr	Electrolytes	TB	DB	OT	PT	SAP	TP	AL		PT	APTT	INR	BT	CT		
1	37223	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
2	37320	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
3	37346	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
4	37368	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
5	37429	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
6	37601	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
7	37793	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
8	37803	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
9	37896	1	N	N	N	N	N	N	N	N	N	N	N	I	N	1	0	N	Negative	Normal
10	38107	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
11	38153	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
12	38212	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
13	38383	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
14	38411	1	N	N	N	N	N	N	N	N	N	N	A	I	I	2	0	N	Negative	Normal
15	38806	2	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
16	38940	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
17	39106	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
18	40147	1	N	N	N	N	N	N	N	N	N	N	A	I	I	3	0	N	Negative	Normal
19	40342	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
20	40589	2	N	N	N	N	N	N	N	N	N	N	N	I	I	4	0	N	Negative	Normal
21	40643	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
22	40725	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
23	41216	1	N	N	N	N	N	N	N	N	N	N	N	I	N	1	0	N	Negative	Normal
24	41375	2	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
25	41480	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
26	41531	3	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal

KEY TO MASTER CHART-1

COMPLICATIONS	
1	ELEVATED INR WITHOUT HAEMORRHAGE
2	ELEVATED INR WITH HAEMORRHAGE
3	ACUTE HEPATITIS
4	HEPATIC ENCEPHALOPATHY
5	MYOCARDITIS
6	ARRHYTHMIAS
7	ACUTE KIDNEY INJURY
8	HYPOGLYCEMIA
9	ARDS

OUTCOME	
Rec/com-	RECOVERED WITHOUT COMPLICATION
Rec/com+	RECOVERED WITH COMPLICATION
DIED	DIED

BP (BLOOD PRESSURE)	
N	NORMAL
L	HYPOTENSION (BP <90/60)
H	HYPERTENSION(BP>140/90)

PR (PULSE RATE)	
N	NORMAL
B	BRADYCARDIA (PR<60/MIN)
T	TACHYCARDIA (PR> 100/MIN)

BLEEDING	
A	ABSENT
P	PRESENT

ABD PAIN (ABDOMINAL PAIN)	
A	ABSENT
P	PRESENT

OTHERS	
0	ABSENT
1	FEVER
2	BREATHLESSNESS

ICTERUS	
A	ABSENT
P	PRESENT

DURATION	
1	< 2 HOURS
2	2 – 6 HOURS
3	6 – 12 HOURS
4	12 – 24 HOURS
5	> 24 HOURS

Vom (VOMITING)	
A	ABSENT
P	PRESENT

RODENTICIDE (TYPE)	
ZP	ZINC PHOSPHIDE
BD	BROMODIALONE
BF	BRODIFACOUM
YP	YELLOW PHOSPHORUS

MARITAL STATUS	
UM	UNMARRIED
M	MARRIED

AMOUNT	
1	< 2 GRAMS
2	2 – 5 GRAMS
3	5 – 10 GRAMS
4	> 10 GRAMS

FORM	
1	POWDER
2	BAR / CAKE
3	PASTE

SYSTEMIC EXAMINATION	
N	NORMAL
1	UNCONCIOUS
2	CREPITATIONS

MASTER CHART - 2 (INVESTIGATIONS)

27	41976	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
28	42422	3	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
29	42440	1	N	N	N	N	N	N	N	N	N	N	A	I	N	2	0	N	Negative	Normal
30	42942	2	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
31	43393	1	N	N	N	N	N	N	N	N	N	N	N	I	N	1	0	N	Negative	Normal
32	43869	1	N	N	N	N	N	N	N	N	N	N	N	I	I	4	0	N	Negative	Normal
33	44014	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
34	44085	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
35	49295	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
36	44309	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
37	44313	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
38	44500	2	N	N	N	N	N	N	N	N	N	N	N	I	N	1	0	N	Negative	Normal
39	44653	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
40	45029	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
41	45686	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
42	45692	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
43	45803	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
44	45848	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
45	45880	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	I	Negative	Normal
46	46014	3	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
47	46294	3	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
48	46452	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
49	46572	2	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
50	46574	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
51	698	1	N	N	N	N	N	N	N	N	N	N	N	I	I	2	0	N	Negative	Normal
52	736	1	N	N	N	N	N	N	N	N	N	N	N	I	I	4	0	N	Negative	Normal
53	856	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
54	934	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal

MASTER CHART - 2 (INVESTIGATIONS)

55	943	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
56	1099	2	I	I	N	I	I	I	I	I	N	N	T	I	I	3	1	I	Negative	Normal
57	1210	3	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
58	1468	1	N	N	N	N	N	N	N	N	N	N	N	I	N	1	0	N	Negative	Normal
59	1545	2	N	N	N	N	N	N	N	N	N	N	N	I	N	1	0	N	Negative	Normal
60	1636	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
61	2076	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
62	2340	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
63	2369	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
64	2693	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
65	2730	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
66	3354	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
67	3607	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
68	3628	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
69	3944	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
70	4002	1	N	N	N	I	I	I	I	I	N	N	N	I	I	1	1	I	Negative	Normal
71	4919	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
72	5212	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
73	5347	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
74	5556	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
75	5594	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
76	5670	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
77	6172	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
78	6375	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
79	6465	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
80	6565	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
81	6870	1	N	N	N	I	I	I	I	I	N	N	T	I	I	4	1	I	Negative	Normal
82	7624	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal

MASTER CHART - 2 (INVESTIGATIONS)

83	8316	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
84	8678	2	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
85	8710	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
86	8784	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
87	8796	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
88	9103	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
89	9332	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
90	9582	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
91	9765	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
92	9992	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
93	10148	1	N	N	N	N	N	N	N	N	N	N	A	I	N	2	0	N	Negative	Normal
94	10538	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
95	11519	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
96	11536	2	N	N	N	N	N	N	N	N	N	N	N	N	N	2	0	N	Negative	Normal
97	11569	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
98	11591	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
99	12791	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
100	14976	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
101	15248	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
102	15336	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
103	15800	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
104	16259	1	I	I	N	I	I	I	I	I	N	N	N	I	I	2	0	I	Negative	Normal
105	16366	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
106	16458	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
107	16468	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
108	17217	1	N	N	N	N	N	N	N	N	N	N	N	I	I	2	0	N	Negative	Normal
109	17323	2	N	N	N	I	I	I	I	I	N	N	N	I	N	1	0	N	Negative	Normal
110	17327	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal

MASTER CHART - 2 (INVESTIGATIONS)

111	17400	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
112	17654	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
113	17708	1	N	N	N	N	N	N	N	N	N	N	I	I	2	0	N	Negative	Normal
114	18137	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
115	18146	2	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
116	18201	1	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
117	18444	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
118	18475	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
119	18675	1	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
120	18707	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
121	18809	2	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
122	18980	2	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
123	19366	1	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
124	19419	1	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
125	19578	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
126	19764	1	N	N	N	I	I	I	I	I	N	N	T	I	I	3	1	I	Negative	Normal
127	21051	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
128	21194	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
129	21203	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
130	21395	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
131	21407	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
132	21793	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
133	22119	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
134	22325	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
135	22425	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
136	22503	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
137	22493	1	N	N	N	N	N	N	N	N	N	N	N	I	I	2	0	N	Negative	Normal
138	22524	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal

MASTER CHART - 2 (INVESTIGATIONS)

139	22543	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
140	22865	2	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
141	22862	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
142	24217	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
143	24674	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
144	24804	1	N	N	N	I	I	I	I	I	N	N	N	I	N	1	0	N	Negative	Normal
145	23145	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
146	23969	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
147	23999	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
148	24692	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
149	24821	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
150	24924	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
151	24972	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
152	24994	1	N	N	N	N	N	N	N	N	N	N	N	I	I	2	0	N	Negative	Normal
153	25395	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
154	25406	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
155	25436	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
156	25694	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
157	25737	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
158	25740	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
159	25914	1	N	N	N	N	N	N	N	N	N	N	N	I	I	2	0	N	Negative	Normal
160	25917	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
161	26111	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
162	26765	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
163	26401	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
164	26463	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
165	26523	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
166	26574	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal

MASTER CHART - 2 (INVESTIGATIONS)

167	26985	1	N	N	N	N	N	N	N	N	N	N	N	I	I	4	0	N	Negative	Normal
168	27066	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
169	27474	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
170	27481	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
171	27617	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
172	28249	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
173	28359	3	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
174	28550	1	N	N	N	I	I	I	I	I	N	N	N	I	N	2	0	N	Negative	Normal
175	28873	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
176	28970	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
177	29168	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
178	29321	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
179	29685	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
180	29801	1	N	N	N	N	N	N	N	N	N	N	N	I	I	2	0	N	Negative	Normal
181	29947	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
182	29975	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
183	30161	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
184	30307	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
185	30352	2	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
186	30367	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
187	30501	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
188	30514	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
189	30681	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
190	30760	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
191	31134	2	N	N	N	I	I	I	I	I	N	N	N	I	N	1	0	N	Negative	Normal
192	31423	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
193	31447	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
194	31596	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal

MASTER CHART - 2 (INVESTIGATIONS)

195	31875	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
196	32348	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
197	32568	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
198	32644	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
199	32829	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
200	32906	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
201	32925	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
202	33055	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
203	33318	1	N	N	N	N	N	N	N	N	N	N	A	I	I	3	0	N	Negative	Normal
204	33520	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
205	33630	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
206	33764	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
207	33846	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
208	34158	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
209	34196	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
210	34486	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
211	34596	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
212	34609	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
213	34614	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
214	34674	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
215	34761	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
216	34897	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
217	35020	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
218	35080	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
219	35118	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
220	35189	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
221	35340	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
222	35413	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal

MASTER CHART - 2 (INVESTIGATIONS)

223	35490	1	N	N	N	N	N	N	N	N	N	N	N	I	I	4	0	N	Negative	Normal
224	35515	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
225	35985	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
226	35995	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
227	36483	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
228	36740	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
229	36809	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
230	36980	1	N	N	N	I	I	I	I	I	N	N	N	I	N	1	0	N	Negative	Normal
231	37211	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
232	37337	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
233	37367	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
234	37706	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
235	37737	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
236	37728	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
237	38047	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
238	38101	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
239	38139	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
240	38393	1	N	N	N	I	I	I	I	I	N	N	N	I	N	1	0	N	Negative	Normal
241	38462	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
242	38672	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
243	38959	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
244	38969	1	N	N	N	N	N	N	N	N	N	N	N	I	N	1	0	N	Negative	Normal
245	39027	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
246	39348	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
247	39420	1	N	N	N	N	N	N	N	N	N	N	N	I	I	2	0	N	Negative	Normal
248	39419	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
249	39810	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
250	39912	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal

MASTER CHART - 2 (INVESTIGATIONS)

251	40034	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
252	40050	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
253	40352	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
254	40486	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
255	40583	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
256	40857	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
257	40879	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
258	41064	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
259	41235	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
260	41317	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
261	41377	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
262	41878	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
263	42198	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
264	42305	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
265	42469	1	N	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
266	42472	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
267	42496	1	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
268	42610	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
269	42809	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
270	42995	3	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
271	43003	2	N	N	N	N	N	N	N	N	N	N	N	I	N	2	0	N	Negative	Normal
272	43687	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
273	43120	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
274	43119	2	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
275	43130	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
276	43337	1	N	N	N	N	N	N	N	N	N	N	A	N	N	0	0	N	Negative	Normal
277	43476	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
278	43939	1	N	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal

MASTER CHART - 2 (INVESTIGATIONS)

279	44067	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
280	45057	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
281	49248	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
282	49343	3	N	N	N	I	I	I	I	I	N	N	T	I	1	1	I	Negative	Normal
283	49426	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
284	49447	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
285	49945	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
286	50029	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
287	50078	1	N	N	N	I	I	I	I	I	N	N	N	I	1	0	N	Negative	Normal
288	50131	2	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
289	50149	2	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
290	50365	1	N	N	N	N	N	N	N	N	N	N	N	I	2	0	N	Negative	Normal
291	50552	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
292	50555	3	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
293	50970	1	N	N	N	N	N	N	N	N	N	N	I	I	3	0	N	Negative	Normal
294	51120	2	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
295	51131	2	N	N	N	N	N	N	N	N	N	N	I	I	4	0	N	Negative	Normal
296	51148	1	N	N	N	N	N	N	N	N	N	N	I	I	2	0	N	Negative	Normal
297	51303	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
298	52132	2	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
299	52393	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
300	52475	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
301	52473	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
302	52695	1	N	N	N	N	N	N	N	N	N	N	N	N	0	0	N	Negative	Normal
303	53603	1	N	N	N	N	N	N	N	N	N	N	I	I	4	0	N	Negative	Normal

KEY TO MASTER CHART - 2

SUGAR mg/dl	
1	>90
2	55 – 90
3	< 55

INR	
0	< 1.1
1	1.1 – 2
2	2.1 – 3
3	3.1 – 4
4	>4

CBC	
N	NORMAL
T	THROMBOCYTOPENIA
A	ANEMIC

		NORMAL	INCREASED
Ur	Urea	N	I
Cr	Creatinine	N	I
TB	Total Bilirubin	N	I
DB	Direct Bilirubin	N	I
OT	SGOT	N	I
PT	SGPT	N	I
SAP	Alkaline phosphatase	N	I
TP	Total Proteins	N	I
AL	Albumin	N	I
PT	Prothrombin Time	N	I
APTT	Activated partial prothrombin time	N	I
BT	Bleeding Time	N	I
CT	Clotting Time	N	I