STUDY ON THE EPIDEMIOLOGY, CLINICAL PROFILE AND OUTCOME OF THE POISONING IN CHILDREN

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CERTIFICATE

This is to certify that the dissertation titled "**STUDY ON THE EPIDEMIOLOGY, CLINICAL PROFILE AND OUTCOME OF THE POISONING IN CHILDREN"** of **Dr. G. GOPIKRISHNAN** in partial fulfillment of the requirements for **M.D. Branch – VII (Paediatrics)** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in February 2006. The period of study was from September 2004 to August 2005.

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DECLARATION

I, **Dr. G. GOPIKRISHNAN** solemnly declare that dissertation titled, **STUDY ON THE EPIDEMIOLOGY, CLINICAL PROFILE AND OUTCOME OF THE POISONING IN CHILDREN"** is a bonafide work done by me at Institute of Social Paediatrics, Govt. Stanley Medical College & Hospital during Sept. 2004 to Aug 2005 under the guidance and supervision of our Director **Prof. DR. T.K. VASANTHAMALLIGA, M.D., D.C.H.**.

The dissertation is submitted to Tamilnadu, Dr. M.G.R. Medical University, towards partial fulfilment of requirement for the award of **M.D. Degree (Branch – VII) in Paediatrics.**

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ABBREVIATIONS

1)	DSP	:	Deliberate self poisoning
2)	CNS	:	Central Nervous System
3)	ECG	:	Electro Cardio Gram
4)	IV	:	Intravenous
5)	ALT	:	Alanine Amino Transferase
6)	ACE	:	Angio Tensin Converting Enzyme
7)	ASV	:	Anti snake venom
8)	CSF	:	Cerebro Spinal Fluid
9)	ELISA	:	Enzyme Linked Immunosorbent Assay
10)	DIC	:	Disseminated Intravascular coagulation
11)	PICU	:	Paediatric Intensive Care Unit
12)	LOC	:	Loss of Consciousness

CONTENTS

INTRODUCTION

Children being an integral and more vulnerable section of our society, have been one of the principal victim of all the social ills, poisoning being no exception. Childhood poisoning run the entire gamut from accidental ingestion in toddlers and preschool children falling prey to their own curiosity; to intentional overdoses in the adolescents. These involve besides somewhat obsolescent traditional poisons, the in fashion intoxications with recreational drugs as well as chronic exposure to industrial chemical.

High incidence of poisoning in preschool children, is a direct consequence of the developmental stage of the child[36]. As the infant beings to crawl, creep and then walk around one year of age, his human instincts lead him into exploring the environment, putting the objects into his mouth being a part of this exploration. By two and half to three years of age, child's motor development and ingenecity make accessible to him things potentially noxious. The fourth year of life heralds a decline in the incidence of accidental poisoning, not withstanding a further motor development and co-ordination as the child now tends to be selective in choosing things for purpose of ingestion, putting to good use his least experiences.

Poisoning in children is one of the commonest emergencies encountered in paediatric practice. With ever increasing use of various chemical substances in the household and complexities of environment, the incidence of acute poisoning episodes is ever increasing. The causes and types of poisoning differ from place to place. These depend upon may factors such as education, socio-economic status, local beliefs and custums and also on the type of population, whether urban or rural[2].

Most of the poisoning in children is accidental in nature and most cases of accidental poisoning are preventable. Continuing morbidity and mortality due to poisoning is a serious challenge to the paediatricians and public health officials[35].

Accidental poisoning in children may be as a result of oral ingestion of poisonous substances (house hold products, chemicals, drugs, pesticides, etc) or parenteral poisoning (bites by Snakes and scorpion stings etc)[4]. Easy availability of injurious agents and environment are mainly responsible for poisoning in children. Further the risk of accidental poisoning is augmented by removing toxic substances from their original containers and putting them into drinking bottles. These substances are often mistaken by children for water and ingested[6].

Datas in different types of poisoning from different parts of the country are necessary to assess the magnitude of this childhood problem.

The reported incidence of childhood poisoning in various studies varies from 0.3%– 7.6% [1,2] which constitutes a significant number of admissions to the paediatric wards.

The incidence and type of poisoning would differ from place to place and it would have a special bearing on the emergency paediatric care of that area.

Hence this study was undertaken to find out the epidemiology, clinical profile and outcome of the poisoning in children which will help for better understanding in the management of poisoning in children.

AIM OF THE STUDY

The aim of the study is to study the epidemiology, clinical profile and out come of the poisoning in children under 12 years of age admitted in an urban referral centre in Chennai.

REVIEW OF LITERATURE

Poisons are as old as mankind or perhaps even older[36]. Their description can be found in the ancient Egyptian, Babylonian, Hebrew and Greek literature. Poisons have been described in Atharuva Veda (1500 BC), Kalpasthana, Chikitsasthana and Uttarasthana of the shastras have described symptoms and antidotes of poisons in detail.

Poisonings form an inescapable part of paediatric practice. It is not unusual to be confronted with a child with suspected poisoning in an emergency situation. Obviously, instanteous accurate diagnosis and prompt institution of appropriate therapy is life saving in this scenario. However, the diversity of poisonings in different settings and their variety of presentations can make this a daunting task.

The common types of poisons in children are given below :

i) Kerosene

ii) Chemical poisons

iii) Medicaments

iv) Pesticides

v) Corrosives

vi) Food poisons

vii) Animal poisons

a. Scorpion sting

b. Snake bite

Several studies have been done on poisoning in children.

KEROSENE OIL POISONING

Kerosene oil remains the most common household substance involved in accidental ingestion [3].

Kerosene which is used as cheap cooking fuel in urban household and for lighting in rural areas, accounted for 48.8% cases. The amount ingested couldn't be calculated properly. However it was never more than 10-15ml[5].

Toxicity is manifested primarily in gastrointestinal, CNS or respiratory systems [15,16.].

In Sitaraman S et al[3] study during 1982-1984, 2 new features were observed. One was pleural effusion which developed 5 days after

the ingestion and responded to antimicrobial therapy and another one was hemiparesis after a convulsive episode with in an hour of admission and recovered uneventfully.

Wolsdrof J [17] proved via an experimental model that CNS manifestations were secondary to pulmonary pathology and hypoxia.

X-ray chest showed features of pneumonitis in 40.3% of cases[5].

The mortality rate was 4.3%, the highest amongst the quoted studies. All the cases presented with severe manifestations of massive aspiration or were in coma at the time of admission.

Aspiration usually occurs at the time of ingestion, when coughing and gagging are common but can be secondary to vomiting that commonly occurs after ingestion[36]. The propensity of a hydrocarbon to cause aspiration pneumonitis is inversely proportional to its viscosity. Pneumonitis does not result from dermal absorption or from ingestion in the absence of aspiration.

Hydrocarbons can be absorbed after ingestion, inhalation or dermal contact. Most hydrocarbons have anesthetic properties and can cause transient CNS depression. Chlorinated solvents will produce hepatic toxicity. A few hydrocarbons have associated with renal

toxicity. A number of volatile hydrocarbons are commonly abused by inhalation. These substances can sensitize the myocardium with the risk of dysarrhythmias and sudden death. Chronic abuse can lead to cerebral atrophy, neuropsychological changes, peripheral neuropathy and renal diseases. The volatile hydrocarbons are lipid solvents and can cause defatting of the skin, producing local irritation or chemical burns.

Transient mild CNS depression is common after hydrocarbon ingestion. Aspiration is characterised by coughing, which usually is the first clinical finding. Chest radiographs may be normal for as long as 8-12 hours after aspiration. Respiratory symptoms may remain mild or may rapidly progress to respiratory failure. Fever occurs and may persist for as long as 10 days after aspiration. Accompanying Leukocytosis may be misleading because in most cases of aspiration pneumonitis no bacteria are present in the lungs. Chest radiographs may remain abnormal long after a patient is clinically normal and should not be used to guide acute treatment. Pneumatoceles may appear in the chest radiograph 2-3 Weeks after exposure

Emesis is contraindicated because of the risk of aspiration. Like wise gastric lavage is contraindicated, except under special circumstances, because of the risk of vomiting and aspiration.

Activated charcoal is not useful, because it doesn't bind the common hydrocarbons. If hydrocarbon induced pneumonitis develops, respiratory treatment is supportive. Corticosteroids should be avoided. Prophylactic antibiotics should not be given because bacterial pneumonia occurs in only a very small percentage of cases, respiratory failure has been successfully treated with both standard ventilation and with extracorporeal membrane oxygenation.

The incidence of accidental kerosene oil poisoning has dropped by half in 1980-1989 as compared to 1970-1979[7]. This could be due to increasing use of liquefied petroleum gas for cooking in urban house holds and better availability of electricity in rural areas.

POISONING DUE TO MEDICAMENTS AND CHEMICALS

The incidence of poisoning due to chemicals and medicaments was 35.7%. Though not yet posing a major threat as in developed countries the proportion was definitely high.

Poisoning due to phenothiazine group of drugs (chlorpromazine, promethazine and prochloperazine) is increasing gradually because of more frequent use of these drugs. Drowsiness, peculiar behaviour with

restlessness and signs of extra pyramidal involvement such as abnormal movements were the presenting features.

Apart from individual idiosyncrasy, inadvertent over dosage is frequently responsible for toxic symptoms. In vomiting and diarrhoea, dehydration is a frequent occurrence. Even a therapeutic dose could be potentially toxic because of spuriously high serum and tissue levels of the drugs as a result of hemoconcentration and diminished renal excretion[6].

Phenothiazine drugs are used primarily in psychotic illnesses[36]. They also find some use in treatment of nausea and vomiting, intractable hicoughs, preoperative adjuncts in anaesthesia and as antidepressants. Over dosages in children are either due to accidental ingestion of drugs available in home when one of the family member is taking drugs or inappropriate doses advised by unqualified practitioners.

Poisoning due to these substances may present with the following signs and symptoms.

Cardiovascular :

- Tachycardia
- Hypotension
- Ventricular arrhythmias
- Complete heart block.

Central nervous system :

- In differences to environmental stimuli
- Lethargy
- Coma
- Convulsions
- Hypothermia or hyperthermia with rhabdomyolysis

Extra pyramidal symptoms :

- Acute dystonic reactions
- Akasthisia
- Pseudo parkinsonism
- Tardive dyskinesia
- Choreiform movements of trunk or limbs.

- Neuroleptic malignant syndromes (rigidity, hyperthermia and stupor or coma).

Autonomic nervous system :

- Tachycardia
- Hypertension
- Diaphoresis
- Dyspnoea
- Incontinence

Pulmonary :

- Usually tachypnoea
- Rarely respiratory depression

Gastrointestinal :

- Nausea
- Vomiting
- Decreased bowel sounds

Eye :

- Blurring of vision
- Miosis
- Eye pigmentation

Skin :

- Dry and pigmented
- Dermatitis

Allergic and idiosyncratic :

- Jaundice
- Leukopenia
- Agranulocytosis

ECG Changes :

- Flattening and inversion of T-waves
- Prominent U weaves
- ST depression
- Prolonged PR, QRS and QT intervals.

Management consists of :

1) Secure airway and establish adequate ventilation
2) Establish in IV line using a large bore cannulation
3) All comatose patients should receive
    a. Nalaxone 0.02mg/kg
    b. 25% IV dextrose (2ml/kg)
4) Gastric lavage or emesis with syrup of ipecac, depending upon the level of consciousness.
5) Activated charcoal through lavage tube (1-2 g/kg)
6) Cathartic : Magnesium sulphate 250mg/kg orally.

Barbiturate and dilantin over dose occurred mostly in epileptic children or in those children belonging to families where at least one family member was epileptic[7].

Paracetomal is the most widely used analgesic and antipyretic[36]. The prime target organ of paracetamol toxicity is the liver. In addition to hepatotoxicity, renal tubular damage and hypoglycaemic coma may also occur.

CLINICAL FEATURES :

Stage I : This stage lasts for first 24 hours. Child presents with anorexia, vomiting, nausea, malaise, pallor and diaphoresis.

Stage II : This stage is seen 24 hours – 48 hours. It is characterized by resolution of symptoms of stage I with upper quadrant abdominal pain and tenderness. Mild hepatomegaly and jaundice may also present.

Stage III : This stage is seen 48 hours to 96 hours after ingestion. Maximum liver function abnormalities are seen during this period.

Stage IV : Stage of resolution and extends from four days to 2 weeks.; It is characterised by resolution of hepatic dysfunction.

MANAGEMENT :

I General measures and gastric lavage within 4 hours of its ingestion.

II Specific treatment to prevent acute liver damage : administration of N-acetylcysteine, preferably orally and within 8-16 hours.

Oral N-acetylcysteine : loading dose 140mg/kg. Maintenance dose : 70mg / kg at interval of four hours for 17 such doses.

Intravenous – acetylcysteine : Loading dose 150mg/kg IV in 5%. Dextrose (200ml) over 15 minutes. Maintenance dose : 50mg/kg in 5% Dextrose (500ml) over 4-8 hours for 3 doses.

III Supportive treatment consists of correction of hypoglycaemia by intravenous glucose and haemodialysis in acute renal failure.

PESTICIDES POISONING

The incidence of accidental poisoning by pesticides is increasing[3]. This is due to their more frequent domestic and agricultural use. Rat poisons and organophosphorus compounds like TIK-20 and Fenitrothion constitute more than 60% of the cases. Cases manifested as headache, abdominal cramp, salivation, muscular tremor, slow pulse and meiosis.

CORROSIVE POISONING

Corrosive poisoning (4.7%)[1] were due to ingestion of mineral acids, organic acids, vegetable acids and alkalies.

Involvement of mucus membrane of mouth, tongue and oesophagus produce salivation, inability to open the mouth and dysphagia. Dysphagia in mild form persisted even upto 2 weeks.

Niayaz A et al[8] in their study found that the incidence of corrosive poisoning was the least common (2.7%) mostly due to caustic soda. One child had taken hydrochloric acid. The cases were managed conservatively and recovered without any complications.

Sites likely to be affected by local effect :

- Skin of exposed parts of body and the face
- Mouth, throat
- Upper gastrointestinal tract
- Respiratory tract

Effects of corrosives :

***Early effects* :**

- Burning pain, tingling sensation.
- Vomiting often blood stained
- Dysphonia due to laryngeal edema

Late effects :

- Perforation of stomach and oesophagus
- Pulmonary edema or bronchopneumonia

Delayed effects :

- Laryngeal stricture
- Oesophageal stricture
- Pyloric fibrosis
- Pulmonary fibrosis

Mineral acid is the most common type of corrosive poisoning in children[36]. They are sulphuric acid, nitric acid, hydrochloric acid.

They corrode and cause tissue destruction by

I) Extraction of water from tissues

II) Coagulation of cellular protein and formation of acid albuminates

III) Conversion of haemoglobin to haematin

As a result, they cause local irritation, bleeding and sloughing of mucous membrane and skin. Apart from shock other remote systemic effects of mineral acids are rare. They usually get concentrated at the pyloric end of stomach, causing scarring and stricture formation. They may damage oesophagus and other areas of the stomach resulting in necrosis and perforation.

The clinical features depend on the mode of poisoning, concentration of mineral acid, Amount of acid used, duration of contact and age of child. Following ingestion of mineral acids severe pain in the

mouth, pharynx, chest and abdomen occurs, followed by hematemesis and bloody diarrhoea. The mucosa of mouth becomes black to brown with sulphuric acid yellow with nitric acid and grey brown with hydro chloric acid.

Frequently profound shock develops with mineral acid poisoning. Some times metabolic acidosis, liver and renal dysfunction haemolysis and DIC may be seen in severe cases. The features of mediastinitis and peritonitis may develop in surviving children resulting from early or late oesophageal and gastric perforation.

Lateral x-ray of the soft tissues of neck to evaluate upper airway compromise and chest and abdominal x-rays should be done to look for sings of oesophageal or gastric perforation in servere cases.

Oesophagoscopy and gastroscopy are diagnostic procedures of choice in all documented or suspected cases of corrosive ingestion to assess the extent and severity of the injury. To avoid perforation these procedures are performed in the first 24 hours and if possible with in the first hour of poisoning.

After giving proper care to airways, circulation and breathing, the dilution should be done immediately. The diluent of choice is milk.

Emetics and gastric lavage are contra indicated. A soft stomach tube or Levine tube cane be passed with care within an hour of ingestion and milk of magnesia is administered to neutralise the corrosive acids. If patient develops respiratory distress, endotracheal intubation, positive pressure ventilation, cricothyrotomy or tracheostomy may be done accordingly and patancy of air way should be maintained. Early administration of steroids is advocated in an attempt to decrease the incidence or severity of stricture formation and respiratory tract obstruction from laryngeal edema. It should be started preferably with in 48 hours. The steroid of choice is prednisolone in a dose of 2mg/kg/day. The duration of steroid therapy is for atleast 2 weeks or until oesophagus and / or stomach heal. Antacids should be used for burns of the stomach.

Oesophageal stricture or gastric out let obstruction may require subsequent dilatation and bougienage. In order to avoid perforation, oesophageal dilatation should be delayed 4-6 weeks until healing is complete.

FOOD POISONING

Buhariwalla R.J. et al[19] in their study found that in 18 out of 20 cases there was history of ingestion of food cooked in the previous day.

More than one member of the family was admitted at the same time in majority of the cases.

Sitaraman S. et al [3] noted considerable variation in the incidence of food poisoning.

SCORPION STING

Scorpion sting is an acute life threatening, time limiting medical emergency[20].

Case fatality rates of 3-22% were reported among children hospitalised for scorpion stings in India[21]. Among the 86 species of scorpion in India, Mesobuthus tamulus and palamneus swammer dami are of medical importance[22].

Cardiovascular effects are particularly prominent following the stings by Indian red scorpion (Mesobuthus tamulus)

Mahadevan S[9] in his study reports that scorpion retreat in the crevices of dwellings during the day only to emerge at night. Thus most stings are reported at nights. Scorpion stings are primarily due to accidental contact with scorpion.

Santhanakrishnan BR et al [24] in their study observed the incidence of the following clinical features. Peripheral failure in 80.7%, persistent tachycardia in 87.3%, E.C.G. changes suggestive of myocardial injury in 56.5%, pulmonary edema in 5.6%, seizures in 2.8% and bradycardia in 2.8%.

Prazosin a competitive post synaptic $\alpha 1$ adrenoreceptor antagonist, should be the first line of management, since α receptors stimulation plays a major role in the evolution of clinical spectrum[9].

The venom of scorpion is acidic[36] and it contains toxalbumin (neurotoxic), proteinases, phospholipases (both haematotoxic and myotoxic), cardiotoxins and charybdotoxin which is a specific inhibitor of high conductance calcium activated potassium channel.

Scorpion venom in addition to local irritant effect, acts as a powerful stimulus for

1) Massive release of catecholamines.

2) Suppression of insulin secretion

3) Elevation of plasma angiotensin II level.

The pathological changes, metabolic disturbances and cardiovascular manifestations produced by scorpion venom toxicity can be explained with above events.

The clinical manifestations are either local or systemic. The local manifestations are intense local pain, selling, ecchymosis and rarely tissue or bone necrosis.

NEUROLOGICAL

I) Autonomic nervous system :

These are the earliest and most prominent manifestations also known as the “autonomic storm”

- Profuse perspiration
- Tachypnoea
- Excessive salivation
- Vomiting
- Lacrimation
- Mydriasis
- Frequent passage of stools and urine
- Priapism and ejaculation.

II) Central Nervous Sytem :

- Encephalopathy
- Convulsions (Focal or generalised)
- Hemiplegia and other focal neurological deficits
- Transient blindness (amaurosis fugax)

III) Cardiovascular :

- Hypertension / Hypotension
- Arrhythmias
- Gallop rhythm
- Varying degrees of conduction block
- Apical systolic murmur
- Focal myocardial infarction
- Myocarditis
- Congestive heart failure
- Shock

ECG Changes :

In children with myocarditis serial ECG is helpful. Changes may be as follows:

- ST Segment - normal or depressed
- T-wave – flat or inverted.
- Deep Q-wave in lead I and AVL

- Various degrees of heart block.
- Arrhythmia (atrial of ventricular)

Echocardiography :

- Myocardial dysfunction either focal or generalised and ventricular dilatation.

IV) Respiratory :

- Dyspnoea
- Cyanosis
- Haemoptysis
- Pulmonary edema

V) Gastrointestinal :

- Abdominal pain
- Hematemesis
- Malena
- Pancreatitis
- Pseudo pancreatic cyst
- Rised serum amylase

VI) Metabolic:

- Metabolic acidosis
- Hyperglycemia, hyperkalemia
- ↑free fatty acids

- ↑cholesterol and triglycerides
- ↑ serum lactate

VII) Renal:

- Hematuria
- Oliguria
- Acute renal failure

VIII) Hematological :

- Increased erythrocyte fragility
- Disseminated intravascular coagulation.

IX) Hepatobilliary :

- Raised transaminases
- Raised Bilirubin
- Dilatation of branches of hepatic artery and vein.
- Intravascular thrombosis
- Subcapsular haemorrhage
- Focal hydropic degeneration
- Focal necrosis

X) Miscellaneous :

- Muscle fasciculation
- Tetany like contractures.

Diagnosis :

Diagnoses is based on

1) History of scorpion sting
2) Clinical features
3) Lab investigations
    a. Urine analysis
    b. Blood glucose
    c. Estimation of ALT
    d. Blood gas analysis
    e. ECG

Management of scorpion sting : It is divided into 2 groups I) local management II) Systemic Management.

Local Management : A ligature should be applied immediately proximal to the site of sting to delay absorption of toxins. The ligature should the released at frequent intervals in order to allow small amounts of toxin to reach the circulation which can be eliminated by the detoxifying mechanism of the body. The wound should be washed with plain water, ammonia, borax or potassium permanganate followed by cooling of the affected part with ice. Immobilisation of the affected part should be done. The affected area should be infiltrated with a local

anaesthetic agent preferably 2% xylocaine hydrochloride to alleviate pain. If the child has been previously immunised against tetanus one dose of tetanus toxoid should be administered.

SYSTEMIC MANAGEMENT :

The management is chiefly directed at neutralizing the toxin by antiserum and supportive therapy for complications. Antivenom therapy has been differently advocated by many workers. Administration of antivenom effectively neutralises, prevents and reverses the cardiovascular, haemodynamic, metabolic and electrocardiographic changes induced by scorpion venom. As the venom accumulates in cardiac tissues and act indirectly through the release of autopharmacological substances, treatment with sympatholytic agents (α or β blockers) may be more effective and rapid.

In the past the most widely used lytic cocktail consisted of chlorpromazine (50mg), promethazine (50mg) and pethidine (100mg) in 50ml of 5% dextrose in a dose of 0.3ml/kg/hour IV. It was once considered to be the mainstay of therapy because it induces a state of suspended animation, thereby reducing the cerebral metabolism and further ensueing complications. It is no longer used for managing scorpion stings. This is due to greater frequency of complications

observed during lytic cocktail therapy such as hypotension, respiratory depression and convulsions.

α blocker (prazosin) antagonises the effects of catacholamines and thus prevents further damage of myocardium but it cannot reverse the damage. Prazosin 0.4mg/kg/day orally in two-three divided doses is used either alone or along with sodium bicarbonate and / or insulin.

Recently insulin has been recommended for the treatment of scorpion sting and is being widely used. The dose is 0.1-0.2IU/Kg/day subcutaneously in three divided doses with frequent monitoring of blood sugar to prevent hypoglycaemic attacks.

Diuretics should not be used in scorpion bite even in the presence of pulmonary edema because of their dehydrating effect, alteration in blood viscosity and stimulation of renin-angiotensin system.

Glucocorticoids are contra indicated in the treatment of scorpion envenomation because of their catabolic and antiinsulin action, which only aggravates the complications. It also stimulates renin – antiotensin system, hence glucocorticoids are contra indicated in non-cardiogenic pulmonary edema.

Hypertension should be controlled with calcium channel blockers, vasodilators, and ACE inhibitors. ACE inhibitors are particularly effective because of elevated angiotensin II level in patients with scorpion sting. Peripheral circulatory failure should be effectively managed with low dose dopamine (5-20μg/kg/mt) along with supportive therapy. It the patient has developed pulmonary edema, it should be managed with 100% oxygen, insulin, vasodilator therapy and ventilatory support. Intravenous calcium gluconate is indicated, if fasciculations and tetany like muscular contractions develop.

Convulsions are managed as per convention. Children who have developed defibrination syndrome, acroosteolysis or encephalopathy should be managed conservatively.

Prevention : In scorpion endemic area protective items like boots, socks and trousers may prevent scorpion sting. Spraying with 10% DDT kills the scorpion.

SNAKE BITE

Snake bites are common in children above 5 years as they prefer playing out door games[4]. There are 4 major virulent types of poisonous

land snakes in India, which are of clinical importance. These are cobras, kraits, Russels viper and Echiscarinata[25].

Maximum number of cases were reported during the month of July and August and maximum belonged to rural areas [27, 28].

The intensity of local reaction is an indicator of severity of envenomation [29,30].

The clinical picture resulting from the bites of kraits and cobra is dominated by neurological disturbances and that of Russels viperi and Echiscarinata is characterised by haemorrhagic disturbances due to defibrination syndrome.

ASV is the main stay of the treatment[11]. Abnormal local site reaction or coagulation disturbances or neurological manifestations are considered as the absolute indication for the use of ASV.

There is one pair of poison gland situated behind the eyes on either side of the poisonous snake[36]. It is a modified salaivary gland.

All poisonous snakes have 2 fangs. These are connulated curved teeth situated on the maxillary bones and are connected with the sac of the gland. When snakes bite poison is poured through fangs into the wound.

Snake venom is a complex mixture of proteins having enzymatic activities. Some important enzymes present in venom are, Proteinase, Transaminase, Hyaluronidase, Phosphodiesterase, 5-nucleotidase, alkaline phosphatase, ribonuclease, deoxyribo nuclease, acid phosphatases, endonucleases and cholinesterases.

Systemic absorption of the venom occurs through the lymphatics. The action of the venom leads to pooling of blood in the microcirculation and subsequent loss of plasma due to increased capillary permeability leading to fall in circulating blood volume. This in turn causes circulatory collapse and death.

Snake venom has the following local effects (1) direct cytotoxic, 2) secondary to ischemia, compression of nerves and infections. Haemototoxic effects are (1) activation of phospholipase of RBC membrane, 2) Formation of lecithin and isolecitin (haemolysis), 3) Activation of coagulation system (Schwarzman like phenomenon), 4) increased adhesiveness (microangiopathy), 5) Conversion of prothrombin to thrombin and fibrinogen to fibrin, 6) Anti coagulant effect. Consumptive coagulopathy. Neurotoxic effects are 1) post synaptic block (Cobra), 2) Presynaptic block (Krait).

It can produce cardiac asystole

Factors affecting severity of snake bites are :

1) Age : younger the age more serious will be the outcome.

2) Location of bite : bite on face and trunk is more dangerous than those on extremities.

3) Size of the snake : larger the snake, worse the prognosis.

4) Secondary infection : presence or absence of clostridia and / or anaerobic organisms in the wound or the skin of the victim.

5) Post snake bite activity : exercise or running after the bite increases the rate of absorption of toxins.

Following lab abnormalities are observed : anaemia, leucocytosis, thrombocytopenia, hypofibrinogenemia, proteinuria, azotemia.

ECG changes are bradycardia, ST segment depression or elevation, T-wave inversion, prolongation of QT interval and various types of arrhythmias. Immunodiagnosis : ELISA is widely used to detect specific snake venom antigen in wound aspirate, serum, urine,

CSF and other body fluids. Treatment can be divided in 2 parts, I first aid II immediate management.

Assure the patient who prevent exertion and vagal syncope and to allay anxiety. Immobilize the limb. Apply a tourniquet above the bite mark about 5cm above the upper limit of edeme or fang mark and shift it proximally every 15 minutes, if swelling spreads further. Tourniquet should be tight enough only to obstruct lymphatic flow but not the venous drainage. A tourniquet can only be applied if snake bite occurs on the limbs. Clean the wound with sterile saline and cover with a sterile dressing. Incision and suction does not help in removal of venom or improve the out come. So this is no longer recommended.

Establish IV and administer fluids and plasma expanders or blood transfusions to restore intravascular volume. Care of the airways and breathing should be given first priority. If there is any evidence of respiratory failure, early intubation and assisted ventilation may be used. Inject tetanus toxoid one dose, if child is immunized. Previously. Appropriate antibiotics must be given to cover both gram positive and gram negative organisms because secondary infections of necrotizing wound may lead to septicaemia. Surgical debridement and immediate split skin grafting is indicated if there are signs of necrosis and

gangrene. Irrigation of eyes with large volume of water is indicated if there is snake venom ophthalmia.

Antivenom is indicated in patients with serious manifestations of envenomation like impaired consciousness, neurotoxicity, hypotension, shock, abnormal ECG, bleeding diathesis, DIC, rhabdomyolysis and acute renal failure. When local swelling at the site of snake bite, involving more than half of the bitten limb with extensive blistering or bruising is also indication for antivenom.

The antivenom is diluted with 10ml of distilled water or isotonic saline and given at the rate of 4ml / minute. Reconstituted antivenin can, however, also be diluted with 3 volumes of normal saline and infused very slowly and then with increased rate if well tolerated over next 1-2 hours.

Neurotoxic envenomation leading to respiratory paralysis is managed by anticholinesterases. Patients who respond to atropine sulphate (50μg/kg) and edrophonium (0.25mg/kg IV) should be given, neostigmine methylsulphate (50-100μg/kg) and atropine 4 hourly or by continuous intravenous infusion.

Contraindications to antivenin therapy are 1) history of atopic disorders 2) sensitive to equine antiserum.

Snake bites can be prevented by using boots, socks, trousers and torch light at night. On encountering a snake, it should not be disturbed, attacked or handled even if thought to be harmless.

Basudeb Chatterjee et al[1] in their study during 1977 – 1979 found that the incidence of childhood poisoning was 1.98% with the kerosene oil poisoning was at the top (44%) followed by poisoning due to medicaments and chemicals (35.7%).

Surjit Singh et al [2] in their study found that the mean age of the poisoning was 3.17 years with age ranging from 1½ months to 12 years.

More than 50% of poisoning occurred in Children of 5 years of age or younger[13]. Almost all of these exposures are unintentional and reflect the propensity for children in this age group to put virtually anything in their mouth. Poisoning exposure in children 6-12 years of age are much less common.

Niayaz A et al[8] in their study during 1983-1988 found that out of 670 cases 67.6% of cases were under the age of five years and 22.5% were infants. Only 10.4% were in the age group of 9-12 years.

Children between 1-3 years age are most vulnerable to accidental poisoning as they are mobile, inquisitive and cannot differentiate between harmful and harmless things[7].

Christo KK et al [12] in a study found that poisoning in children below 5 years age tends to be accidental and it was more often deliberate self poisoning (DSP) in older children. For this the possible risk factors are stress of school work, bullying at school, failure at school, failure in love and conflicts with parents[7].

The incidence of poisoning among male and female was 1.25:1 [8]. The high risk children are typically independent, active, restless and most often boys.[14.]

In Niayaz A et al[8] study the urban rural ratio was 1.13:1 and in Basudeb Chatterjee et al[1] study it was 3:1. This indicates that majority of children came from urban area. This could be because of transport difficulties in rural areas with majority of these children being treated at rural dispensaries or district hospitals and only more serious cases reaching at our hospital[2].

Surjit Singh et al[2] in their study found that accidental poisoning was more common in children belong to low socioeconomic group and

less common in children of upper socioeconomic group. This could be due to better environmental conditions, increased awareness among the parents of higher income group regarding poisonous potentialities of drugs and better insight regarding prevention of accidental poisoning.

MORTALITY IN CHILDREN DUE TO POISONING

Though over all mortality was low, it occurred mostly in older children[7].

Death due to unintentional poisoning in young children is uncommon[13]. Because generally they consume one mouthful of liquid poison or make a small bite of toxic agent.

Death rate was 25% in insecticide poisoning and mortality was more in snake bite. Death was more in children who reported late[32].

PREVENTION OF POISONING IN CHILDREN

Most cases of the poisoning in children could have been prevented[19]. The paediatrician or family physician can play a very vital part in the preventive aspect.

i) Kerosene should not be left in small tins within easy reach of these children. Gastric lavage is not necessary and may be harmful.

ii) Medicines and household chemicals should be locked up, labels are properly preserved and the usual practice of changing bottle containers should be avoided.

iii) School teachers should show the poisonous seeds to the students and warn them of the danger.

iv) Pesticides must not be left in easy reach of the children and in house hold pots.

v) Food previously cooked should be either discarded or heated properly. The usual practice of just warming it should be discouraged.

vi) In scorpion endemic areas and snake infested areas protective items like boots, socks and trousers should be used.

These steps will help us in preventing morbidity and mortality in children due to poisoning.

MATERIALS AND METHODS

STUDY DESIGN :

- Prospective Cohort Study design

STUDY PLACE :

- Institute of Social Paediatrics,

  Govt. Stanley Medical College and Hospital,

  Chennai-600 001.

STUDY PERIOD :

- September 2004 – August 2005.

SAMPLE SIZE :

174 cases

INCLUSION CRITERIA:

Children who were admitted with the history of any type of poisoning (Accidental / Suicidal / homicidal) were included in the study.

EXCLUSION CRITERIA

1) Unknown poisons
2) Other chronic diseases presenting with similar clinical features were excluded

METHODOLOGY :

All patients who fulfil the inclusion criteria were included in the study and by using a preset proforma, datas regarding epidemiology,

clinical profile were analysed. These patients were followed up till they were discharged and the outcome was analysed.

STATISTICAL METHODS USED

- Demographic variables, environmental factors and out comes were expressed by frequencies with their percentages.
- Difference between the incidence of poisoning were analysed by using proportion test and one group chi-square test.
- Association between different environmental factors, demographic factors and out come of the study were analysed by using Pearson Chi-square Test and Yates corrected Chi-square test.

OBSERVATION AND RESULTS

Total number of patients admitted in our paediatric ward during the study period was 3369. Number of cases admitted in paediatric intensive care unit (PICU) was 740. Among these cases admitted with the history of poisoning was 174 cases which accounted 5.16% of total admissions and 23.5% of PICU admissions.

TABLE I
SEX DISTRIBUTION

Sex	No. of Cases	Incidence	
Male	94	54%	Proportion Test Z = 1.39 P = 0.17
Female	80	46%	
Total	174	100%	

In our study 174 children were admitted with the history of poisoning. Among them, number of males 94 (54%) and females 80 (46%).

TABLE II
INCIDENCE OF POISONING IN DIFFERENT PAEDIATRIC AGE GROUPS

Age Group	No. of cases	Incidence	
< 1 year	5	2.9%	Chi Square Test $X^2 = 73.1$ P = 0.01
1-2 years	81	46.7%	
3-6 years	56	32.2%	
7 – 12 years	32	9.2%	
Total	174	100%	

All the patients were in the age group of 0-12 years. Out of that 5 (2.9%) cases were in the age group of < 1 year. 81 (46.7%) cases were in the age group of 1-2 years. 56 (32.2%) and 32 (9.2%) cases were in the age group of 3-6 years and 7-12 years respectively. Maximum number of cases (46.7%) were in the age group of 1-2 years and minimum number of cases were in the age group of < 1 year.

ENVIRONMENTAL FACTORS INFLUENCING POISONING

I) DEMOGRAPHY

TABLE III

Place	No. of Cases	Percentage
Urban	152	87.4%
Rural	22	12.6%
Total	174	100%

Among the poisoning cases the urban rural difference is given in the above table.

II) OVER CROWDING[35]

TABLE IV

Over Crowding	No. of Cases	Percentage	
Present	109	62.6%	Proportion test Z = 4.58 P = 0.001
Absent	65	37.4%	
Total	174	100%	

The above table shows that over crowding was present in 109 (62.6%) cases and there was no over crowding in 65 (37.4%) cases.

III) STORAGE FACILITIES FOR THE POISONING SUBSTANCES

TABLE V

Storage facility	No. of cases	Percentage	Proportion test Z= 16.26 P = 0.001
Available	2	1.4%	
Not available	140	98.6%	
Total	142	100%	

On analysing the storage facilities available, in 140 (98.6%) cases storage facilities were not available and it was available in 2 (1.4%) cases.

IV) SOCIO-ECONOMIC STATUS

TABLE VI

Grade	No. of Cases	Percentage
I	5	2.9%
II	34	19.5%
III	97	55.7%
IV	38	21.8%
Total	174	100%

All the cases were divided in to IV groups based on Kuppuswamy scale (modified)[34]. 5 (2.9%) cases belonged to Grade I Upper class, 34 (19.5%) cases belonged to Grade II Upper middle, 97 (55.7%) cases belonged to Grade III lower middle and 38 (21.8%) cases belonged to grade IV Upper lower socioeconomic group.

V) WORKING PARENTS

TABLE VII

Working Parents	No. of Cases	Percentage
Singe Parent Working	158	90.8%
Both the parents working	16	9.2%
Total	174	100%

It is found that 9.2% of poisoning occurred when both the parents were working and remaining 90.8% occurred when single parent was working.

VI) CARE TAKER AT THE TIME OF POISONING

TABLE VIII

Care Taker	No. of Cases	Percentage
Father	4	2.3%
Mother	145	83.3%
Grand Parents	2.3	13.2%
Others	2	11%
Total	174	100%

The analysis of the person who was looking after the child during the time of poisoning shows that in 83.3% cases mother was the caretaker and in 13.2% grand parents were the care takers and father and others were care takers in 23% and 11% of the cases respectively.

TYPE OF POISONS

TABLE IX

Type of Poisons	No. of Cases	Percentage
Kerosene oil	82	47.1%
Animal Poisons	32	18.4%
Chemical poisons	24	13.8%
Food Poisons	8	4.6%
Medicaments	7	4%
Pesticides	5	2.9%
Corrosives	3	1.7%
Miscellaneous	13	7.5%
Total	174	100%

On analysing the type of poisons, kerosene poisoning accounted for 82(47.1%) cases which was followed by animal poisons 32(18.4%) which consists of 23 cases of scorpion sting and 9 cases of snake bite. Chemical poisons accounted for 24 (13.8%) cases which consists of 20 cases of liquid fabric whitener and 4 cases of Dettol. Food poisoning accounted for 8 cases which includes 6 cases of eating previous day cooked food and 2 cases of eating rotten dove eggs. Medicament poisoning accounted for 7(4%) cases which includes 2 cases of chlorpromazine, one case each of haloperidol, benzene hexa chloride, paracetamol, nilgris oil and ayurvedhic muscle relaxant liniment. Pesticides and corrosives accounted for 5 (2.9%) and 3 (1.7%) cases respectively. Miscellaneous group consists of 13 (7.5%) cases which

includes 5 cases of mosquito coil poisoning 4 cases of camphor poisoning, 3 cases of mushroom poisoning and 1 case of oleander seed poisoning.

PURPOSE FOR WHICH THE POISONING SUBSTANCE WAS KEPT IN THE HOUSE

TABLE X

Purpose	No. of Cases	Percentage
Fuel	79	55.6%
Lighting	3	2.1%
Cleaning	8	5.6%
Washing	18	12.7%
Medicaments for illness	7	4.9%
For insects / pests	10	7.1%
Other purpose	17	12%
Total	142	100%

On analysing the purpose for which the poisoning substance was kept in the home, kerosene was used as fuel in 79 (55.6%) cases and for lighting in 3 (2.1%) of cases. Substances used for cleaning 8 (5.6%) and washing 18 (12.7%). In 7(4.9%) cases the poisoning was due to medicaments used for the purpose of various diseases (taken orally or applied externally). In 10 (7.1%) cases the poisoning was due to substances kept for various insects and pests.

NATURE OF THE EXPOSURE OF POISONING

TABLE XI

Nature of the exposure	No. of Cases	Percentage
Accidental	171	98.3%
Homicidal	2	1.1%
Suicidal	1	0.6%
Total	174	100%

On analysing the nature of the exposure of the poisoning, 171 (98.3%) cases were accidental 2 (1.1%) cases were homicidal and 1 (0.6%) case was suicidal.

MODE OF EXPOSURE OF POISONING

TABLE XII

Mode of exposure	No. of cases	Percentage
Ingestion	142	81.6%
Stings / bites	32	18.4%
Total	174	100%

The above table indicates that ingestion was the common mode of exposure of poisoning in children 142 (81.6%) which was followed by stings / bites in 32(18.4%) cases.

SYSTEM WISE INVOLVEMENT IN POISONING

TABLE XIII

System	No. of Cases	Percentage
Respiratory System	79	45.4%
Gastrointestinal System	49	28.2%
Central nervous system	13	7.5%
Cardiovascular system	10	5.7%
No system involved	23	13.2%

On analysing the system wise involvement in various poisoning respiratory system was involved in 79 (45.4%) cases, where as gastrointestinal and central nervous systems were involved in 49 (28.2%) and 13 (7.5%) cases respectively. Cardiovascular system was involved in 10 (5.7%) cases. No system was involved in 23 (13.2%) cases.

CLINICAL FEATURES OF INDIVIDUAL COMMON POISONING

TABLE XIV

Types of Poisons	Total Cases	%	Resp. Distress	Cough	Vomiting	Abd. Pain	Drowsiness /LOC/Seizures	Hyper tension	Asymp-tomatic
Kerosene oil	82	47.1	45 (54.9%)	31 (37.8%)	0	3 (3.7%)	2 (2.4%)	0	1 (1.2%)
Animal poisons	32	18.4	1 (3.1%)	0	0	0	5 (15.6%)	9 (28.1%)	17 (53.1%)
Chemical poisons	24	13.8	4 (16.7%)	0	9 (37.5%)	8 (33.3%)	0	0	3 (12.5%)
Food Poisons	8	4.6	0	0	8 (100%)	0	0	0	0
Medi-caments	7	4	0	0	4 (57.1%)	0	3 (42.9%)	0	0
Pesticides	5	2.9	3 (60%)	0	2 (40%)	0	0	0	0
Corrosives	3	1.7	0	0	2 (66.7%)	1 (33.3%)	0	0	0

On analysing the various clinical features of individual poisoning, kerosene poisoning presented with respiratory distress in 45(54.9%) cases, cough in 31 cases (37.8%), abdominal pain in 3 (3.7%) cases and drowsiness in 2 (2.4%) cases. Among animal poisons 1(3.1%) case of snake bite was presented with respiratory distress and 5 (15.6%) cases were presented with LOC. 3(9.3%) cases of snake bites were asymptomatic. 9(28.1%) scorpion sting cases were presented with hypertension and 14 cases of scorpion stings were asymptomatic. In chemical poisoning 9(37.5%) cases were presented with vomiting, 8(33.3%) presented with abdominal pain and 4(16.7%) cases presented

with respiratory distress. 3 (12.5%) cases were asymptomatic. In food poisoning all cases 8(100%) presented with vomiting. In medicaments poisoning 4 (57.1%) cases presented with vomiting and 3 (42.9%) cases presented with drowsiness. In pesticides poisoning respiratory distress in 3 (60%) cases and vomiting in 2 (40%) cases. In corrosive poisoning 2 (66.7%) cases were presented with vomiting and one case (33.3%) was presented with abdominal pain.

DURATION OF STAY IN PICU

TABLE XV

Type of Poisons	No. of Cases	Percentage	Duration in PICU		
			< 24 hrs	24 – 48 hrs	> 48 hrs
Kerosene oil	82	47.1%	4 (4.9%)	75 (91.5%)	3 (3.6%)
Animal poisoning	32	18.4%	1 (3.1%)	28 (87.5%)	3 (9.4%)
Chemical Poisoning	24	13.8%	5 (20.8%)	19 (79.2%)	0
Food Poisons	8	4.6%	0	8 (100%)	0
Medicaments	7	4%	0	7 (100%)	0
Pesticides	5	2.9%	2 (40%)	2 (40%)	1 (20%)
Corrosives	3	1.7%	0	1 (33.3%)	2 (66.7%)

The above table shows the average duration of stay in PICU for most of the poisoning cases was 24-48 hrs.

OUTCOME

TABLE XVI

Outcome	No. of Cases	Percentage
No. Sequelae	19	97.2%
With Sequelae	2	1.1%
Death	3	1.7%

On analysing the out come of various poisoning in this study, 109(97.2%) cases recovered without any Sequelae. 2(1.1%) cases of acid poisoning recovered with Sequelae of severe oesophagitis and gastritis. They were followed up regularly in medical gastro enterology department. No. of mortality was 3(1.7%), 1 case of snake bite and 2 cases of pesticides poisoning.

DISCUSSION

In this urban tertiary paediatric hospital 5.16% of total admission was due to poisoning. Poisoning cases accounted for 23.5% of PICU admissions.

The incidence of poisoning in various studies are given below :

Study	Incidence
Buhariwala RJ et al (1962 – 1966)	7.64%
Basudeb Chatterjee et al (1977 – 1979)	1.98%
Kumar V et al (1984 – 1988)	1.8%
Ganga N et al (1996 – 1999)	6.9%
PRESENT STUDY (2004 – 2005)	**5.16%**

The above table shows that the incidence of poisoning in the present study is similar to other studies.

In the present study the male female ratio was 1.2:1. Though there was marginal increase in the incidence of poisoning in males the statistical analysis does not show any significant difference.

Male female ratio in other studies are given below :

Study	Male : Female
Buhariwalla RJ et al (1962 – 1966)	2:1
Sitaram S et al (1982 – 1984)	3.2:1
Niayaz A et al (1983-1988)	1.25:1
Kumar V et al (1984-1988)	2:1
Khadgawat R et al (1987 – 1993)	1.6:1
Ganga N et al (1996 – 1999)	1.5 : 1
PRESENT STUDY (2004-2005)	**1.2:1**

The above table shows the male female ratio in the present study is similar to other studies.

On analysing the incidence of poisoning in different paediatric age groups, 46% cases occurred in 1-2 years of age group followed by 32.2% cases in 3-6 years of age group. Least incidence was in infants (2.9%). It is found that significant high incidence of poisoning was seen in the age group of 1-2 years.

On analysing various environmental factors which were influencing the poisoning it is found that 9.2% of poisoning occurred when both the parents were working. Remaining 90.8% occurred when single parent was working. The percentage of single parent working is very high in our society. Hence this figure cannot be considered significant without a case control study.

Significantly high incidence of poisoning (55.7%) was found in the grade III lower middle class of Kuppusamy scale when compared to other grades and significantly less incidence (2.9%) was found in grade I upper class.

Among the urban and rural population, statistically high incidence of poisoning (87.4%) was found in the urban population. Since our centre is situated in the urban area these figures may be misleading.

During the time of poisoning, 83% of cases were looked after by their mother when compared to grand parents (13.2%) and father (2.3%). The high incidence of this figure is due to, in most of the families mother is looking after the children and doing household works like washing and cooking.

Presence of over crowding (in 62.6% cases) and lack of storage facilities (in 98.6% cases) were also found to be statistically significant risk factors for poisoning in children.

On analysing the various types of poisoning the incidence of kerosene oil poisoning was the highest (47.1%) which was followed by envenomation (18.4%), chemical poisoning (1.38%) and food poisoning (4.6%). The incidence of poisoning due to medicaments, pesticides and corrosives were 4%, 2.9% and 1.7% respectively.

Santhana Krishnan B.R. et al [24] observed the following types of poisoning in a study. Kerosene oil in 32.5%, drugs in 24.9%, vegetable alkaloids in 17.3%, Neem oil in 12.4% and miscellaneous in 12.9% of cases.

Khadgawat R. et al [5] in their study observed the following types of poisoning. Kerosene oil (48.8%), medicaments (11.7%), snake bite (11.2%), dhatura (8.1%), food poisoning (7.2%) and paris green (3.1%).

Buhari walla R.J. et al [15] observed the following types of poisoning. Kerosene oil poisoning in 59.7%, chemicals and drugs in 13.7%, vegetable poisons in 9.7%, food poisoning in 6.6%, pesticides in 6.3% and miscellaneous in 4%.

The commonest poisoning in the present study was kerosene oil poisoning which is similar to the above mentioned various studies.

Incidence of Kerosene Oil Poisoning in various studies are given below :

Study	**Incidence**
Buhairwalla et al (1962-1966)	59.7%
Reddi YR et al (1963 – 1964)	40.3%
Basudeb chatterjee et al (1977-1979)	44 %
Sitaraman S. et al (1982-1984)	33.7%
Khadgawat R et al (1987-1993)	48.8%
PRESENT STUDY (2004-2005)	**47.1%**

The above table shows the incidence of kerosene poisoning in the present study is similar to various other studies.

In our study the most of the poisoning incidence happened in side the house and most of the poisoning substances were household products . Kerosene was used as a fuel in 55.6% cases and for lighting in 2.1% cases, various chemicals were used for cleaning (5.6%) and

washing (12.7%) purposes. Medicaments for various illness were used in 4.9% cases. Various substances for insects and pests were used in 7.1% cases.

On analysing the nature of exposure, in 98.3% cases the poisoning was accidental. In 1.1% cases it was homicidal and in 0.6% case it was suicidal. This observation is similar with the Surjit Singh et al [7] study.

On analysing the mode of exposure of poisoning the most common mode of exposure was ingestion in 81.6% cases which was followed by Stings / bites in 18.4% cases.

Ingestion is the most common route of poisoning exposure[13].

On analysing the systems involved in various poisoning, respiratory system was most commonly involved (45.4%) which was followed by gastrointestinal system (28.2%), central nervous system (7.5%) and cardiovascular system (5.7%).

On analysing the clinical features of individual common poisons, kerosene poisoning was presented with respiratory distress in 54.9%, cough in 37.8%, abdominal pain 3.7%, drowsiness in 2.4% and asymptomatic in 1.2% cases.

Among animal poisoning 15.6% snake bite cases were presented with LOC and 28.1% scorpion stings were presented with hypertension. Remaining 56.3% animal poisoning cases were asymptomatic.

Chemical poisoning were presented with vomiting in 37.5%, respiratory distress in 16.7%, abdominal pain in 33.3%, and asymptomatic in 12.5% cases. All food poisonings (100%) were presented with vomiting. In medicament poisoning 57.1% had vomiting and 42.9% had drowsiness. In pesticides poisoning 60% had respiratory distress and 40% had vomiting. In corrosive poisoning 66.7% had vomiting and 33.3% had abdominal pain.

In Basudeb Chatterjee et al[1], the clinical features of Kerosene poisoning were Kerosene Odour in 89.2% cases, vomiting in 81% cases, cough in 78.4% cases, fever in 51.3% cases, lung crepitations in 10.8% cases and restlessness in 2.7% case.

In Reddi Y.R. et al[33] the clinical features of kerosene poisoning were respiratory system features in 47.7% cases, vomiting in 22.7% cases, central nervous system features in 33.3% cases and fever in 4.7% cases.

Santhanakrishnan BR et al[24] study found the following clinical features due to kerosene oil poisoning : vomiting (25%) cough (13%) grunting (5%), abdominal pain (3%) breathlessness (13%).

On analysing the duration of stay in PICU the average duration of stay of many poisoning cases was 24-48 hrs.

On analysing the out come of paediatric poisoning in the study, we noted favourable out come in 97.2% cases, Sequelae in 1.1% cases and death in 1.7% cases, which shows, effective management of poisoning cases reduces mortality and morbidity.

Mortality due to poisoning in various study are given below.

Study	**Mortality rate**
Reddi YR et al (1963-1964)	1.3%
Santhanakrishnan B.R. et al (1970)	7%
Surjit singh et al (1970-1979)	12.5%
Surjitsingh et al (1980-1989)	4.8%
Sitaraman S et al (1982-1984)	2.4%
Niayaz A et al (1983-1988)	1.8%
Kumar V et al (1984-1988)	3%
PRESENT STUDY (2004-2005)	**1.7%**

The above table shows the mortality rate in the present study is at par with other studies.

SUMMARY AND CONCLUSIONS

1) The incidence of poisoning in this study was 5.16% which accounted for 23.5% of PICU admissions.

2) There was no difference in the incidence of poisoning between male and female.

3) The commonest age group affected due to poisoning was 1-2 years of age (46%).

4) The highest incidence of poisoning was observed in lower middle socio-economic group (55.7%) and lowest incidence in upper socioeconomic group (2.9%).

5) Over crowding and lack of storage facilities increased the risk of poisoning in children.

6) The commonest nature of exposure was accidental (98.3%) which was followed by homicidal (1.1%) and suicidal (0.6%).

7) The commonest mode of exposure was ingestion (81.6%) which was followed by stings / bites (18.54%)

8) The commonest type of poisoning was kerosene oil ingestion (47.1%) which was followed by envenomation (18.4%) and chemical poisoning (13.8%).

9) Common clinical features of kerosene oil poisoning were respiratory distress (54.9%) and cough (37.8%).

10) The commonest system involved in poisoning in children was respiratory system (45.4%) which was followed by gastrointestinal system (28.2%) and central nervous system (7.5%).

11) The average duration of stay in PICU for most of the poisoning cases was 24-48 hrs.

12) The mortality rate in children due to poisoning was 1.7%. The morbidity rate was 1.1% and good out come in 97.2% cases.

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KEY TO MASTER CHART

1) Sl.No.
2) Name
3) Age
    1. < 1 year
    2. 1- 2 years
    3. 3-6 years
    4. 6 – 12 years
4) Sex
    1. Male
    2. Female
5) Working parents
    1. Father
    2. Mother
    3. Both
6) Socioeconomic Status
    1. Upper class
    2. Upper middle
    3. Lower middle
    4. Upper lower
    5. Lower
7) Care taker at the time of poisoning
    1. Father
    2. Mother
    3. Grand parents
    4. Others
8) Over crowding
    1. Present
    2. Absent
9) Storage Facilities
    1. Available
    2. Not available
    3. Not applicable
10) Purpose of keeping the poisoning substance at home
    1. Fuel
    2. Lighting
    3. Cleaning
    4. Washing
    5. Medicaments for illness
    6. For insects and pests
    7. Other purpose
    8. Not applicable
11) Demography
    1. Urban

2. Rural

12) Nature of Exposure
   1. Accidental
   2. Homicidal
   3. Sucidal

13) Type of poisons
    1. Hydrocarbons
    2. Chemicals
    3. Medicaments
    4. Animal poisons
    5. Corrosives
    6. Food poisons
    7. Pesticides
    8. Miscellaneous
14) Mode of Exposure
    1. Ingestion
    2. Stings / bites
15) First aid given at home
    1. Vomiting induced
    2. Vomiting not induced
    3. Tornique applied
    4. Lime / Onion applied
    5. Nothing done at home
16) Time duration between poisoning and reporting to hospital
    1. < 30 mins
    2. 30 – 60 mins
    3. 1-2 hrs
    4. 2-3 hrs
    5. 3-24 hrs
    6. > 24hrs
17) Presenting Features
    1. Cough
    2. Respiratory distress
    3. Vomiting
    4. Fever
    5. Abdominal pain
    6. Drowsiness / altered sensorium / seizures / loss of consciousness
    7. Respiratory paralysis
    8. Hypertension
    9. Asymptomatic
18) Duration in ICU
    1. < 24 hrs
    2. 24-48 hrs
    3. > 48 hrs
19) Duration in General Ward
    1. < 1 day
    2. 1-2 days
    3. 2-3 days

4. 3-4 days
5. > 4 days

20) System involved
1. Respiratory system
2. Cardiovascular system
3. Gastrointestinal system
4. Central nervous system
5. No system involved

21) Investigations
1. X-ray changes
2. ECG / Echo changes
3. Endoscopy changes
4. Normal investigations

22) Outcome
1. Improved without sequlae
2. Improved with sequlae
3. Died

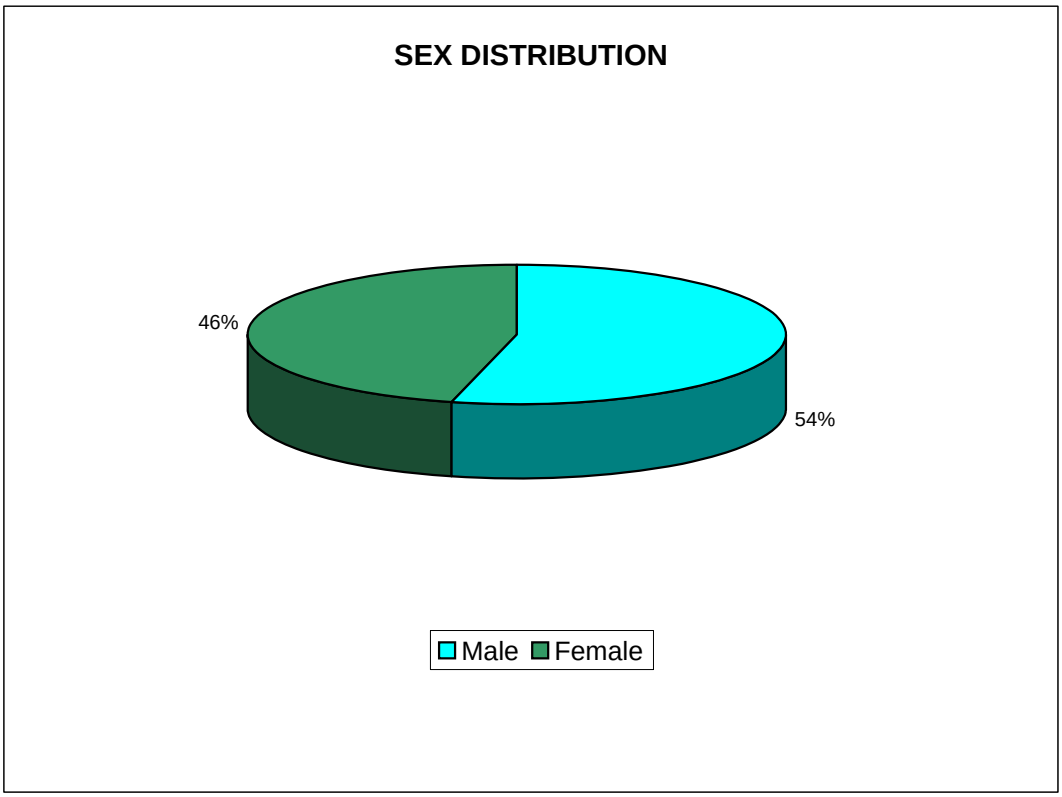
MASTER CHART																				
Sl. No.	Name	Age	Sex	Working Parents	Socioeconomic Status	Care taker at the time of poisoning	Overcrowding	Storage facilities	Purpose of keeping the poisoning substance at home	Demography	Nature of exposure	Type of Poisons	Mode of Exposure	First aid given at home	Time duration between poisoning and reporting to hospital	Presenting features	Duration of ICU	Duration in General ward	System Involved	Outcome
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	22
1	Arul Raj	3	1	1	2	2	1	2	3	1	1	2	1	1	2	5	2	2	3	1
2	Pavithra	3	2	1	3	2	2	2	7	1	1	8	1	1	2	9	1	1	5	1
3	Akash	2	1	1	4	2	1	2	5	1	1	3	1	5	3	3	2	2	3	1
4	Manigandan	3	1	1	3	2	1	2	5	1	1	3	1	1	2	3	2	2	3	1
5	Prasanth	2	1	3	2	3	1	2	6	1	1	8	1	5	3	5	2	2	3	1
6	Hemalatha	4	2	1	3	2	2	3	8	2	1	6	1	1	3	3	2	2	3	1
7	Sanjay	2	1	1	2	2	1	2	3	1	1	2	1	1	2	3	2	2	3	1
8	Ajeeth	3	1	1	2	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
9	Abubakkar	2	1	1	3	2	2	2	1	1	1	1	1	5	2	2	2	3	1	1
10	Jenardhanam	2	1	1	3	2	1	2	1	1	1	1	1	1	3	2	2	4	1	1
11	Selvam	3	1	3	2	3	1	2	1	1	1	1	1	5	2	1	2	2	1	1
12	Deepika	2	2	1	3	2	1	2	7	1	1	8	1	5	2	3	2	2	3	1
13	Keerthika	2	2	1	4	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
14	Sujitha	3	2	1	3	2	2	2	7	1	1	8	1	1	3	3	1	1	3	1
15	Gokul	3	1	1	2	2	2	3	8	1	1	4	2	4	3	9	2	2	5	1
16	Karthik	4	1	3	3	3	1	2	4	1	1	2	1	1	2	5	2	2	3	1
17	Kowsalya	2	2	1	4	2	1	2	7	1	1	8	1	5	2	9	2	2	5	1
18	Vasanthakumar	2	1	1	3	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
19	Vanmathi	2	2	1	2	2	2	2	4	1	1	2	1	1	3	9	1	1	5	1
20	Velan	2	1	3	3	3	1	2	1	1	1	1	1	5	2	1	2	2	1	1
21	Rashma	2	2	1	4	2	2	2	1	1	1	1	1	1	2	2	2	3	1	1
22	Sanjay	3	1	1	3	2	2	2	1	1	1	1	1	5	2	1	2	2	1	1
23	Yuvashree	2	2	1	2	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
24	Renuka	4	2	1	3	2	2	2	1	1	3	1	1	5	2	5	2	2	3	1
25	Nithya	2	2	1	3	2	1	2	1	1	1	1	1	1	2	1	2	2	1	1
26	Meenakshi	4	2	3	4	3	1	2	1	1	1	1	1	1	3	2	2	3	1	1
27	Akash	3	1	1	3	2	2	2	4	1	1	2	1	1	2	3	2	2	3	1

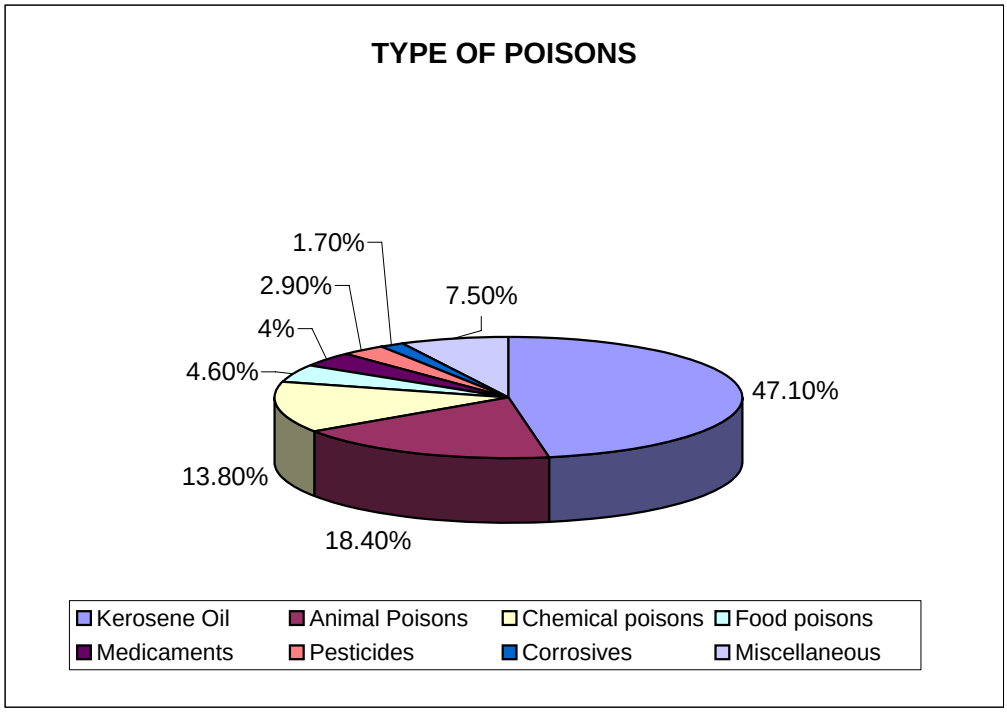
28	Alex	2	1	1	2	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
29	Suganya	2	2	1	3	2	1	2	4	1	1	2	1	1	2	2	2	2	3	1
30	Sowmiya	3	2	1	3	2	1	3	8	2	1	4	2	3	3	9	2	2	5	1
31	Arasu	2	1	1	4	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
32	Jenefer	2	2	3	3	3	1	2	1	1	1	1	1	5	2	1	2	2	1	1
33	Vijay	2	1	1	3	2	2	2	1	1	1	1	1	1	2	2	2	4	1	1
34	Nirmala	1	2	1	3	2	2	2	6	1	1	8	1	1	2	3	2	2	3	1
35	Sharmini	2	2	3	2	3	1	2	1	1	1	1	1	1	2	2	2	3	1	1
36	Logesh Kumar	2	1	1	3	2	1	2	1	1	1	1	1	5	2	2	2	4	1	1
37	Janani	2	2	1	3	2	2	3	8	2	1	4	2	3	3	9	2	2	5	1
38	Sanjay	3	1	1	2	2	1	2	4	1	1	2	1	1	2	5	2	2	3	1
39	Vetrivelu	4	1	1	3	2	1	2	6	2	1	7	1	1	3	2	1	1	1	3
40	Suresh	3	1	1	4	2	2	3	8	1	1	4	2	4	2	9	2	2	5	1
41	Nivedha	3	2	3	3	3	1	2	4	1	1	2	1	1	2	5	2	2	3	1
42	Ragul	2	1	1	2	2	2	2	1	1	1	1	1	1	2	2	2	3	1	1
43	Parameshwari	4	2	1	3	2	2	3	8	1	1	4	2	3	2	9	2	2	5	1
44	Sandhya	2	2	1	4	2	2	2	1	1	1	1	1	1	2	2	2	3	1	1
45	Hussaina	2	2	1	3	2	1	2	6	1	1	8	1	1	2	3	2	1	3	1
46	Dhanush kumar	2	1	1	2	2	1	2	1	1	1	1	1	5	2	2	2	3	1	1
47	Gowri	2	2	1	4	2	1	2	1	1	1	1	1	1	2	2	2	4	1	1
48	Janani	4	2	3	3	2	2	3	8	2	1	4	2	3	3	8	2	3	2	1
49	Akash	2	1	1	2	1	1	2	1	1	1	1	1	1	1	1	1	2	1	1
50	Jayachandran	2	1	3	1	2	1	2	1	1	1	1	1	1	6	2	2	3	1	1
51	Hema	2	2	1	3	2	1	2	1	1	1	1	1	1	1	1	2	2	1	1
52	Naresh	2	1	1	2	2	2	2	1	1	1	1	1	1	2	2	2	3	1	1
53	Senthil	3	1	2	3	3	1	2	5	1	1	3	1	5	3	6	2	2	4	1
54	Md. Ibrahim	2	1	1	4	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
55	Priyanka	2	2	1	3	2	1	2	1	1	1	1	1	5	1	1	1	2	1	1
56	Pavithra	3	2	1	2	2	1	3	8	1	1	4	2	5	2	9	2	2	5	1
57	Ruban	2	1	1	3	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
58	Surekha	4	2	1	4	2	2	3	8	1	1	4	2	4	2	9	2	2	5	1
59	Vignesh	3	1	1	3	2	1	3	8	2	1	4	2	5	3	8	2	3	2	1
60	Dhanasekar	4	1	1	3	2	2	3	8	1	1	4	2	5	2	9	2	2	5	1
61	Vasanth	4	1	1	2	2	1	2	4	1	1	2	1	1	2	5	2	2	3	1
62	Varshan	2	1	1	3	2	2	2	4	1	1	2	1	5	2	5	2	2	3	1
63	Balaji	3	1	1	4	2	1	2	1	1	1	1	1	1	2	2	3	4	1	1
64	Pooja	2	2	1	1	2	1	2	1	1	1	1	1	1	2	5	2	2	3	1
65	Senthilkumar	4	1	1	2	2	1	2	6	1	1	8	1	1	2	5	2	2	3	1
66	Amerunissa	3	2	1	3	3	1	2	4	1	1	2	1	1	2	3	2	2	3	1
67	Salbunisha	2	2	1	3	2	1	2	1	1	1	1	1	5	2	1	2	2	1	1

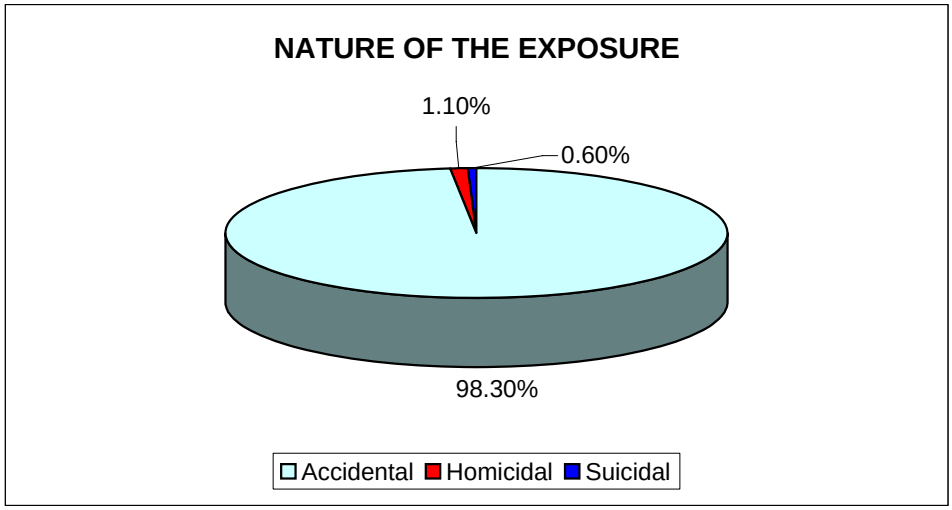
68	Sathish	2	1	1	4	2	2	2	1	1	1	1	1	1	3	2	2	4	1	1
69	Arunkumar	2	1	1	3	3	1	2	1	1	1	1	1	5	2	1	2	2	1	1
70	Kalaivani	4	2	1	2	2	1	2	7	1	1	6	1	5	5	3	2	2	3	1
71	Kasthuri	4	2	1	3	2	1	2	7	1	1	6	1	5	5	3	2	2	3	1
72	Karthik	3	1	1	4	2	1	2	7	1	1	6	1	5	5	3	2	2	3	1
73	Wesley	3	1	1	3	2	2	3	8	2	1	4	2	5	2	9	2	2	5	1
74	Surya	3	1	1	2	2	2	2	7	1	1	6	1	5	5	3	2	2	3	1
75	Thilothammal	4	2	1	3	2	2	2	7	1	1	6	1	5	5	3	2	2	3	1
76	Prakash	3	1	1	4	2	1	2	3	1	1	2	1	1	2	3	2	2	3	1
77	Abinaya	2	2	1	3	2	1	2	4	1	1	2	1	5	2	9	1	1	5	1
78	Lavanya	2	2	1	2	2	1	2	1	1	1	1	1	5	2	1	1	2	1	1
79	Yogesh	2	1	1	3	3	2	2	4	1	1	2	1	1	2	3	2	2	3	1
80	Nivetha	2	2	1	4	2	1	2	1	1	1	1	1	5	2	2	2	2	1	1
81	Pradeep	2	1	1	2	2	2	3	8	1	1	4	2	5	2	8	2	3	2	1
82	Sneka	3	2	1	3	2	1	2	1	1	1	1	1	5	2	1	2	1	1	1
83	Brinda Rachal	3	2	1	2	2	2	2	1	1	1	1	1	1	2	2	2	2	1	1
84	Vijayakumar	3	1	2	3	1	1	2	6	1	1	7	1	1	2	2	1	1	1	3
85	Gokula Kannan	3	1	3	4	2	2	3	1	1	1	1	1	5	2	2	2	2	1	1
86	Kanchana	3	2	1	1	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
87	Abidha	3	2	1	2	2	1	2	4	1	1	2	1	1	2	3	2	1	3	1
88	Prakash Raj	3	1	1	3	2	2	2	5	1	1	3	1	5	1	6	2	3	4	1
89	Rekha	3	2	3	3	3	2	2	5	1	1	3	1	1	2	6	2	3	4	1
90	Natheem	3	1	1	2	2	1	3	8	1	1	4	2	5	2	9	2	2	5	1
91	Oliyavan	2	1	1	3	2	1	2	1	1	1	1	1	1	6	2	2	4	1	1
92	Naveen	2	1	1	4	2	2	2	7	1	1	8	1	1	2	3	2	2	3	1
93	Keerthana	2	2	1	3	2	1	2	1	1	1	1	1	1	2	2	3	5	1	1
94	Priya	3	2	1	2	2	1	2	1	1	1	1	1	1	1	9	1	1	5	1
95	Sibi	2	1	1	3	2	2	2	3	1	1	5	1	5	2	3	3	3	3	2
96	Rashna	3	2	1	3	2	2	3	8	1	1	4	2	4	2	8	2	3	2	1
97	Bashra Banu	1	2	1	2	2	1	2	1	1	1	1	1	5	2	1	2	2	1	1
98	Usena	2	2	1	3	2	2	2	1	1	1	1	1	1	2	1	2	2	1	1
99	Sridevi	2	2	1	4	2	2	2	1	1	1	1	1	5	1	1	2	2	1	1
100	Sujatha	4	2	1	2	2	2	3	8	2	1	4	2	3	2	6	3	5	4	1
101	Jegan	4	1	1	3	2	1	3	8	2	1	4	2	4	2	9	2	2	5	1
102	Sriballu	2	1	1	3	2	1	2	7	1	1	8	1	5	3	6	2	2	4	1
103	Amudha	4	2	1	2	2	2	3	8	2	1	4	2	3	6	2	3	3	4	1
104	Magesh	2	1	1	4	3	1	2	1	1	1	1	1	1	2	1	2	2	1	1
105	Manimegalai	2	2	1	3	2	1	2	1	1	1	1	1	1	1	2	2	2	1	1
106	Sowmiya	3	2	1	4	2	1	2	2	1	1	1	1	1	2	2	2	3	1	1
107	Chandru	2	1	1	3	2	2	2	2	1	1	1	1	5	5	1	2	2	1	1

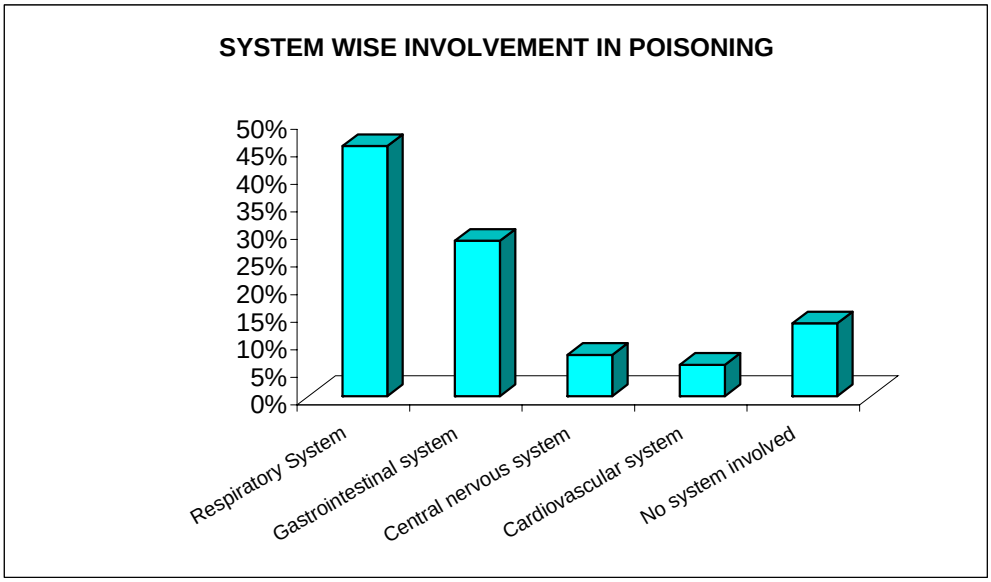
108	Iswarya	4	2	1	3	2	1	3	8	1	1	8	1	5	1	5	2	2	3	1
109	Karthik	4	1	1	3	2	1	3	8	1	1	6	1	1	5	3	2	2	3	1
110	Vinodh Kanna	4	1	1	2	2	1	3	8	1	1	6	1	1	5	3	2	2	3	1
111	Jeethan	3	1	1	3	2	1	2	1	1	1	1	1	5	1	1	2	2	1	1
112	Sriram	2	1	1	1	2	2	2	1	1	1	1	1	1	1	6	2	5	4	1
113	Logesh Kumar	3	1	2	4	1	1	3	8	2	1	4	2	4	3	9	2	2	4	1
114	Pavithra	3	2	2	3	3	1	2	1	1	1	1	1	5	5	2	2	2	1	1
115	Ragulraj	2	1	1	4	3	1	2	1	1	1	1	1	5	1	2	2	2	1	1
116	YuvaSree	3	2	1	3	2	1	3	8	1	1	4	2	4	2	8	2	3	2	1
117	Monica	2	2	1	2	2	2	2	4	1	1	2	1	1	2	3	2	2	3	1
118	Vishali	3	2	1	3	2	1	2	1	1	1	1	1	1	5	2	2	2	1	1
119	Sathyapriya	2	2	1	3	3	2	2	1	1	1	1	1	1	2	1	2	2	1	1
120	TamilKumaran	2	1	1	4	4	1	2	1	2	1	1	1	5	5	2	2	4	1	1
121	Thenmozhi	3	2	1	3	2	1	2	5	1	1	3	1	5	1	3	2	2	3	1
122	Geetha Rani	4	2	3	3	3	2	3	8	2	1	4	2	3	6	6	3	5	4	1
123	Shaik	2	1	1	4	2	2	2	4	1	1	2	1	5	2	5	2	2	3	1
124	Kalaiarasi	3	2	2	3	2	2	1	6	2	2	7	1	1	4	3	3	3	3	1
125	Saranya	3	2	2	3	2	2	1	6	2	2	7	1	1	4	2	2	3	2	1
126	Shyamsundar	3	1	1	4	2	1	3	8	1	1	4	2	5	1	9	2	2	5	1
127	Sathiya	3	2	1	3	2	1	2	1	1	1	1	1	1	6	2	2	3	1	1
128	Thenmozhi	4	2	1	3	4	2	3	8	2	1	4	2	3	4	6	1	1	4	3
129	Arjun	3	1	1	4	2	1	2	4	1	1	2	1	1	1	5	1	2	3	1
130	Miruthila	2	2	1	3	2	1	2	1	1	1	1	1	2	2	2	2	3	1	1
131	Dhanush kumar	2	1	3	2	3	1	2	1	1	1	1	1	2	6	2	2	3	1	1
132	Pavithra	3	2	1	3	2	2	2	1	1	1	1	1	2	2	1	2	2	1	1
133	Santhosh	2	1	1	3	2	2	2	1	1	1	1	1	2	2	6	2	2	4	1
134	Magesh	3	1	1	3	2	2	3	8	1	1	4	2	4	2	8	2	3	2	1
135	Pooja	1	2	1	4	2	2	2	1	1	1	1	1	2	2	2	2	3	1	1
136	Vignesh	2	1	1	3	2	1	2	1	1	1	1	1	1	2	2	2	2	1	1
137	Sneka	2	2	1	3	2	2	2	6	2	1	7	1	1	3	3	2	2	3	1
138	Dillikumar	2	1	1	3	2	2	3	8	2	1	4	2	5	3	8	2	3	2	1
139	Divya	4	2	3	4	3	2	3	8	1	1	4	2	3	2	9	2	2	5	1
140	Soundharya	2	2	1	3	2	1	2	7	1	1	8	1	1	3	6	2	2	4	1
141	Goverdhanan	4	1	1	3	2	1	3	8	1	1	4	1	4	2	8	2	3	2	1
142	Thagera	3	2	1	4	2	1	2	4	1	1	2	1	5	2	9	1	2	5	1
143	Sanjaykumar	2	1	1	3	2	1	2	1	1	1	1	1	5	2	1	2	2	1	1
144	Vidya	3	2	1	3	2	2	2	5	2	1	3	1	1	3	3	2	2	3	1
145	Ranjita	3	1	1	3	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
146	Sanjay	2	1	1	4	2	1	2	3	1	1	5	1	5	1	3	2	3	3	1
147	Anitha	4	2	1	4	2	1	2	3	1	1	5	1	5	2	5	3	4	3	2

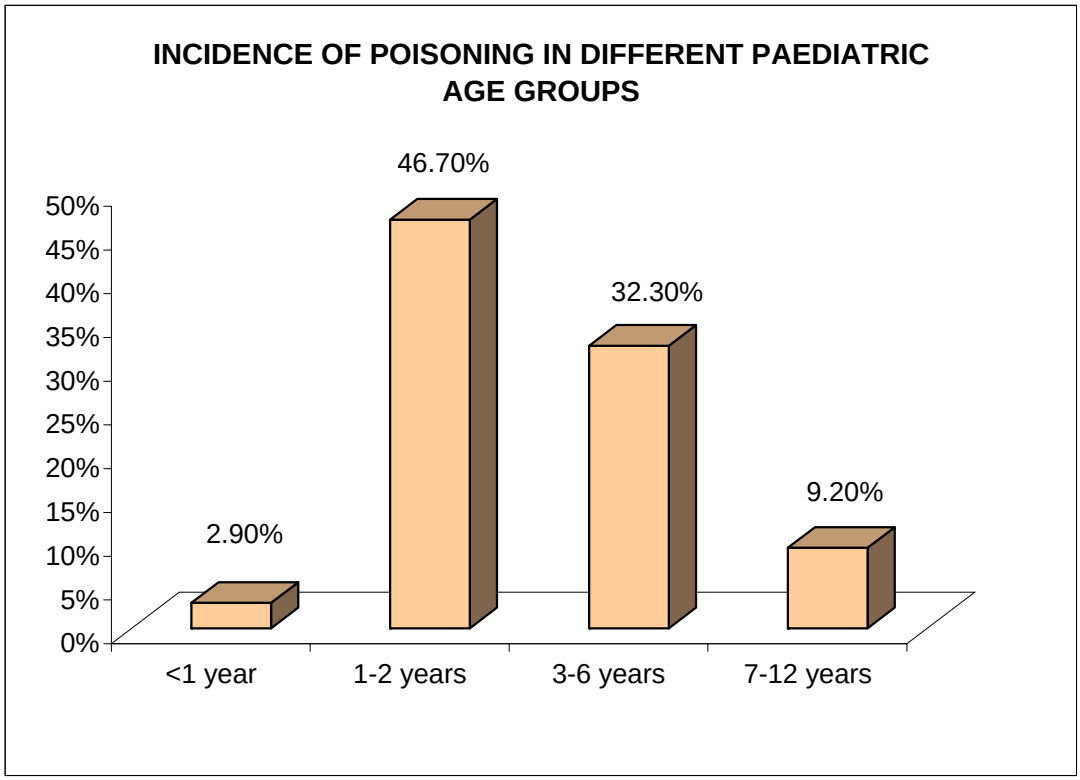
148	Rosi	4	2	2	3	2	1	2	1	1	1	1	1	5	1	5	2	2	3	1
149	Praveen	3	1	1	1	2	1	2	2	1	1	1	1	1	1	1	2	3	1	1
150	Gowreswaran	2	1	1	4	2	1	2	1	1	1	1	1	1	2	1	2	2	1	1
151	Jayasurya	1	1	1	3	2	2	2	1	1	1	1	1	1	2	2	2	3	1	1
152	Vishnupriya	3	2	1	3	2	1	2	3	1	1	2	1	1	2	2	1	2	3	1
153	Praveena	2	2	1	4	2	2	2	1	1	1	1	1	5	1	1	2	2	1	1
154	Senthil	2	1	1	3	2	2	3	8	2	1	4	2	3	3	9	2	2	5	1
155	Arul Raj	2	1	2	3	3	2	2	7	1	1	2	1	1	2	3	2	2	4	1
156	Sathiskumar	3	1	1	2	2	1	2	1	1	1	1	1	1	2	1	2	3	1	1
157	Manimaran	4	1	1	3	2	1	3	8	1	1	4	2	4	2	6	2	2	5	1
158	Karan raj	3	1	3	3	3	1	3	8	1	1	4	2	5	2	6	2	2	5	1
159	Bhaath	3	1	1	4	2	2	3	8	2	1	4	2	3	3	9	2	2	5	1
160	muthukumar	4	1	1	3	2	1	3	8	1	1	4	2	4	2	8	2	3	2	1
161	Vishnupriya	2	2	1	2	3	1	2	3	1	1	2	1	1	1	3	2	1	3	1
162	Abirami	2	2	1	3	2	1	2	1	1	1	1	1	5	2	1	2	2	1	1
163	Akash	2	1	1	3	2	1	2	1	1	1	1	1	1	2	1	2	3	1	1
164	Prasanth	2	1	1	4	2	1	2	1	1	1	1	1	1	2	1	2	3	1	1
165	Rajasekar	3	1	1	3	2	2	2	4	1	1	2	1	1	2	2	2	2	3	1
166	Vimalraj	4	1	1	3	2	2	2	6	1	1	8	1	1	2	3	2	2	3	1
167	Prasanth	2	1	1	4	2	1	2	1	1	1	1	1	1	2	2	2	3	1	1
168	Vinodh	2	1	1	3	2	1	2	1	1	1	1	1	2	1	1	2	2	1	1
169	Sweetha	2	2	2	3	1	1	2	1	1	1	1	1	2	2	2	2	2	1	1
170	Sri	2	1	1	4	2	1	2	1	1	1	1	1	2	2	1	2	2	1	1
171	Berlin	1	1	1	3	2	1	2	1	1	1	1	1	2	2	1	2	2	1	1
172	Pavithra	4	2	1	3	2	2	2	4	1	1	2	1	2	2	2	2	1	3	1
173	Akash	4	1	1	3	2	1	2	1	1	1	1	1	1	2	2	3	3	1	1
174	Shankar	4	1	1	3	2	2	3	8	2	1	4	2	3	2	9	2	2	5	1


SEX DISTRIBUTION
46%
54%
Male
Female

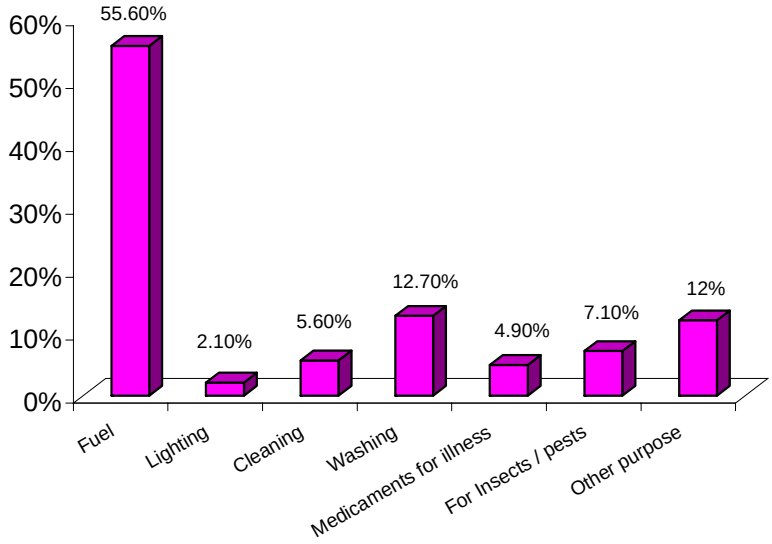

TYPE OF POISONS
1.70%
2.90%
7.50%
4%
4.60%
47.10%
13.80%
18.40%
Kerosene Oil
Animal Poisons
Chemical poisons
Food poisons
Medicaments
Pesticides
Corrosives
Miscellaneous

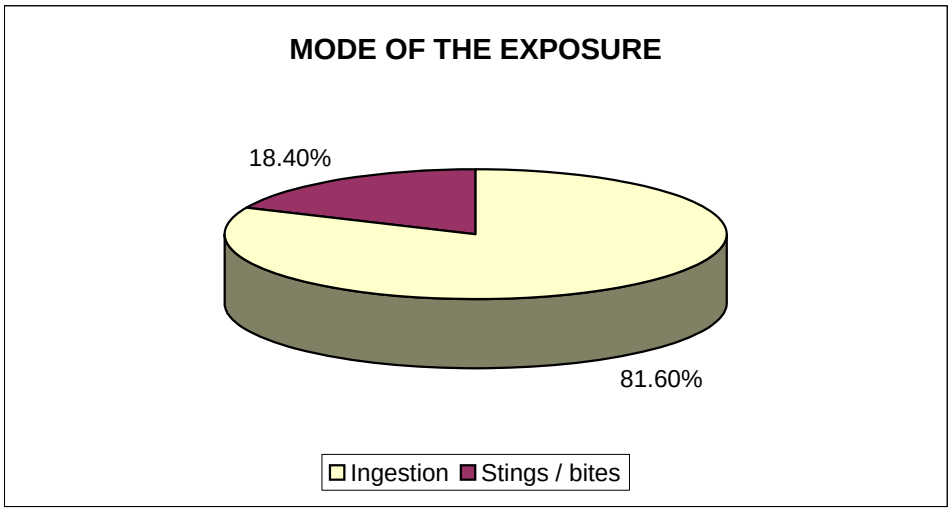

NATURE OF THE EXPOSURE
1.10%
0.60%
98.30%
Accidental
Homicidal
Suicidal

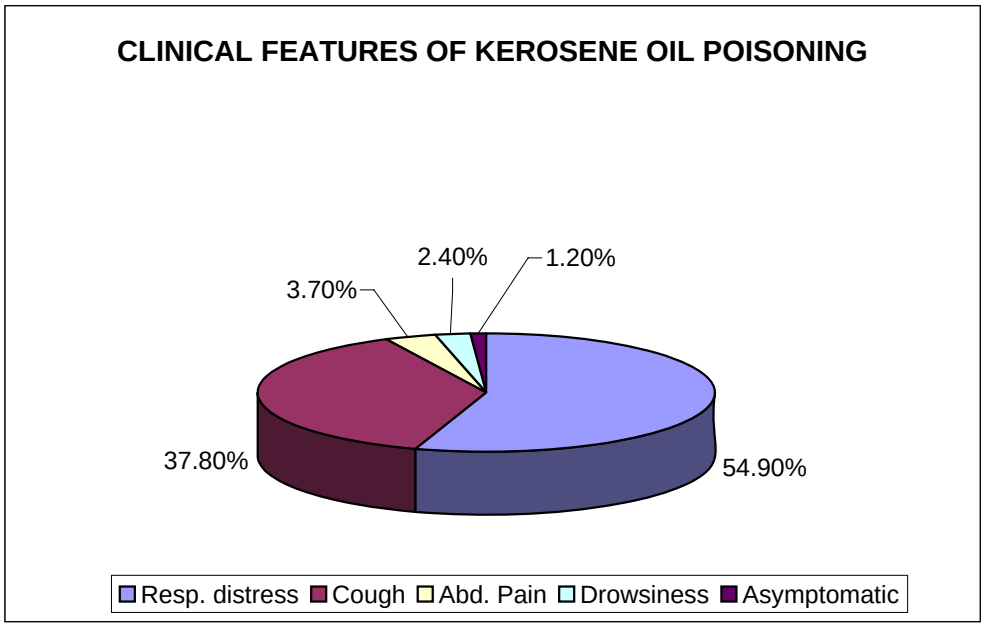

SYSTEM WISE INVOLVEMENT IN POISONING
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
Respiratory System
Gastrointestinal system
Central nervous system
Cardiovascular system
No system involved


INCIDENCE OF POISONING IN DIFFERENT PAEDIATRIC AGE GROUPS
46.70%
32.30%
9.20%
2.90%
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
<1 year
1-2 years
3-6 years
7-12 years

PURPOSE FOR WHICH THE POISONING SUBSTANCE WAS KEPT IN THE HOUSE




MODE OF THE EXPOSURE
18.40%
81.60%
Ingestion
Stings / bites


CLINICAL FEATURES OF KEROSENE OIL POISONING
2.40%
1.20%
3.70%
37.80%
54.90%
Resp. distress
Cough
Abd. Pain
Drowsiness
Asymptomatic