A STUDY ON RECENT TRENDS IN ADOLESCENT SUICIDE: A HOSPITAL BASED STUDY AT BOKARO GENERAL HOSPITAL

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INTRODUCTION

Poisoning is when cells are injured or destroyed by the inhalation, ingestion, injection or absorption of a toxic substance. Key factors that predict the severity and outcome of poisoning are the nature, dose, formulation and route of exposure of the poison; co-exposure to other poisons; state of nutrition of the child or (fasting status); age and pre-existing health conditions[1].

Poisoning as defined by Paracelsus as "All things are poison and nothing is without poison, only the dose permits something not to be poisonous". Today Poison is by definition, a substance that causes injury, illness or death to organisms by chemical reaction or molecular activity. Toxins and poisons are dealt under a medical branch called toxicology. This medical branch deals with pharmacokinetics and pharmacodynamics of various poisons, toxins and drugs when taken in excess amount, and clinical features associated with these substances, and management options to counteract them timely. These compounds enter into the body in various ways and leave behind a series of local and systemic effects. Poisoning refers to the enduring of harm or death due to a toxin and is a qualitative term used to define the chemical compounds potential to inflict harm in the body.

Pattern of poisoning varies from countries to countries depending on availability of the poisons, drugs, socioeconomic status of the population, religious and cultural influences in that region[6].

Poisoning is now the number 1 cause of injury death in the United States surpassing that from motor vehicle collisions. The majority of deaths are unintentional. In adolescents poisoning is the third leading cause of injury related death[2].

Poisoning in India is a major cause of worry. Hospital based data shows that poisonings constitute 0.33-7.6% of total hospital admissions in children[3,4].

Poisoning can be intentional or unintentional. Unintentional or accidental exposure makes up 80-90% in infants to preschool children, where as older children or adolescents have much higher rates of intentional or suicidal poisonings[3].

Poisoning can be classified into three types mainly; 1) Accidental, 2) Suicidal, 3) Homicidal[5].Accidental poisons usually involve a young child who accidentally poisoning themselves without wanting to cause harm to their body[5].

Children under 5 years are most at risk due to their exploratory behavior, finger-mouth activity, pica, temperament that leans towards hyperactivity. They ingest the agents which of ease of access, attractiveness, taste, color, and etc[3,6].

In pediatric emergency accidental poisoning is one of the major causes for morbidity and mortality[7]. In developed countries household products were more common. In developing countries like India insecticides, drugs and kerosene oil more common. The decreasing trends of accidental poisoning related to chemicals and drugs in developed countries due to introduction of child proof packs and bottles[8].

In contrast to accidental poisoning which is more common in1-6yrs, suicidal poisoning is more common in adolescents and aged 12-18yrs[8].stress in the family, stress in the school and psychiatric disorder with depressive disorder being common in young age[9]. Off different modes of suicide, poisoning is more common. The choice of poisoning depends on availability, cost, regional consideration, and working environment. Suicidal poisoning is major contributor to morbidity and mortality throughout the world. Especially in developing countries like India[10].

Poisoning is result of either acute or chronic exposure. Most are acute and presented as emergency typified by child who invades storage area for household cleaners or medicine cabinet chronic poisoning is due to toxicity produced over a period of time where substance accumulates in the body, producing toxic results as in lead and other heavy metal poisoning[11].

Pattern of poisoning has shown a great change over last ten years with the introduction of new products for domestic use, new chemicals in farming and industry easy of availability of many drugs and their abuse. Great Changes have taken place in the pattern of living and social customs. There is also break down of joint families and both parents working outside left children alone or in the care of an older sibling making them most susceptible to get poisoned accidentally. Once it has been thought that deliberate self harm as a problem of industrialized world. But recent work has begun to emphasize its importance in the developing world. Pesticide poisoning from occupational, accidental and intentional exposure is a major problem in developing world[12]. Poisonings are common among young children as they explore their environment and put new objects into their mouth. In some series, children account for as many as 80% of poisoning cases presenting to hospital[12].

Along with ingested poisons, poisoning due to animal bites especially snake bites is a one of common emergency and it is an occupational hazard in tropical countries like India. These are countries where farming is a major source of employment. And also in rural areas in developing

countries children are often employed on the farm thus incurring an increased risk of making contact with snakes. Children also having small body weight so snake bite in is relatively great impact on them[11,15].

It is important cause of death in rural patients in developing countries and also it is a neglected public health problem. Younger age at presentation, anemia and distance walked after the bite may be independent predictors of morbidity and mortality in children with snake bites[13]. Recently World Health Organization included it in the list of neglected tropical diseases, as many cases are unreported due to traditional treatment in rural areas not reaching hospitals. In 21st century it could be the most neglected of all tropical diseases[14, 15].

According to estimates 4,21,000 envenomings and 20000 deaths occur worldwide from snake bites in each year, figures may be as high as 18,41,000 envenomings and 94000 deaths, especially in areas of sub-Saharan Africa and South Asia where anti venoms are hard to obtain. India has the highest estimated annual envenoming and deaths 81,000, and11000 respectively. Making it is the most heavily affected country in the world[15]. Children were more severely affected because of their smaller volume relative to venom dose[16].Snake bite is an important occupational and rural hazard because India has always been a land of Exotic snakes[17].

According to Govt. of India recent data in 2013 a total of 134980 cases of snake bites and 1180 deaths and in 2014 a total of 137658 cases and 1122 deaths due to snake bites occurred. And these numbers actually high as there is underreport of data[18].

As compared with adult poisoning childhood poisoning are mostly accidental and can be preventable with simple measures. There are many epidemiological studies on childhood poisoning due to different agents. All indicating that especially in developing countries, like India improvement in implementation of prevention programs can significantly reduce the problem.

Poisoning is very common in Indian scenario. It is also common in children causing much of a distress to the parents and caregivers. Thus it is important to quantify the burden of the problem. It is also necessary to determine the most common reasons and analyze the most vulnerable group for the same. It is thus important to identify this group of children and find out the suitable options of its prevention if any.

Despite clear cut magnitude of problem, and lack of precise data regarding childhood poisoning in India, this study was done to undertake this study with the aim of quantifying the problem.

The present study is to find out childhood poisoning due to different poisons and toxic substances and find out prevalence of accidental poisoning in children.

AIMS AND OBJECTIVES

The present study is a prospective observational study on pediatric poisoning cases admitted in emergency department of Bokaro General Hospital, Bokaro with the following aim and objectives.

1. To know the magnitude of poisoning in pediatric age group <18 yrs
2. To know the manner of poisoning in pediatric age group
3. To know the common age group involved
4. To know the commonest type of poison encountered
5. To know the common sex group involved
6. To know seasonal variation if any
7. To know the factors associated with poisoning

REVIEW OF LITERATURE

The toxins are in the air we breathe and in the water we drink; in the walls of our homes and the furniture has it within them. It afflicts rich and poor, young and old[19].

The theme of suicide appears several times in ancient Greek literature. Most of the information found in mythology, but the suicide in a mythological tale, although in terms of motivation and mental situation of heroes may be in imitation of similar incidents of real life, in fact is linked with the principles of the ancient Greek religion. In ancient Greek philosophy there were the deniers of suicide, who were more concerned about the impact of suicide on society and also these who accepted it, recognizing the right of the individual to put an end to his life, in order to avoid personal misfortunes. Doctors did not approve in principal the suicide and dealt with it as insane behavior in the development of the mental diseases, of melancholia and mania. They considered that the discrepancy of humors in the organ of logic in the human body will cause malfunction, which will lead to the absurdity and consequently to suicide, either due to excessive concentration of black bile in melancholia or due to yellow bile in mania. They believed that greater risk to commit suicide had women, young people and the elderly. As therapy they used the drugs of their time with the intention to induce calm and repression in the ill person, therefore they mainly used mandragora. These may be due to avoid torture and the disgrace of rape, strong grief, the erotic disappointment, for financial reasons, in elderly physical illness and cachexia. While stabbing a sword in the body for self killing was widespread in men and soldiers[20].

History of poisoning dates back to before 4500 BC. Sumerians were probably the first to document poisons effect. The ancients initially used plant substances like aconite and curare to poison their hunting arrows and darts. Poisons have been used for many purposes across the span of human existence, most commonly as weapons, anti-venoms, and medicines. Poison has allowed much progress in branches, toxicology, and technology, among other sciences[21,22].

All over human history, intentional application of poison had used as a method of assassination, murder, suicide, and execution. Poisons lethal effect can be combined with its apparently magical powers; an example is the Chinesegu poison. Poisons also employed in gunpowder warfare.

For example, in the 14th century Chinese text of the huolongjing written by JiaoYu out lined the use of a poisonous gunpowder mixture to fill cast iron grenade bombs[21,22].

In the 8th Century AD, poisoning took another step forward when an Arab chemist successfully transformed arsenic into an odorless, tasteless powder that would elude detection for at least ten centuries, thus providing the sinister world of poisoners with the convenient and deadly inheritance powder.

The Victorian era is generally regarded as the heyday of poisoners. Indeed, it is the period from which many of the world's most notorious poisoners hail. While people who poisoned for personal gain are to be found through the ages, now that poison was readily available to commoners, potential poisoners now had a new incentive; life insurance. In fact, poison was so popular as a homicide weapon, and so readily available in various forms, that laws such as the Arsenic Act of 1851 had to be introduced to bring the crime under control. Interestingly, one of the most well known contemporary deaths by arsenic may not have been homicide after all. When Napoleon became ill in the autumn of 1820, he was convinced that he was the target of poisoning. For years it was believed that he had been poisoned by French and British conspirators. It was not until recently that it was shown that the wallpaper in his house contained arsenic, and that the metabolism of mould on the wall paper was likely to have caused release of the poison in gaseous form. It should also be pointed out that many medicines of the period contained arsenic, which could have also contributed to Napoleon's demise[23].

In ancient India, the poisons arsenic, aconite and opium we knew. They were used by women to get rid of oppressive husbands. The Mahabharat, which is usually ascribed to the fifth or sixth century B.C. mentions that Bhimsen, the Hindu Samson was poisoned by his cousin Duryodhan whom he had defeated in a duel. In a semi-historical legend of mid-India, it is narrated that the grandfather of Asoka, Chandra Gupta, a contemporary of Alexander the Great, sent to latter monarch in the guise of a present, a fascinating girl who was a 'poison maiden' fed on poison until she was so saturated with venom that her embrace would prove fatal to an ordinary mortal. The mere conception of the idea of such a Borgia-like siren would imply considerable familiarity with poisoning. In Mohamedan times, poisoning was recognized form of capital punishment and was unusually rife in harem intrigues and against political foes and prisoners.

In Indian context history shows that poisons were not only known but were also used to destroy enemies and get rid of prisoners.

Indian surgeon Sushruta is said to have defined the various stages of slow poisoning. He also mentions antidotes and the use of traditional substances to counter the effects of poisoning. Poisoned weapons were used in ancient India and also in war tactics poisons were used. A

verse in Sanskrit reads "Jalamvisrava yetsarmavamavisravyamcadusayet", which means "waters of wells were to be mixed with poison and thus polluted." Chanakya (350-283BC) was adviser of first Maurya Emperor Chandragupta (c.340-293BC). He suggested employing means such as seduction, secret use of weapons, and poison for political gain. He also urged detailed precautions against assassination-tasters for food and elaborate ways to detect poison[24].

By the 20th century, literature concerning the manufacture of poisons had left its cobwebby corners in the apothecary for the bookshelves of those who sought the knowledge and it was not just about getting rid of unwanted family any more by using poisons as weapons. On the other hand, the growth of the field of toxicology also brought about the controlled use and circulation of poisonous substances.

In the mean time, new and obscure poisons were beginning to emerge in the market, many of them legal medical drugs such as fentanyl, insulin, and various muscle relaxants including succinylcholine and Pavulon. New methods of poisoning were introduced, as well the resourceful use of common household chemicals such as antifreeze. Antifreeze was introduced as a murder weapon in the 1980s by Shirley Allen, who used it to kill her husband for insurance money.

Poisoning due to suicidal and homicidal are more common in India than in western countries as India is agriculture based country to feed for growing population by producing food grain. This leads to over usage of pesticides to increase crop yield. Apart from this easy availability, low cost and unrestricted sale of pesticides are the one of the key reason for suicidal poisoning, and being both common [25,26].

In one study fall from height, burns, bite (snakes/ scorpions/insects/ dogs), and road traffic accidents were the four leading causes of injury among children below 14 yrs[27].

The pattern of poisoning changing fast in the last three decades from common poisons like opium, arsenic, dattura, oleander etc. in the past to present pattern like insecticides, barbiturates, diazepam etc.

Since last three decades many studies done on poisoning to find the commonest type, most common age and sex involved, the manner of poisoning, the socioeconomic relation with poisoning, risk factors, potential preventive measures if any.

A study done on childhood poisoning in New Delhi at AIIMS, by retrospectively analyzing data from National Poisons information centre over three year period in children aged less than

18 years showing accidental poisoning was the commonest (79.9%). Most vulnerable age group was one to 6yr old, followed by intentional attempts (20.2%) in children 12 to 18 yr old. Oral route was most common (96.8%) followed by dermal exposure (3.2%) comprising bites and stings. Males (63.11%) more involved than females (36.88%).The various agents include household products (47%), drugs(21.8%), industrial chemicals (7.9%), agricultural pesticides (9.1%), bites and stings(3.2%), plants(1.5%), miscellaneous products (5.3%) and unknown products(4%). Bites and stings mainly caused by snakes and scorpion stings. The actual incidence might be high due to underreporting to the centre, but they do give trend in India. Which indicating strong emphasis on a prevention programs which can at least reduce the occurrence of accidental poisoning[28].

According to world report on child injury prevention by World Health Organization, the global death rate from poisoning for children younger than 20yrs is 1.8 per 100000 populations. For low and middle income countries it is four times higher than high income countries. In India the reported figures for fatal poisoning ranged between 0.65 and 11.6%. Fatal poisoning is highest among infants and decreasing with age until 14yrs. After that there is an increase again in children 15yrs of age and older. In developed and some developing countries common agents involved are medicines sold over the counter, Such as paracetamol, cough and cold remedies, iron tablets, anti histamines and anti inflammatory drugs; Prescription medicines like anti depressants narcotics and analgesics, recreational drugs such as cannabis and cocaine; household products such as bleach disinfectants, detergents, cleaning agents, cosmetics and vinegar; pesticides including insecticides, rodenticides and herbicides; poisonous plants like dattura; animal or insect bites. In low to middle income countries hydrocarbons used for fuel and lighting, such as paraffin oil (kerosene oil). Boys more involved than girls although some exceptions western pacific region[11].

Another study done on Indian poisoning incidents, by prospectively assessing patterns, severity and clinical outcome by Churi S et al, found total poisoning incidents were 212. Most of these due to pesticides (n=111), household products(n=22), medicines(n=67), bites(10). Among pesticides organophosphate pesticides were more common. And most of these occur from rural area (n=129). The major cause of poisoning was suicidal (n=178) followed by accidental (n=34). Males (n=132) outnumbered than females (n=80), Major route of exposure was found to be oral, followed by external, and inhalational exposure.

Mean age 26.65±12.65 yrs, male to female ratio 1.65:1, higher in rural than urban, higher among literates, most of them belongs to middle and low socio-economic status[29].

A study done by prospectively among children with poisoning presenting to the Pediatric emergency department of a tertiary care hospital in Delhi showing accidental poisoning formed 1.05% of all pediatric admissions. There were 80.7% children between 1 to 5 yrs of age with male predominance. Mortality was 7.7% and 11.3% children required pediatric intensive care unit (PICU). Kerosene oil poisoning remained the commonest accidental poisoning, Followed by HCL poisoning as the second commonest poisoning (17.3%) in children. This was followed by poisoning with various drugs used by adults in the house[30].

A study done epidemiology of acute poisoning in children at Ain shams university in Cairo, Egypt over 5yr period showing that 52% were younger than 6yrs of age, 11% were 6 to 12yrs, 37% were >12yrs. Unintentional poisoning accounted for 68.5%. in adolescents 84.1% ingestions were with self harm intent. In all age groups most frequent causative agents were due to drugs, among which non-opioid analgesics, antipyretics, and anti rheumatics were common. Most common non pharmaceutical agents were corrosives in preschool children and pesticides in adolescents. Among pesticides organophosphorous compounds and carbamates were common. The case fatality rate was 0.3% primarily from pesticide ingestion[31].

A study was done in Pediatric Emergency Departments of Tikur Anbessa, Yekatit 12, and Zewditu Memorial Hospital. Children under 14 years were included In this study. The mean age of the victims was 65.5 months. There was Slight Females (51.6%) predominance than males. Among this 74.2% of total children poisoning occurred in home. Unintentional poisoning occurred in 77.5% of the children. Whereas intentional poisoning was seen in 15.5%, the common causes of intentional poisoning were as in decreasing order drugs, organophosphate and sodium hypochlorite. Children aged more than 5 years were more likely to have intentional poisoning. And also it has been found that young age was related to an earlier presentation as compared to children older than 2 years. It also founded that no post exposure counseling given for children with poisoning, nordid the children have psychosocial evaluation by child psychiatrist and follow up after the incident[32].

A study done at tertiary care centre in North India on childhood poisoning among children <12 yrs, found that mean age 3.12± 2.04. The majority of patients was in the age group of 1-3 yrs. Males outnumbered females by a factor of two.

The majority of patients resided in urban areas. Kerosene(27.9%), drugs(19.8%) and insecticides (11.7%) were most frequently implicated. Almost all (96.9%) ingestions were accidental poisonings. After 6 hrs of observation 32.4% were asymptomatic. Symptoms related to toxic ingestion noted in 67.6%. common serious symptoms include altered sensorium,

respiratory distress, seizures, ataxia, hypotension, cyanosis and burns. In developed countries majority of poisonings are due to common non toxic household products. In developing countries most patients require hospitalization because of severe symptoms related to the dangerous nature of toxins ingested. And also found that consultation with poison cell results in improved patient management[33].

A study done on profile of poisoning in children at Indira Gandhi Medical College in Shimla found that a total of 165 children between age 0-18 years were admitted with poisoning in study period. The incidence of poisoning was 1.28%. Commonest age group was between 11-18 yrs. male to female ratio was 1.17:1. Most of them belongs to middle (20%) and low(75.7%) socio-economic status, thirty one patients(18.8%) from urban population, and 134(81.2%) were from rural areas. The average duration of stay in hospital was 2.7 days. Accidental mode of poisoning (92.8%) is more common than suicidal (7.8%). Age less than 5 years especially 18 months to 3 year old children accidental poisoning was common, whereas in older children and adolescents intentional poisoning was common, stress has been implicated for increased incidence of intentional poisoning in adolescents. 38.2% of cases were due to poisoning by insecticides and pesticides, followed by plant poisoning, kerosene oil poisoning is seen in 4.9% of patients. And 60% of patients presented to hospital after 5 hours of exposure[34].

A prospective study on Trends of acute poisoning cases in a tertiary care hospital in Odisha, India, found that majority (43%) were less than 5 years. Males (70%) of cases, Median duration of presentation being 17 hours. 65.7% received pre hospital treatment. Snake bites (27.7%) followed by kerosene (17.3%) and insecticide (12.4%) poisoning being the most common offending agents. Accidental exposure being 85.8%, suicidal cases more common in >11 yrs (68%). Most common presentation being respiratory distress(32.5%), cellulitis of limb (24.5%), and altered sensorium (18.3%). mortality being 7% overall. Outcome is 92% survival. Even though most common age group affected is still <5yrs, there has been an increasing trend in older children, and adolescents with suicidal poisoning[35].

A study done in Lucknow on pediatric poisoning trend showing among 100 poisoning cases in children less than 15years 62% were under 5 years of age, males were 70%.

Incidence more common in urban areas (55%) as compared to rural (45%). 36% of cases reported in rainy season. Kerosene and snake bites were most common offending agents. Maximum number of kerosene oil poisoning was reported in summer, while snake bites and organophosphorus poisoning reported commonly in rainy season. Mortality was found to be 4%[36].

A study done on characteristics of children with acute carbon monoxide poisoning in Ankara, among children age group of one month and 16 yrs. Found that Education level were low in 86.2% of mothers and 52.6% fathers. All families had low income and 48.8% did not having formal housing. The source of the acute carbon monoxide poisoning was stoves in 71.2% of cases and hot-water heaters in 28.8% of cases. Three or more people were poisoned at home in 85.1% of the cases. The most frequent symptoms of poisoning were headache and vertigo (58.8%). Median carboxyhemoglobin levels at admission to the hospital and discharge were measured as 19.5% and 1.1%. atre evaluation it was found that most of them had taken the necessary precautions after the poisoning incidence(86.3%)[37].

Another study on poisoning in children aged 18 years and below from an educationally and economically advanced urban area of south India found that deliberate self harm (DSH)(70.2%) most common fallowed by accidental (29.8%).121 poisoning cases constituting 0.5% of total pediatric admissions. Median age of children with accidental poisoning was 2 yrs, while in DSH it was 16yrs. Most common agents in accidental poisoning include household items like drugs, insecticides, cleaning solutions. One third of children with DSH had an underlying psychiatric disorder. A majority (75%) of children were brought to hospital within six hours. The poisoning severity score was none or mild in 91(75.2%), while 17 had moderate and 10 had severe poisoning. The overall mortality was 2.5%[38].

Another study from tertiary care hospital in South India found that poisoning contributed for 5.3% of total admissions. Household products topped the list with 112 cases among 332 of total poisoning cases during study period, followed by agricultural products (n=88), animal bites and stings (n=69), drugs(n=48). Among household products kerosene was most common (n=95). Males were affected in 42 of 59 bites (71%), 26 of 48 organophosphorus poisonings (54%), 12 of 48 drug poisoning (25%), 45 out of 95 (47.3%) in kerosene poisoning, 10 out of 33(33%) in rat poisoning. 76 of 95(80%) Kerosene poisoning occurred maximally in 1- 4 year age group. 90% of organophosphorus poisoning was observed in more than 10 years of age. 64% of all drug poisoning was seen after 10 years of age[39].

A study done at Kasturba Hospital, Manipal on trends of poisoning among children, also revealing as most common substance in the pediatric poisoning cases was insecticides followed by venomous bites, drug over dosage, kerosene consumption, corrosives etc. most common route was ingestion (84%), followed by injection which included snake bites and bee stings (16%). 95.3% were survived, and 4.7% cases were expired[40].

A retrospective study over ten years done on childhood poisoning in Warri, Niger Delta, Nigeria, during study period 156 children aged 0-16 yrs were diagnosed as poisoning. The male to female ratio is 2:1, and 75% of children aged less than 5yrs. Most of the patients were from low socio-economic class. Most of the poisonings were unintentional and occurred through ingestion (97.6%). Kerosene (56.6%) was the major substance leading to poisoning. Alcohol ranked second in the study. Poisoning from drugs was the third most common, in this category most of the cases from highest income group. Most of the patients presented with mild symptoms and the mortality rate was 7%[41].

A study conducted by D.Manikyamba, N. Madhavi on clinical profile in children admitted in a tertiary care centre in Kakinada and Guntur in India. The study done over 2years period during which out of 6398 pediatric admissions 183(2.86%) were due to poisoning. Commonest poisoning consumed was kerosene (50%) followed by acids and alkalis (13%) and pesticides(6.02%). Accidental poisoning was most common and mostly in the age group of 1-3 years (64%). Male to female ratio was 1.56:1, more cases occurred among rural areas and during summer season. Poisoning due to bites and stings was 44.26% of total cases with 58% due to snake bites and 24.7% due to scorpion stings. Most of the bites and stings were in males from rural areas in the age group of 10-12%. 2 cases were due to inhalational poisoning. Out of 100 cases of ingested poisons 54 were due hydrocarbon compounds, 13 cases due to corrosives, 12 cases were admitted with pesticides and insecticides, 9 cases were due to ingestion of drugs, 9 cases due ingestion of plant products. 21% of ingested poisoning belonged to lower middle, 22% cases were from upper lower and 57% of cases from lower socio economic class. 94 cases accidental and 6 cases were suicidal. 96 cases survived and 4 cases were expired. Out of 50 kerosene oil poisoning 43 were asymptomatic and 7 were symptomatic, which include tachypnea, fever, irritability, and vomitings. Out of 81 cases of various bites and stings, 47 were due to snake bites, 20 were due to scorpion stings 1 case due to centipede bite, 13 unknown bites. Out of these 78 were survived, 3 were expired. Among snake bites 68.1% were non poisonous snake bites and 31.9% due poisonous snake bites. Out of 15 poisonous snake bites, 7 were viper bites, 3 were cobra bites and 5 were krait bites. Local edema (7) and cellulitis (7) were the commonest clinical features seen in viper bites. Ptosis (5), headlag (1), diplopia (4) and respiratory muscle weakness(3) were the common features in krait and cobra bites. Average time interval between appearance of clinical features and scorpion sting was 1 to 6 hours. Common clinical features of scorpion sting were local pain (80%), cool peripheries (80%) and tachycardia (70%). Other features included sweating (60%), vomiting(50%),

hypertension (40%) and paraesthesias (30%). Priapism was seen in 4 children, 1 child expired due to cardiogenic shock[42].

A study done by Ram P, Kanchan T, Unnikrishnan B on pattern of acute poisonings in children below 15yrs in Mangalore, South India. During study period 81 children below 15yrs with poisoning admitted. 50.6% were boys and 49.4% girls. The mean age of study group was 6.8yrs. Mean age was observed to be higher in females than males. The maximum number of cases were observed in the below 5 years age group (n = 45). A male predominance was evident in the below 5 years age group, while a female predominance in the age group between 10 and 15 years. Kerosene (n = 23, 28.4%) and organophosphate compounds (n = 16, 19.8%) were the most common agents responsible for poisoning in children. The majority of the poisoning cases were reported to the hospital within 12 h of the incident (n = 65, 83.3%). The mortality in paediatric poisoning was observed to be 7.4%. The study reports agrochemicals and hydrocarbons to be the most commonly implicated agents in paediatric poisoning. The majority of the children (n = 68, 84.0%) recovered, while seven patients had left the hospital against medical advice (8.6%)[43].

A study done by Debata PK, Deswal S, Kumath M on causes of unnatural deaths among children and adolescents in Northern India - a qualitative analysis of postmortem data. All unnatural deaths in children aged 1-19 years from April 2010 to March 2011 were identified from the postmortem database. Retrospective analysis of Postmortem data during one year revealed total of 434 unnatural deaths in children aged 1-19 years. The most vulnerable age group included children between 11-19 years (74.5%). Females (51.6%) marginally outnumbered the males (48.4%). Flame Burns (58.3%) was the commonest cause of death in all age followed by road traffic accidents 15%, electrocution 7.8% & Poisoning accounted for 6%. The most frequent victims were adolescents (74%) almost 3 times that of other age group. This study showed Adolescents were the most common victims of the unnatural death with flame burn being the most common cause followed by RTA, electrocution and poisoning[44].

Another study done by Retrospectively analyzing Acute Organophosphorous Poisoning cases admitted to the tertiary care teaching hospital in South India. Among 101 patients males were more commonly involved than females (M:F 2.5:1). The mean age of the patients was 28 years (range 2-72 years, SD ± 14.3 years). Mean time to receive treatment was 5.2 ± 7.4 (range 1-48 h). About 45.5% patients received first aid before coming to the hospital. The reason was suicide in 88.1% cases and accident in 12 (11.9%, all children). Seventy-nine patients received pralidoxime (PAM) and the mean duration was 1.7 ± 1.1 (range 1-4 days). Atropine was given

in all patients. Mean duration was 5.1 ± 3.1 (range 1-19 days). Mean hospital stay was 7.5 ± 4.7 days (range 1-26 days). Mortality was 9.9% in the present series[45].

A study done by Basavaraj, Pushpalatha on clinical profile and outcome of acute pediatric poisoning in urban tertiary care hospital, Bengaluru, A total of 50 patients presented with acute poisoning during the study period. Majority of children were in the age group between 12 to 18 years (30 cases, 60%). Median age of patients was 10 years. The majority of patients resided in urban areas. Drugs (40%), Kerosene oil (24%) and Insecticides (20%) were the agents most frequently implicated. 52% cases were suicidal in nature and the above children belonged to age group of 12 to 18 years. Almost all cases in 1-6 years age group were accidental in nature. One case of kerosene oil consumption died whereas outcome in other cases was good[46].

A retrospective study done by Vasavada H, Pankti D on clinical profile and outcome of children presenting with poisoning, a total 176 patients with definite history of poisoning admitted. The mean age was 4.5 years with an age range of 0.25 to 15 years. The overall male female ratio was 1.17:1. The most common poison was kerosene used as fuel, incidence of insecticide poisoning was low. The mortality was 5.1%. Most patients were from urban background (83.5%), as many as 59.65% patients presented within first 4 hours after exposure. Accidental poisoning was found in 98.59% of patients rest being suicidal. Mean duration of hospital stay was 3.3 days and vomiting was the commonest clinical manifestation[47].

EPIDEMIOLOGY OF POISONING

Magnitude of poisoning:

Poisoning as a worldwide problem.

Poisoning is a medical emergency. The number patients admitted with poisoning increasing day by day. According to the WHO Global Burden of Disease project, an estimated 345 814 people of all ages died worldwide as a result of “accidental” poisoning in 2004. The majority of these accidental poisonings were among adults, 13% occurred among children and young people under the age of 20 years. Among 15–19-year-olds, poisoning ranks as the 13th leading cause of death. A survey of 16 middle-income and high-income countries revealed that, of the different external causes of unintentional injury death among children aged between 1 and 14 years, poisonings ranked fourth in 2000–01, after road traffic crashes, fires and drowning. This

is much higher in low income countries. Acute poisoning accounted for an estimated 45 000 deaths annually in children and young people under the age of 20 years. The global death rate from poisonings for children younger than 20 years is 1.8 per 100 000 population. For high-income countries the rate is 0.5 per 100 000 while for low-income and middle income countries it is four times higher, at 2.0 per 100 000[11].

In 2008, the number of poisoning deaths exceeded the number of motor vehicle traffic deaths and was the leading cause of injury death for the first time since at least 1980. During the past three decades, the poisoning death rate nearly tripled, while the motor vehicle traffic death rate decreased by one-half. During this period, the percentage of poisoning deaths that were caused by drugs increased from about 60% to about 90%[2,48].

A population based cohort study done by Tyrrell EG, Orton E, Tata LJ on changes in poisonings among adolescents in UK between 1992 and 2012 found that Overall poisoning incidence increased by 27% from the period 1992-1996 to 2007-2012, with the largest increases in intentional poisonings among females aged 16-17 years, and alcohol related poisonings in females aged 15-16 years[49].

Poisoning in Indian scenario:

In India Hospital based data shows that poisonings constitute 0.33-7.6% of total hospital admissions in children[3,4]. Available data indicate that among children less than 18yrs, 10-15% of deaths, 20-30% of hospital registrations and 20% of disabilities are due to injuries. These include Road Traffic Injuries (RTI's), drowning, falls, burns, and poisonings. These result in 1,00,000 deaths in children every year in India. Drowning and burns are major causes of mortality in less than 5 y, while RTIs, falls and poisoning are leading causes in 5-18 y. A shift in the occurrence of suicides to younger age groups of 15-20 y is a matter of serious concern in recent years. More number of males, those in rural areas, and majority of poor income households are affected due to injuries[50].

Suicide death rates in India are among the highest in the world. A large proportion of adult suicide deaths occur between the ages of 15 years and 29 years, especially in women[51].In one study done by Singh SP, Aggarwal AD, Oberoi SS et al. on study of poisoning in North India, found that Deaths due to poisonings gradually increasing as with increasing of age, with peak in 21-30 age group. In 10-20 age group it was found to be 18.18%[52]. Another study done by

Akbar S, Asma R et al, on unintentional injuries among children (up to 12yrs) admitted in a tertiary care hospital in North Kerala, found that Poisoning 89 (22.3%) was the most common cause of unintentional injury, followed by Road Traffic Accidents (RTA) 76 (19%) and fall 68 (17%), male children were more as compared to Female (1.8:1)[53].

The young preschool child is most susceptible to accidental poisoning. Data from various socio-cultural groups indicate that 2 to 3 year old children account for over half of the reported cases and 80 to 90% are under 5 yrs of age. Usually, this pattern of occurrence is related to the developmental stage of the child. As the infant starts crawling and walking around one year of age, his activity and curiosity increase and lead him to explore unfamiliar objects by putting them into his mouth. Later, by about 2 ½ - 3 yrs of age the child's motility and ingenuity allow him access to any unlocked drawer or cupboard at home. And also their finger-mouth activity, temperament leans towards hyperactivity. They ingest the agents which of ease of access, attractiveness, taste, color, and etc[3].

By the age of 4 years, the incidence of accidental poisoning begins to decline despite a further increase in motor development and co-ordination, as the older child tends to select things that taste good for purposes of ingestion. Although accidental poisoning is more common in children less than 5 years and decreasing further with increasing of age until age of 14yrs. After 15years again there is increasing of poisoning cases. In contrast to children less than 5years mostly of which accidental in nature, adolescents more likely have intentional poisoning[3,11,34]. In most cases Stress and psychological problems are reason for this increasing trend in adolescents[34].

A study done by Venkatesh C, Sriram et al found that Accidental poisoning with kerosene is more common in developing countries. Boys aged less than 3yrs from rural background formed significant proportion. Male gender and malnutrition were significantly associated with prolonged hospital stay[54].

However, it is very difficult to find out the exact incidence of poisoning cases in India and the number of people who die due to poisoning. This is because of not reporting to the authorities in cases of survival. However, the magnitude of the poisoning cases in India can be observed by the number of cases reported based on the annual reports of the chemical examiners laboratories from various states which is only a tip of the iceberg.

INCIDENCE OF SNAKE BITES:

Snakebite is a medical emergency in many parts of the world, particularly in the temperate regions. According to 2007 World Health Organization (WHO) report, there are about 5 million snakebite incidences resulting in 2.5 million envenoming, and 125,000 deaths occur annually[55].

According to estimates 4,21,000 envenomings and 20,000 deaths occur worldwide from snake bites in each year, figures may be as high as 18,41,000 envenomings and 94,000 deaths, especially in areas of sub-Saharan Africa and South Asia where anti venoms are hard to obtain. India has the highest estimated annual envenoming and deaths 81,000, and 11,000 respectively. Making it is the most heavily affected country in the world[15,16,17].

According to Govt. of India recent data in 2013 a total of 134980 cases of snake bites and 1180 deaths and in 2014 a total of 137658 cases and 1122 deaths due to snake bite occurred[18].

The Registrar-General of India's "Million Death Study", 2001-2003, is expected to provide reliable evidence of substantial mortality (exceeding 50000 per year) as it is based on Representative, Re-sampled, Routine Household Interview of Mortality with Medical Evaluation ("RHIME"), covering all age groups across the entire country with geographical, seasonal and occupational data[56].

In India highest snakebite deaths noted in Orissa, Madhya Pradesh, West Bengal, Gujarat, Uttar Pradesh , Karnataka[18].

A study by Kshirsagar VY et al on clinical profile of snake bite in children in rural India found that vasculotoxic (90.74%) more common than neurotoxic (9.25%), common in rainy season, and in males age more than 5 yrs. Bite marks on lower limbs in 70.04% of children, with mortality rate was 1.85%[57] .Snake-bite poisoning is a major, difficult undertaking in developing countries, because of under reporting and difficulties associated with identification of snakes. Most victims prefer to be treated by a traditional healer and do not go to hospital[58]. As there is only one-fourth of snakes are poisonous and this is the factor which boosts the success of the witch-craft in treating snake-bites in India.

The major families of poisonous snakes in India are a) Elapidae which includes common cobra (Najanaja*),* king cobra and common krait (bungaruscaerulus), b) viperidae includes Russell's viper, saw scaled or carpet viper (echiscarinatus) and pit viper and c) hydrophidae (sea snakes)[17,59].

In one study by Koirala DP et al. boys were more commonly involved, mostly from rural areas. The commonest site of bite was foot, local swelling and pain were common clinical features. Coagulation profile deranged in majority (80%) of patients, and hematuria (13%) and thrombocytopenia (15%) also noted. Only 44% of the patients received polyvalent anti-snake venom and blood products were required in 51% of the patients. Most patients improved (75%) and the case fatality rate was 2.5%[60].

A study by Punde DP, on management of snake bite in rural Maharashtra, The majority of snake-bites occurred between May and November. Those affected were mainly farmers, students, and housewives. Among those envenomed by poisonous snakes, the mortality was 4.7%[61].

A study by Vaiyapuri S et al on snakebite and its socio-economic impact on the rural population of Tamilnadu, India showing an increasing snakebite trend in economically active age groups between 11 and 50 yrs. A large proportion of bites were categorized as of unknown origin because of some people were not aware of the characteristics of particular snakes, and also not possible to identify the snakes at night time[69].

CLASSIFICATION[62]

Poisons may be classified according to the chief symptoms which they produce as follows

I) Corrosives:

i) Strong acids:

a. Mineral acids or inorganic acids– Sulphuric, nitric, hydrochloric acid.

b. Organic acids – Carbolic, oxalic, acetic, salicylic acids.

ii) Strong alkalies – Hydrates and carbonates of sodium, potassium and ammonia.

iii) Metallic salts – Zinc chloride, ferric chloride, copper sulphate, silver nitrate, potassium cyanide, chromates and bichromates.

II) Irritants:

i) Inorganic:

a. Non-metallic - phosphorus, chlorine, bromine, iodine, carbon tetrachloride.

b. Metallic - arsenic, antimony, mercury, copper, lead, zinc, silver.

c. Mechanical – powdered glass, diamond dust, hair etc.

ii) Organic:

a. Vegetable - Abrusprecatorius, castor, croton, calotropis, aloes etc.

b. Animal - Snake and insect venom, cantharides, potamaine.

iii) Agricultural.

III) **Systemic:**

1. Cerebral:

- CNS depressants: alcohols, general anaesthetics, opioid analgesics, hypnotics, sedatives.
- CNS stimulants: cyclic antidepressants, amphetamine, caffeine, methylphenidate
- Deliriant: datura, belladonna, hyocyamus, cannabis, cocaine, etc.

2. Spinal - Nux vomica, gelsemium.
3. Peripheral - Conium, curare.
4. Cardiovascular - Aconite, quinine, oleander, tobacco, cyanide.
5. Asphyxiants - Carbon monoxide, carbon dioxide, hydrogen sulphide.

IV) **Miscellaneous**: Food poisoning, botulism.

The modes of exposure to poisoning are

1. Ingestion
2. Inhalation
3. Topical
4. Ocular
5. Environmental
6. Trans placental

Classification of Poison according to motive or nature of use:

1. Homicidal: Arsenic, Aconite, Digitalis, Abrus Precatorius, Strychnosnux vomica.

2. Suicidal: Opium, Barbiturate, Organophosphorus, carbolic acid, copper sulphate.

3. Accidental[79]: Aspirin, organophosphorus, copper sulphate, snakes bite, Ergot, CO, CO_2, H2S.

4. Abortifacient: Ergot, Quinine, Calotropis, Plumbago.

5. Stupefying agent: Dhatura, cannabis, chloral hybrate.

6. Agents used to cause bodily injury: Corrosive acids and alkalies.

7. Cattle Poison: Abrusprecatorius, Calotropis, plumbago.

8. Used for malingering: semicarpusanacardium.

TYPES OF POISONING[63]:

1) Fulminant:- Produced by a massive dose of poison. Death occurs very rapidly, sometimes without preceding symptoms, i.e., the patient appears to collapse suddenly.

2) Acute:- Produced by a single large dose or several small doses taken in a short period. Onset of signs and symptoms is usually abrupt.

3) Chronic:- Produced by small doses taken over a long period. Onset is usually insidious.

4) Subacute:- Some poisons also show a subacute type of poisoning which lies somewhere between the latter two extremes mentioned above.

General approach to acute poisoning[64,74,88].

- Resuscitation
    - Airway
    - Breathing
    - Circulation
    - Seizure control
    - Correct hypoglycemia
    - Correct hyperthermia

    - Resuscitation antidotes
- Risk assessment
- Supportive care and monitoring79
- Investigations
    - Screening (ECG, paracetamol)
    - Specific
- Decontamination
- Enhanced elimination
- Antidotes
- Disposition

Resuscitation[64,73].

Assessment and management of immediate threats to the airway, breathing, and circulation in the acutely poisoned patient usually follow conventional lines[65].

Examples of interventions specific to toxicology include sodium bicarbonate and hyperventilation to prevent or terminate ventricular tachycardia secondary to cyclic antidepressants[66], and benzodiazepines to treat tachycardia secondary to sympathomimetic agents. Seizures, hypoglycemia and hyperthermia must be detected and treated promptly to ensure good neurological outcome. Toxic seizures are usually controlled with intravenous benzodiazepines. Barbiturates are second line treatment. Pyridoxine is an additional option for seizures associated with poisoning from Isoniazid. Phenytoin is contraindicated in the treatment of toxic seizures.

The age of the patient can help guide appropriate toxin triage. Infants and non ambulatory toddlers are seldom able to access objects beyond their reach, such as cosmetics and soaps. Therefore, significant toxicity in these children should prompt consideration of parental or caregiver abuse. Most toxin ingestions occur among toddlers and children younger than six years with access to unsecured substances.

In one study by Greenwald PW et al. found that multiple substance exposures have become a greater percentage of cases reported to the AAPCC and have higher fatality rates than single substance exposures[71].

Risk assessment[64]:

Following resuscitation, risk assessment is the next essential step in the management of the poisoned patient.

- Distinct cognitive step
- Quantitative
- Takes into account

a. Agents

b. Doses

c. Time since ingestion

d. Current clinical status

e. Patient factors

Risk assessment is vital as it allows the clinician to make specific decisions about all subsequent management steps that are appropriate to the individual patient at that particular time.

Provided they have a normal mental status, patients with deliberate self-poisoning are generally both willing and able to give a good history from which an accurate risk assessment can be constructed.

Where an alteration in mental status precludes obtaining a direct history, back-up strategies are used to obtain the necessary information. These include asking ambulance officers or family to search for agents, counting missing tablets, checking medical records for previous prescriptions, and questioning relatives about agents potentially available to the patient.

In unknown ingestions, the patient's clinical status should be correlated with knowledge of the agents commonly prescribed in that geographical area.

The agent dose and time since ingestion should correlate with the patients current clinical status. If they do not the risk assessment is revised. For example following cyclic antidepressant deliberate self poisoning life threatening events occur within six hours of ingestion. Patients at low risk can be identified on clinical grounds at six hours after ingestion. In contrast, following deliberate self-poisoning with sustained-release calcium channel blockers, patients may not exhibit clinical features of poisoning during the first few hours but the risk assessment anticipates delayed severe cardiovascular effects[70].

The emergency provider must be aware of the pathophysiologic vulnerabilities of infants and children and substances that are especially toxic[68].

Factors determining the Severity of poisoning and its outcome are interrelated. These include:

a. Type of poison
b. The dose
c. The formulation
d. Age of the child
e. The route of exposure
f. The state of nutrition of the child
g. The presence of other diseases or injuries
h. The presence of other poisons[11].

Investigations[64,91]

In the acutely poisoned patient, screening tests aim to identify occult toxic ingestions for which early specific treatment is indicated. The recommended screening tests for acute poisoning are the 12 lead electrocardiogram (ECG) and the serum paracetamol level.

The ECG is a readily available non-invasive tool that assists in the identification of occult but potentially lethal cardiac conduction abnormalities, such as in cyclic antidepressant cardiotoxicity. Life threatening paracetamol poisoning is occult in the early stages but

progression to fulminant hepatic failure and death can be prevented by timely administration of N-acetylcysteine.

Screening is particularly important where altered mental status precludes obtaining an ingestion history directly from the patient.

Without clinical suspicion or suspected access to illicit drugs, toxicologic screens are not usually useful in guiding treatment. In one study, only 3 percent of screening test results in the pediatric emergency department were positive without suspicion of an exposure.

Gastrointestinal decontamination[67,70,76,77,78,79,83,85,88,99]:

No benefit has been demonstrated when applied routinely to groups of heterogeneous deliberate self-poisoned patient. However, the efficacy of gastrointestinal decontamination in selected rare poisonings has not been evaluated and so the decision remains one of clinical judgment involving a risk benefit analysis. The performance of the procedure should not entail a risk of pulmonary aspiration.

Table - 1: Agents Used for Gastrointestinal Decontamination in Children

AGENT	DOSE	RISKS	CONTRAINDICATIONS
Activated charcoal[76]	1 to 2 g per kg (maximum of 50 to 60 g)	Aspiration, constipation, vomiting	Unlikely to benefit patients who ingested alcohols, strong acids or bases, minerals, iron, lithium, or hydrocarbon
Gastric lavage[76]	10 to 15 mL per kg saline instilled via large-bore orogastric tube, repeated until aspirates clear	Esophageal/laryngeal trauma, aspiration, nausea/vomiting, impaired level of consciousness	Unprotected airway, ingestion of hydrocarbons or corrosives, risk of perforation or hemorrhage

Polyethylene glycol[85] (used with whole bowel irrigation)	500 mL per hour for children nine months to five years of age1,000 mL per hour for children six to 12 years of age	Vomiting, cramping	Unprotected airway, intractable vomiting, gastrointestinal hemorrhage, ileus, perforation, obstruction
Sorbitol (used with activated charcoal)	1 to 2 g per kg	Hypernatremia, dehydration	Obstruction, perforation, ileus

In contrast with meticulous resuscitation and supportive care, gastrointestinal decontamination seldom saves a life. Gastrointestinal decontamination is reserved for severe or life threatening poisoning where supportive care or antidotal treatment alone may not be adequate to ensure a satisfactory outcome.

Enhanced elimination[88,99].

Considerations in treatment include the need for attentive supportive care, pediatric implications for antidotal therapy, and extracorporeal removal[70] methods such as hemodialysis in children[68,83]. Interventions aimed at enhancing elimination of an agent from the body include multiple-dose activated charcoal, haemodialysis, charcoal haemoperfusion, and manipulation of urinary pH[88]. These interventions are employed if it is thought they will reduce mortality, reduce length of stay or complication rate, or reduce the need for other more invasive interventions. In practice, they are rarely indicated because the risk assessment for most acute poisoning is relatively benign or anticipates a good outcome with a short period of supportive care.

For those agents likely to cause more severe poisoning, relatively few drugs possess the pharmacokinetic properties (small volume of distribution, small molecule size, slow endogenous elimination) that render them amenable to enhanced elimination. Risk assessment allows early identification of the need for enhanced elimination before established poisoning develops. Early implementation of these techniques can prevent or minimize toxicity. It also

allows time for the communication, planning, and transport that may be required to access more sophisticated methods of enhanced elimination. Use of enhanced elimination techniques requires predefined clinical or laboratory endpoints for therapy.

Cathartics and whole bowel irrigation[85]:

These are useful in ridding of the lower gastrointestinal tract toxins. However they carry a risk of electrolyte imbalances and dehydration, pain and cramping. Sorbitol is often used with the first dose of activated charcoal and is occasionally given again later. Polyethylene glycol is less likely to cause electrolyte imbalances and is being used with whole bowel irrigation for some poisonings. Although whole bowel irrigation may be helpful for those who have ingested heavy metals or long-acting or sustained-release medications, there are few clinical trials about the effectiveness of this procedure in children.

Hemodialysis and urine alkalinization[88,99].

Hemodialysis may be appropriate for lithium, salicylate, theophylline, methanol, atenolol, Phenobarbital or valproic acid toxicity. Urinary alkalinization with sodium bicarbonate may be used for poisoning with salicylates, tricyclic antidepressants, Phenobarbital, chlorpropamide, chlorophenoxy herbicides, or methotrexate.

Antidotes:

Antidotes are drugs that correct the effects of poisoning. They only exist for a few specific poisonings and many are used rarely[77]. As with any other drug, the indications to administer an antidote are derived from an analysis of potential benefits and risks to that individual patient.

Rational antidote use also requires planning in terms of stocking, storage, monitoring, training, and protocol development.

Toxidromes[67,78]:

Table -2: Toxidromes and Antidotes

TYPE OF POISONING	AGENTS	TOXIDROMES/TOXIC SYMPTOMS	ANTIDOTES[90]
Acetaminophen[78, 80,88]	Acetaminophen	Abdominal pain, nausea/vomiting, elevated aspartate transaminase level (greater than 1,000 IU per L after 24 hours), jaundice, confusion, somnolence, coma, disorientation	N-acetylcysteine (Acetadote)
Anticholinergic	Antihistamines, atropine[81] (Atreza), belladonna alkaloids, toxic mushrooms, psychoactive drugs	Tachycardia, hyperthermia, mydriasis, warm and dry skin, urinary retention, ileus, delirium	—
Anticoagulant[93, 94,95]	Warfarin (Coumadin), rodenticides	Ecchymoses, bleeding, prolonged prothrombin and bleeding times	Vitamin K
Cardiac medication[70,86,87]	Calcium channel blockers[70], beta blockers, digoxin[89]	Bradycardia, arrhythmias, hypotension, dizziness, heart block, nausea, vomiting	Calcium chloride[70], glucagons, digoxin immune fab[89] (Digibind)
Cholinergic[75,76], muscarinic	Carbamates, some mushrooms, organophosphates[75,76,84], physostigmine, pilocarpine (IsoptoCarpine), pyridostigmine	Salivation, lacrimation, urination, diarrhea, bronchorrea, wheezing, bradycardia, vomiting	Atropine/pralidoxime[75,76]
Cholinergic[75,76], nicotinic	Black widow spider bites, carbamates, insecticides, nicotine	Tachycardia, hypertension, fasciculations,	Atropine/pralidoxime[75,76]

		gastrointestinal cramps, emesis, miosis	
Cyanide	Cyanide	Syncope, cyanosis, hypotension, psychosis	Sodium nitrite 3%, sodium thiosulfate 25%
Ethylene glycol, methanol[82,96,97,98]	Antifreeze, rubbing alcohol	Central nervous system depression, respiratory depression, seizures, hypotension, hypoglycemia	Ethanol 10% or fomepizole (Antizol)
Iron[83,84,85]	Iron-containing products[83,85]	Dyspepsia, nausea, vomiting, diarrhea, dark stools	Deferoxamine[83,85] (Desferal)
Opioid[92]	Opioids (e.g., morphine, hydrocodone [Hycodan], methadone)	Hypoventilation, hypotension, miosis, sedation, hypothermia, ileus	Short-acting naloxone, monitor closely for withdrawal symptoms and relapsing sedation
Salicylate[72,99]	Aspirin products	Tinnitus, nausea, vomiting, fever, disorientation, lethargy, tachypnea	—
Sulfonylurea[100,101]	Sulfonylurea	Hypoglycemia, tachycardia, diaphoresis, clammy skin, mental status changes, coma	Octreotide
Sympathomimetic	Amphetamines, caffeine, cocaine, ephedrine, 3,4-methylenedioxymethamphetamine (also called Ecstasy), phenylpropanolamine theophylline, diphenoxylate/atropine (Lomotil)	Tachycardia, hypertension, mydriasis, agitation, seizures, diaphoresis, psychosis, hyperthermia	—

Clues to diagnosis of unknown poisons:

I) Vital Sign Changes:

A) Pulse

Bradycardia Tachycardia

Narcotics Alcohol

Organophosphates[75] Amphetamines

Cyanide Tricyclic antidepressants

Carbon monoxide Salicylates

β-blockers Cocaine

B) Respiration

Bradypnea Tachypnea

Alcohol Amphetamine

Barbiturates Methanol

Narcotics Salicylates

Sedative-Hypnotics Carbon monoxide

C) Blood Pressure

Hypotension Hypertension

Antidepressants Amphetamine

Antihypertensives Sympthomimetics

Antipsychotics Tricyclic antidepressants

Cyanide Antihistamines

D) Temperature

Hypothermia Hyperpyrexia[73]

Ethanol Strychnine

Barbiturates Anticholinergics

Phenothiazines Monoamine oxidase inhibitors

Cyclic antidepressants Sympathomimetics

II) Neuromuscular:

A) Coma

Narcotic depressants

Sedative-hypnotics

Alcohols

Organophosphate insecticide[75,76]

Clonidine

B) Delirium/ Psychosis

Steroids

Heavy metals

Cocaine

Heroin

Methaqualone

C) Convulsions

Aminophylline

Organophosphates[75]

Camphor

Amphetamine

Phencyclidine

D) Ataxia

Diphenylhydantoin

Organic solvents

Alcohol

Carbon monoxide

Hydrocarbons

E) Paralysis

Botulism

Ticks

Paralytic shelfish poisoning

Heavy metals

Poison hemlock

III) Eyes

A. Pupils

Pinpoint (miosis) **Dilated** (mydriasis)

Opium Datura

Organophosphates[75] Amphetamine

Ethanol Cocaine

Barbiturates Methanol

Phenothiazines Meperidine

Phenyclidine Antihistamines

B. Nystagnus:

Sedative-hypnotics

Carbamazepine

Phencyclidine

Barbiturates

Glutethimide

IV) Skin:

A. Jaundice:

Carbon tetrachloride

Naphthalene

Acetaminophen

Heavy metals

B. Cyanosis (Unresponsive to oxygen, as a result of methemoglobinemia)

Anilinedyes

Benzocaine

Phenacetin

Nitrobenzene

C. Pinkness to redness:

Alcohol

Cyanide

Boric acid

Antihistamines

V) Odours:

A) Acetone: Acetone, phenol and salicylates

B) Alcohol: Ethanol (alcoholic beverages)

C) Bitter almond: Cyanide

D) Garlic: Heavy metals (arsenic, phosphorus and thallium), organophosphates

E) Oil of wintergreen: Methylsalicylates

F) Hydrocarbons: Hydrocarbons (gasoline turpentine etc.)

Supportive Care and Disposition:

Childhood poisonings require supportive treatment[77], including monitoring and continued observation. Low-risk patients with minimal symptoms, nontoxic ingestions, and no expected sequelae may be discharged to caregivers after a short observation period. High-risk patients (e.g., intentional ingestions, patients who exhibit continued toxidromes or prolonged symptoms) should be admitted to the hospital for ongoing treatment and extended observation. Psychiatric consultation is appropriate with intentional ingestion. Repeated instances of unintentional poisonings within one family should prompt a discussion about preventive measures, as well as a closer look at the caregiver situation and the possibility of child abuse or neglect**.**

Figure - 1: Different Brands of Pesticides

Figure – 2: Kerosene Oil and Commonly Used Container and Lamps

CLASSIFICATION OF SNAKES[59]

The major families in the Indian subcontinent are:

A. **Elapidae** This family includes cobras, king cobra, kraits, coral snakes, Australasian snakes and sea snakes.

B. **Hydrophidae** sea snakes

C. **Viperidae** There are two subfamilies,

a. typical vipers (Viperinae)

b. pit vipers (Crotalinae)

Pathophysiology[105,106,107,113]:

Snake venom is a complex mixture of proteins having enzymatic activities. Some important enzymes present in the venom are,

Proteinase Phosphodiesterase

Transaminase 5-Nucleotidase

Hyaluronidase ATPase

Phospholipase ABC and D Alkaline phosphatase

Ribonuclease Acid phosphatases

Deoxyribonuclease Endonucleases

Cholinesterases Metallopreoteinase

Effects of Snake Venom[103,111]:

A) Local Effects[108]:

- Direct cytotoxic

- Secondary to ischemia, compression of nerves and infection and oxidative stress

B) Haematotoxic Effects:

i) RBC

- Activation of phospholipase of RBC membrane
- Formation of lecithin and isolecithin (haemolysis).

ii) Platelets:

- Activation of coagulation system [Schwarzman like phenomenon]
- Increased adhesiveness (micro-angiopathy)

iii) Effects on coagulation:

- Coagulant effect (viper), conversion of prothrombin to thrombin, fibrinogen to fibrin.
- Anticoagulant effect – consumptive coagulopathy (fibrinogen)

C) Neurotoxic effects[102,104,110]:

- Post-synaptic block (cobra)
- Pre-synaptic block (krait)

D) Cardiotoxic effect (cobra):

- Cardiac asystole

E) Renal effect[109]:

- ATN
- ARF
- Glomerular disease
- Renal infarction
- Acute cortical necrosis

Factors Affecting Severity of Snake Bite[113]:

Factor	Effect on outcome
Size of victim	Bigger the size, good is the outcome due to less amount of toxin per kg of body weight
Comorbidity	Predisposes to harmful effect of snake venom
Part bitten	Patients bitten on the trunk, face, and directly into bloodstream have a worse prognosis
Exercise	Exertion following snake bite has poor outcome due to enhanced systemic absorption of toxin
Individual sensitivity	Sensitivity of individual to venom modifies the clinical picture
Bite characteristics	Bite number; depth of bite; dry bite; bite through clothes, shoes, or other protection; amount of venom injected; condition of fangs; and duration for which snake clings to the victim, all affect outcome
Snake species	Different species have different lethal dose, lethal period, and aggressiveness
Secondary infection	Presence or absence of pathogenic organisms in the mouth of the snake
Treatment	Nature of first aid given and time elapsed before first dose of antivenom.

Figure - 3: Common Poisonous Snakes in India

Figure - 4: Indian Cobra

Figure - 5: Live and Dead Russel Viper Snake Brought by Patient Attenders in Our Hospital

Laboratory Abnormalities:

In severe cases, Anemia, Leucocytosis, Thrombocytopenia, Hypofibrinogenemia, Protenuria, Azotemia can occur, 20WBCT positive in hematotoxic snake bites.

ECG Findings:

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.

Management[114,122]:

It is important to determine whether the patient has been actually bitten by a poisonous snake. Look for fang marks, presence of local pain, oedema, numbness or weakness. Their absence speaks against snake venom poisoning.

First aid

The case management at the field level should include

a. Reassurance: Reassure the victim that death is not imminent and medical care is available. Control anxiety as excitement will increase heart rate and lead to spread of venom
b. Immobilizing the bitten limb: Make the victim lie flat with bitten limb below the heart level. Immobilize the victim's bitten limb using a splint and lightly put a bandage
c. Remove shoes, rings, watches, jewelry and tight clothing from the bitten area as they can act as a tourniquet when swelling occurs.
d. Get the victim to the nearest secondary or tertiary care hospital where antivenom can be provided.
e. Tell doctor about any symptoms that develop

Do not's

- Do not apply a tourniquet
- Do not wash the bite site with soap or any other solution to remove the venom
- Do not make cuts or incisions on or near the bitten area
- Do not use electrical shock
- Do not freeze or apply extreme cold to the area of bite
- Do not apply any kind of potentially harmful herbal or folk remedy
- Do not attempt to suck out venom with your mouth
- Do not give the victim drink, alcohol or other drugs
- Do not attempt to capture, handle or kill the snake and patients should not be taken to quacks.

Snake bite treatment protocol[114]

Includes dealing with airway, breathing and treatment of shock, administer tetanus toxoid if skin is breached and antibiotics if there is cellulitis or local necrosis.

The treatment can be divided into

Diagnostic phase:

Wherever possible, try to identify the snake responsible. Snake coloration, its pupil shape and bite marks are unreliable means of determining species, generally scalation helps. Ask the victim relatives to carefully bring the snake to hospital if it has been killed and then use the snake identification material to identify it. Determine if any traditional medicines have been used as they can sometimes lead to confusing symptoms. Determine the exact time of bite which helps in determining progression of signs and symptoms.

Hemostatic abnormalities are the prima facie evidence of a viper bite. Cobras and kraits do not cause hemostatic disturbances. Sawscaled vipers do not cause renal failure where as Russell's viper and hump-nosed pit viper do. Russell's viper can also manifest with neurotoxic symptoms in a wide area of India which can cause confusion. Further work is necessary to determine the areas in which this species exists. The neurotoxic symptoms in Russell's viper are believed to be due to presence of a presynaptic toxin like that in common Krait.

All the patients should be kept under observation for a minimum of 24 hours. Many species, particularly the Krait and the hump-nosed pit viper are known for delayed appearance of symptoms which can develop after 6–12 hours.

Investigations:

Twenty-minute whole blood clotting test (20WBCT) is considered as reliable test of coagulation which can be carried out by bedside and is considered to be superior to 'capillary tube' method for establishing clotting capability in snake bite. A few milliliters of fresh venous blood should be placed in a fresh, clean and dry glass vessel preferably test tube and left undisturbed at ambient temperature for 20 minutes.

After that tube should be gently tilted to detect whether blood is still liquid and if so then blood is incoagulable. The test should be carried out every 30 minutes from admission for 3 hours and then hourly after that.

Other useful tests if facilities available:

a. Hb% ,platelet count, peripheral smear, prothrombin time, activated partial thromboplastin time, fibrin degradation products, d-dimer,
b. Urine examination for proteinuria, RBC, hemoglobinuria, Myoglobinuria,
c. Biochemistry for serum creatinine, Urea, Potassium,
d. ECG, X-ray, CT, USG (The use of X-ray and ultrasound are of unproven benefit, apart from identification of clot in viperine bite),
e. Oxygen saturation, arterial blood gas (ABG),
f. Enzyme-linked immunosorbent assay (ELISA) to confirm snake species.

Treatment phase:

Pain can be relieved with oral paracetamol or tramadol. Aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs) should not be administered.

Before removal of the tourniquet, check for the presence of pulse distal to it. If it is absent, ensure doctors presence before removal who should be able to handle complications such as sudden respiratory distress or hypotension. If the tourniquet has occluded the distal pulse, then blood pressure cuff should be applied and pressure should be slowly reduced.

Anti-snake venom (ASV) is the mainstay of treatment. In India, polyvalent ASV, i.e. effective against all the four common species; Russell's viper, common cobra, common Krait and saw-scaled viper and no monovalent ASVs are available. There are known species such as the humpnosed pit viper (Hypnalehypnale) where polyvalent ASV is ineffective. ASV is produced both in liquid and lyophilized forms.

Anti snake venom administration

Anti-snake venom should be administered only when there are definite signs of envenomation, i.e. coagulopathy or neurotoxicity. Only unbound, free flowing venom in bloodstream or tissue fluid, can be neutralized by it. It carries the risk of anaphylactic reaction and should be prepared to handle such reactions.

Prophylaxis for Anti-snake Venom Reactions

Two regimens are normally recommended, i.e. hydrocortisone (100 mg) + antihistamine or 0.25–0.3 mg adrenaline subcutaneously.

Anti-snake Venom Test Dose

Test doses have not been shown to have predictive value in predicting anaphylactic reaction or late serum sickness and not recommended.

Anti-snake Venom Dose

Initial Dose

- Mild envenomation (systemic symptoms manifest > 3 hours after bite) neurotoxic/hemotoxic 8–10 Vials
- Severe envenomation (systemic symptoms manifest < 3 hours after bite) neurotoxic or hemotoxic 8 Vials

Each vial is 10 ml of reconstituted ASV. Children should receive the same ASV dosage as adults.

Further doses

- ASV should be administered either as intravenous infusion (5–10 mL/kg body weight) or as slow intravenous (IV) injection i.e. 2 mL/min).
- ASV should be administered over 1 hour at constant speed and patient should be closely monitored for 2 hours.
- In victims requiring life saving surgery a higher initial dose of ASV is justified (up to 25 vials) solely on the presumption that coagulation will be restored in 6 hours.
- Local administration of ASV near or at the bite site should not be done. It is ineffective, painful and can raise the intra compartmental pressure.

Victims Who Arrive Late

A frequent problem witnessed in our country is victims who arrive in hospital several days after the bite usually with acute renal failure. The key determining factor to decide on ASV treatment is presence of current venom activity as venom can only be neutralized only if it is unattached. Perform a 20WBCT to determine if any coagulopathy is present. If it is present, administer ASV, otherwise treat renal failure. In the case of neurotoxic envenoming where the victim is having symptoms such as ptosis, respiratory failure, etc. it is probably wise to administer 1 dose of 8–10 vials of ASV to ensure that no unbound venom is present. However, at this stage it is likely that most of the venom is bound and respiratory support will be required.

Anti-snake Venom Reactions

Anaphylaxis with ASV may be life-threatening. The patient after ASV administration should be monitored closely and if anaphylaxis is evident, ASV should be discontinued. Antihistaminics can be administered to control the reaction and if severe, adrenaline should be administered. Once the patient has recovered, the ASV can be restarted slowly after 10–15 minutes, keeping close observation. Late serum sickness can be treated with oral prednisolone and/or antihistaminics.

Neostigmine is an anticholinesterase, which is particularly effective in postsynaptic neurotoxins such as those of cobra and is not useful against presynaptic neurotoxin i.e. common Krait and the Russell's viper. Neostigmine test should be performed by administering 0.5–2 mg IV and if neurological improvement occurs, it should be continued every 30 min over next 8 hours.

Repeat Doses of Anti-snake Venom

- Repeat doses of haemotoxic viperine snakes is based on the 6 hour rule upto maximum of 30 vials.
- Repeat doses of neurotoxic snakes is based on the 1-2 hour rule upto maximum of 20 vials

Follow-up:

After discharge from hospital, victim should be followed. If discharged within 24 hours, patient should be advised to return if there is any worsening of symptoms such as bleeding, pain or swelling at the site of bite, difficulty in breathing, altered sensorium, etc. The patients should also be explained about serum sickness which may manifest after 5–10 days.

Figure - 6: Patients with Renal Failure With Facial Puffiness Bitten By Snake And Unknown Insect Bite

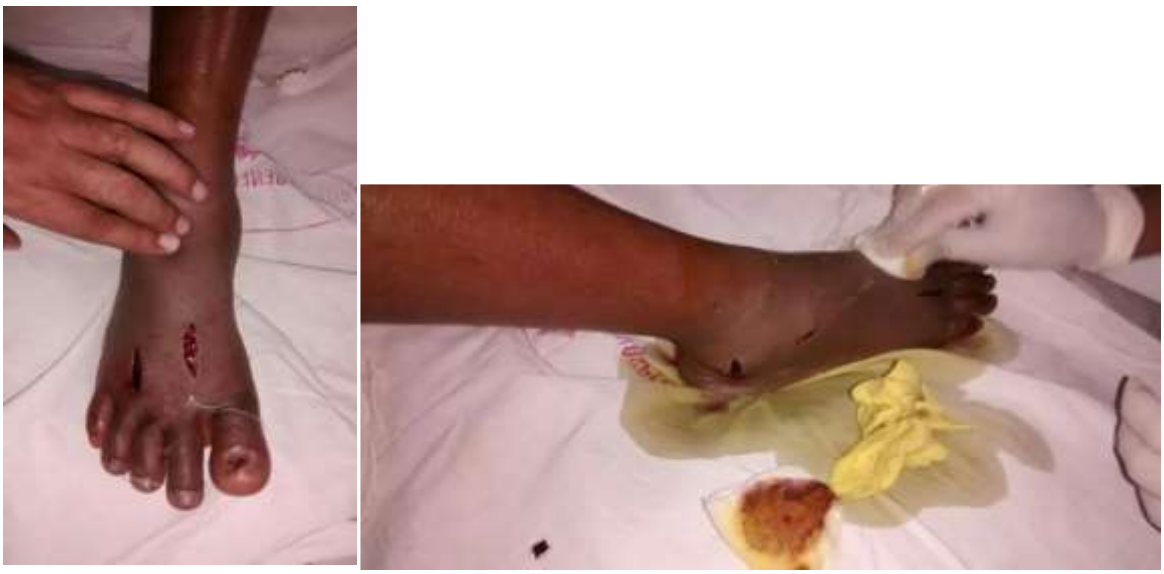


Figure - 7: Patient Showing Local Tissue Reaction with Compartment Syndrome Underwent Fasciotomy

Scorpion sting:

Scorpion envenomation is a threat to more than 2 billion people worldwide with an annual sting number exceeding one million. Acute heart failure presenting as cardiogenic shock or pulmonary edema, or both is the most severe presentation of scorpion envenomation accounting for 0.27% lethality rate. Although adults are more often concerned, children experience more severe envenomations and among them mortality is higher[115,116,117].

There are about 1500 species of scorpions worldwide, out of these 50 are dangerous to human. Among 86 species in India, Mesobuthus tamulus (Indian red scorpion) and Heterometrus swammerdami are of medical importance[118]. Scorpions have crab-like appearance with long, fleshy, five segmented, tail like post-abdomen, ending in a broad sac and a prominent hollow sting. The venom containing glands are present in telson, the last tail segment and the venom passes by a duct attached to these glands. Maximum incidence of scorpion bite occurs in summer and rainy season.

Pathophysiology:

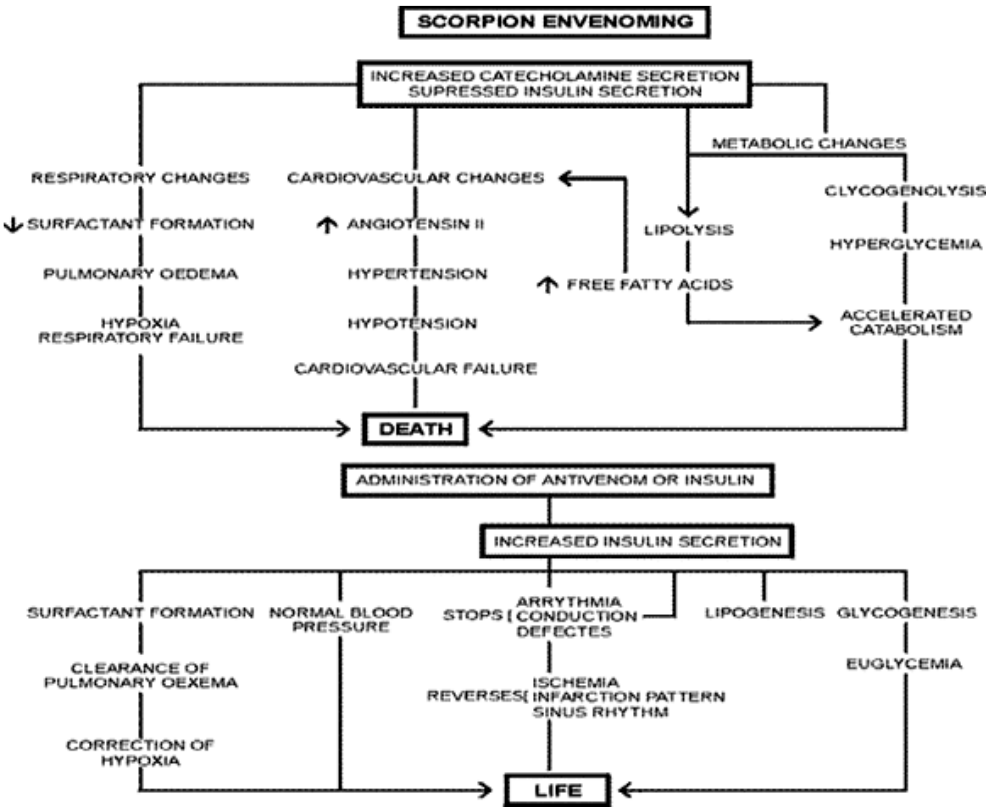
The scorpion venom is water soluble antigenic complex mixture of neurotoxin, cardiotoxin, nephrotoxin, hemolysins, phosphodiesterases, phospholipase, hyaluronidases, histamine, and other chemicals. The primary target of scorpion venom is voltage-dependent ion channels[118].

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.

Alpha toxin inhibits the inactivation of the neuronal sodium channels resulting in Delayed closure of the neuronal sodium channels result in pouring of endogenous catecholamines(epinephrine and nonepinephrine) and other vasoactive peptide hormones such as neuro-peptide-Y and endothelin-1. Their simultaneous stimulation results in an ‘autonomic storm’[119].

Flow Chart – 1: Mechanism of Scorpion Venom on Body and Mechanism of Action of Antivenom or Insulin



Early cardiovascular dysfunction is related

- **Vascular phase** of scorpion envenomation, which is related to catecholamine related vasoconstriction leading to sharp increase in left ventricular after load, there by impeding left ventricular emptying, and increasing left ventricular filling pressure.
- **Myocardial phase** occurs, characterized by a striking alteration in left ventricular contractility, low cardiac output and hypotensive state[115].

Clinical features[119]:

These are due to autonomic storm resulting in

a. Early parasympathetic effects

b. Long lasting sympathetic effects.

The clinical manifestations are either local or systemic. The local manifestations are intense local pain, swelling, 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 stool and urine
- Priapism and ejaculation

II. CNS:

- Encephalopathy
- Convulsions (focal or generalized)

Cardiovascular:

- Hypertension / hypotension
- Arrhythmia
- 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 or ventricular)

Echocardiography; Myocardial dysfunction either focal or generalized and ventricular dilatation.

Respiration:

- Dyspnoea
- Cyanosis
- Pulmonary oedema

Gastro-intestinal:

- Abdominal pain
- Hematemesis
- Malena

Metabolic:

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

Renal:

- Hematuria
- Oliguria
- Acute renal failure

Hematological:

- Increased erythrocyte fragility
- Disseminated intravascular coagulation

Hepatobiliary:

- Raised transaminases (increased AST, ALT)
- Raised bilirubin

Miscellaneous:

- Muscle fasciculation
- Tetany like contractures

Diagnosis:

Scorpion envenomation is diagnosed on the basis of;

- History of scorpion sting
- Clinical features
- Lab investigations[118]
- Urine analysis
- Blood glucose
- Estimation of serum amylase, lactic acid, AST, ALT and LDH
- Blood gas analysis
- ECG
- Chest X-Ray

<u>Management of Scorpion Sting:</u>

Management of scorpion sting has been divided into two groups; i.e., Local management and Systemic management.

Local Management:

- A ligature should be applied immediately, and it should be 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 affected part should be done.
- Pain management by local anaesthetic agent preferably 2% xylocaine hydrochloride.
- Tetanus toxoid should be administered.

Systemic management[115,119,120,121]:

- Supportive management for complications like ventillatory support for respiratory failure, dobutamine infusion for cardiac support,
- If the patient has developed pulmonary oedema, it should be managed with100% 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, encephalopathy should be managed conservatively.
- **Specific treatment** with neutralizing scorpion venom with scorpion antivenom(SAV) A single 30 mL dose of monovalent antivenom (Haffkine Biopharma) was added to 100 mL of normal saline, and infused intravenously over 30 minutes.
- And prazocin (30 μg/kg every 3 hourly). Early administration as early as within 4 hrs improves the outcome.

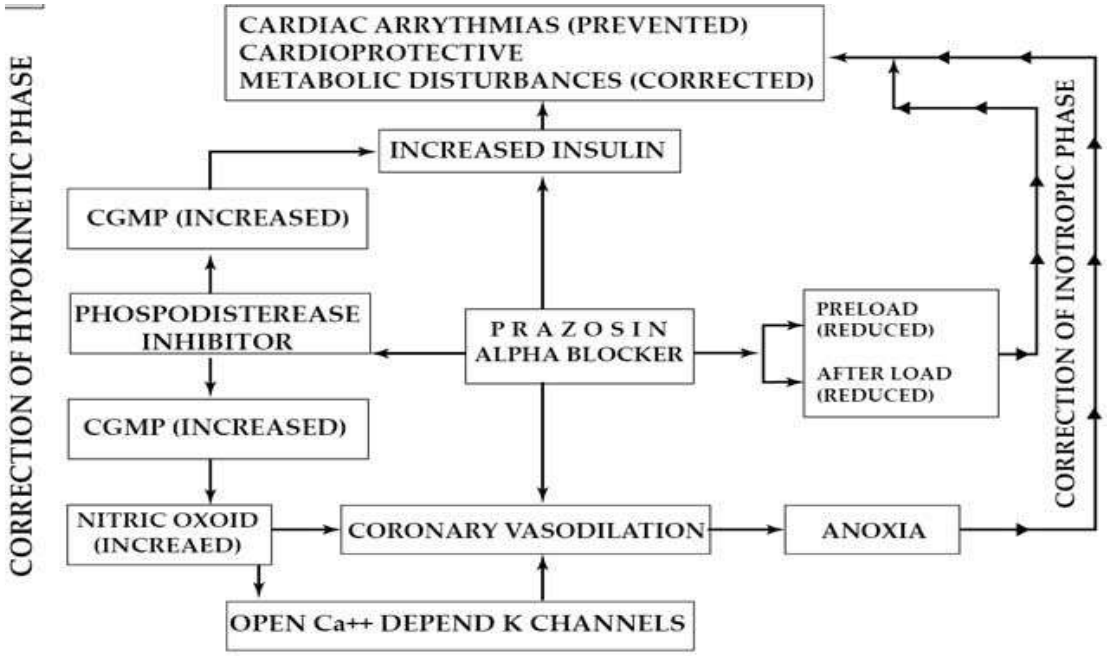


Fig. 14 : Effects of prazosin

- Steroids worsens the clinical manifestations
- Antihistamines, should be avoided
- Calcium channel blockers also should be avoided
- Diuretics enhance the development of pulmonary edema and shock, and must be avoided in severe scorpion envenomation[119].
- Atropine should not be given routinely. The only real indication of atropine is the presence of severe bradycardia with or without hypertension.

MATERIALS AND METHODS

Study area:

Department of Bokaro General Hospital, Bokaro Steel City, Jharkhand

Study population:

All children aged 18 years and below attending to emergency department and admitted with History of either Deliberate Self Harm or Accidental Poisoning.

Study design:

Cross sectional Observational Study.

Sample size calculation:

Previously researchers have performed studies on the incidence of poisoning in children aged up to 18 yrs. The incidence found in these articles ranges 0.33 – 7.6%[3,4]. Therefore, assuming 2% as the incidence of poisoning in children with 0.6% margin of error, the minimum required sample size at 5% level of significance is 2100 cases.

Formula used: n =

Where *p* is the observed incidence of poisoning in children aged up to 18 yrs

$q = 1 - p$

d is the margin of error

is the ordinate of standard normal distribution at α% level of significance.

Study period:

The present study was conducted in the Department of Pediatrics, Bokaro General Hospital, Bokaro Steel City, Jharkhand from January 2015 to December 2015.

Inclusion criteria:

- All patients with history of consumption of poison having positive and significant signs and symptoms, accompanied or unaccompanied by container or poison.
- Patients with doubtful history of consumption of poison but with definite signs and symptoms of acute poisoning
- All patients with history of bites having positive and significant signs and symptoms due to poisonous creatures like snakes, scorpions, bees and insects.
- Patients with doubtful history of bites due to poisonous creatures but with definite acute onset of signs and symptoms locally or systemically.

Exclusion criteria:

- Food poisoning
- Idiosyncratic reactions to drugs
- Patients with history of bite but having no signs and symptoms either locally or systemically
- Chronic poisonings due to heavy metals, air pollutants etc.

Methodology:

All records regarding the type of poisoning (homicidal, suicidal or accidental) was taken by a detailed history and then grouped under 3 categories according to the age groups (1-5 years, 6-12 years and 13-18 years) were divided. Analysis of the cases with respect to age, sex, type of poison, mode of ingestion/ bite, seasonal variation, from Rural or Urban area they have came, reasons of poisoning, socioeconomic status and outcome was analyzed.

Statistical Methods:

Descriptive statistics was analyzed with SPSS version 17.0 software. Continuous variables are presented as mean±SD. Categorical variables are expressed as frequencies and percentages

Nominal categorical data between two categorical variables was compared using Chi-squared test or Fisher's exact test as appropriate.

A p value less than 0.05 will be taken to indicate a significant difference.

Ethical consideration:

The proposed study was approved by institutional ethical committee of Bokaro General Hospital, Bokaro Steel City.

RESULTS AND OBSERVATIONS

In the present study 2255 children aged 18 years and below admitted in emergency department over 1 year from January 2015 to December 2015. Among these 123 children were presented with history and/or symptoms and signs of poisoning, and admitted for the same. It was 5.45% of total emergency admissions.

For the purpose of better study we divided patients into :

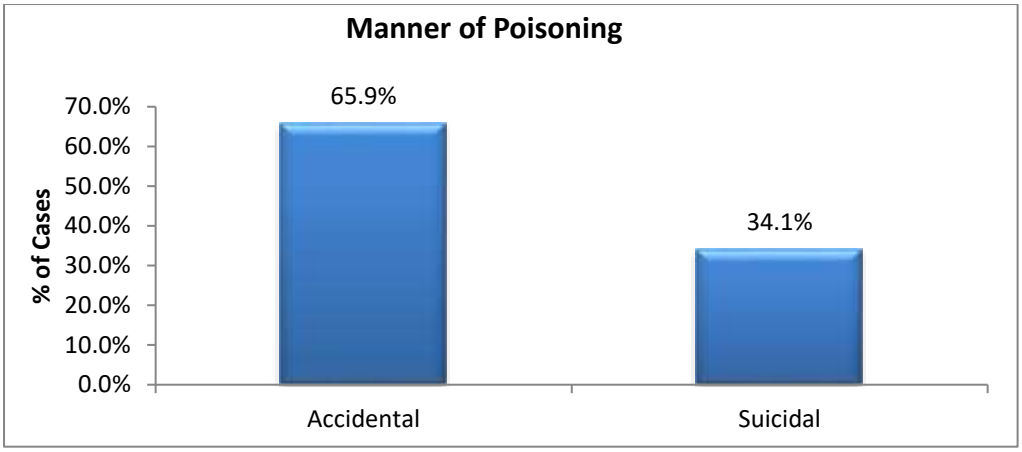
- Poisoning in general
- Accidental poisoning (excluding insect bite)
- Suicidal poisoning
- Insect bite poisoning

Manner of poisoning

Among total 123 poisoning cases, accidental poisoning was noted in 81(65.9%) children. Poisoning due to suicidal tendency noted in 42 (34.1%).

Table – 3 : Manner of Poisoning

Manner of Poisoning	**Frequency**	**%**
Accidental	81	65.9%
Suicidal	42	34.1%
Total	123	100%



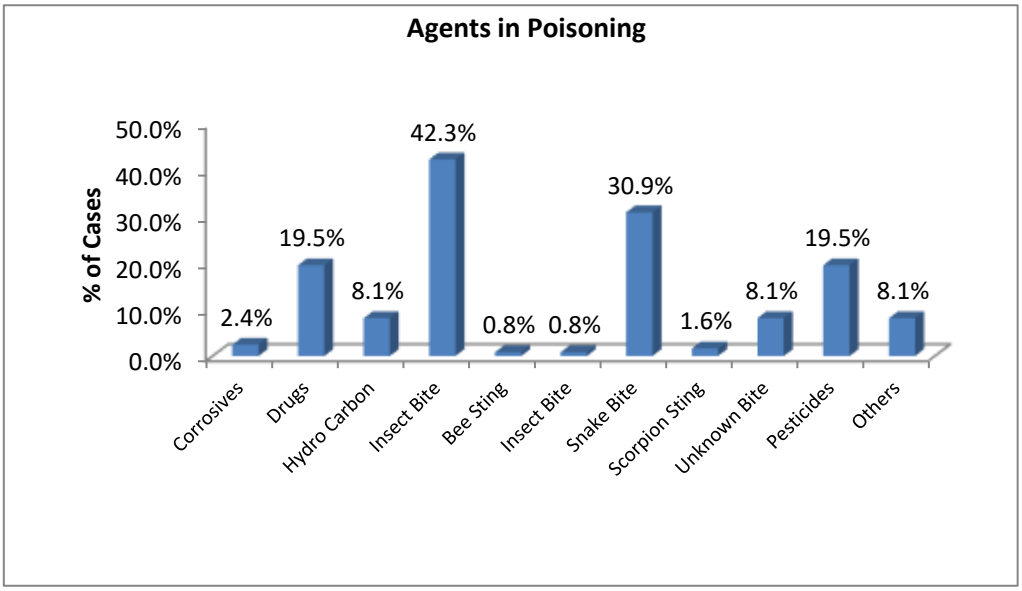
Graph – 1: Manner Of Poisoning

AGENTS IN POISONING IN GENERAL:

Table – 4: Agents In Poisoning

Group	Frequency	%
Corrosives	3	2.4%
Drugs	24	19.5%
Hydro Carbon	10	8.1%
Insect Bite	52	42.3%
Bee Sting	1	0.8%
Insect Bite	1	0.8%
Snake Bite	38	30.9%
Scorpion Sting	2	1.6%
Unknown Bite	10	8.1%
Pesticides	24	19.5%
Others	10	8.1%
Total	123	100%

Poisoning due to various agents include insect bites (n=52) 42.3%, drugs (n=24), pesticides (n=24) constitutes 19.5% each, hydrocarbons (n=10) 8.1%, corrosives (n=3) 2.4%, others (n=10) 8.1%.



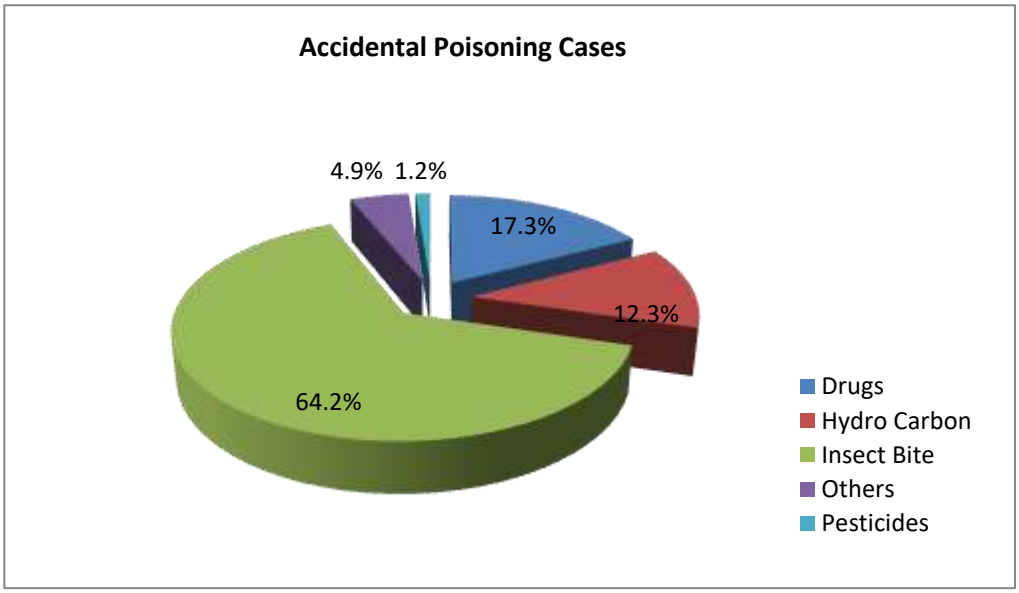
Graph – 2: Agents in Poisoning

ACCIDENTAL POISONING INCLUDING INSECT BITES:

Table – 5 : Agents in Accidental Poisoning

Accidental poisoning		
	Frequency	**%**
Drugs	14	17.3%
Hydro Carbon	10	12.3%
Insect Bite	52	64.2%
Others	4	4.9%
Pesticides	1	1.2%
Total	81	100%

In this study most common causes of accidental poisoning was insect bites (n=52), drugs (n=14), hydrocarbons (n=10) others (n=4) include H_2O_2, plant poisoning, bhang and all out each, and a single case of pesticide poisoning was seen.



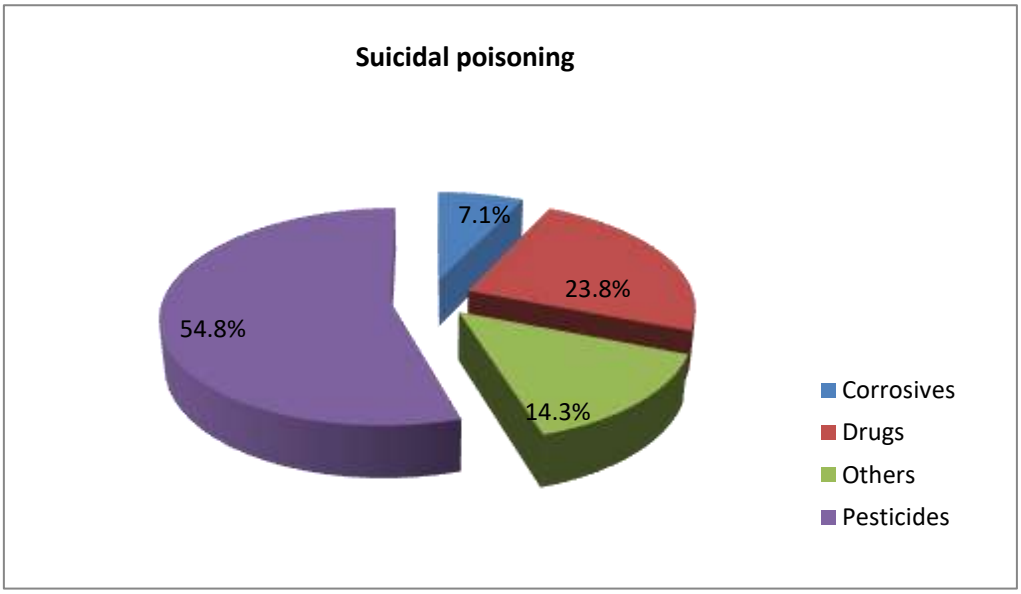
Graph – 3: Agents In Accidental Poisoning

SUICIDAL POISONING:

Table – 6: Agents in Suicidal Poisoning

Suicidal poisoning		
	Frequency	**%**
Corrosives	**3**	7.1%
Drugs	10	23.8%
Others	6	14.3%
Pesticides	23	54.8%
Total	42	100%

Suicidal poisoning was due to pesticides (n=23), drugs (n=10), corrosives (n=3) and others (n=6) include cut injury of arm (n=2), all out (n=2) and miscellaneous (n=2) was seen.



Graph – 4 : Agents in Suicidal Poisoning

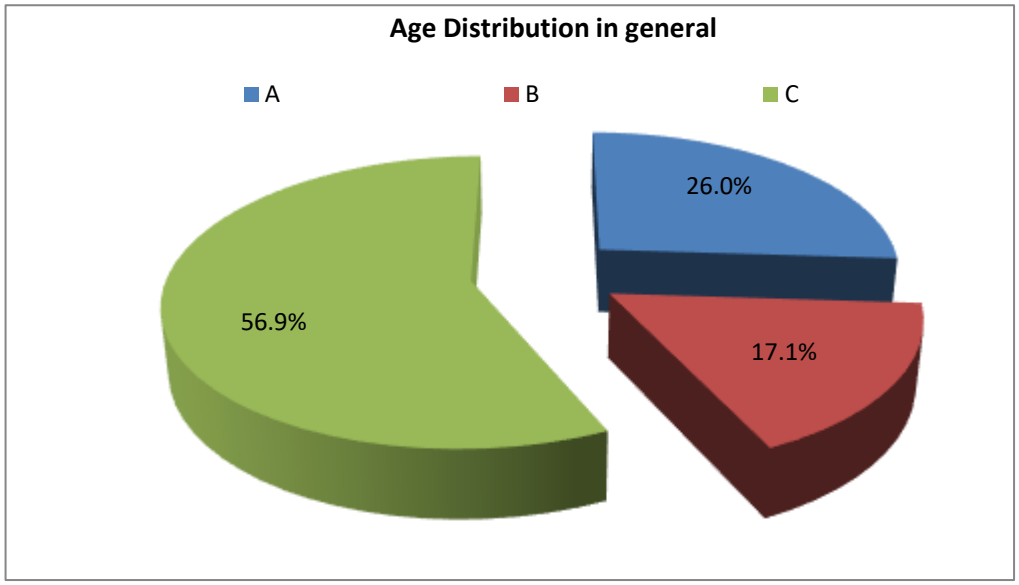
AGE:

In general:

Table – 7: Age Distribution (In General)

Age Groups	Frequency	%
0-5yrs	32	26.0%
6-12yrs	21	17.1%
13-18yrs	70	56.9%
Total	123	100%

In this study children between 13-18yrs (n=70) were most effected 56.9%, followed by 0-5yrs (n=32) which was 26% of total poisoning, followed by 6-12yrs (n=21) 17.1%.



Graph – 5: Age Distribution (In General)

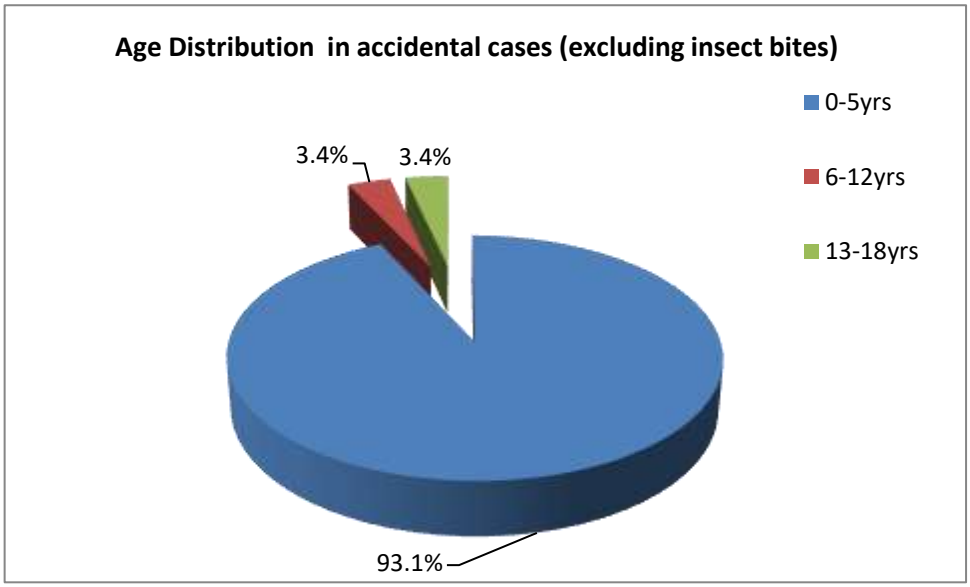
IN ACCIDENTAL POISONING EXCLUDING INSECT BITES:

Table – 8: Age Distribution in Accidental Cases

(Excluding Insect Bites)

Accidental poisoning		
Age Groups	**Frequency**	**%**
0-5yrs	27	93.1%
6-12yrs	1	3.4%
13-18yrs	1	3.4%
Total	29	100%

In this study most common age involved in accidental poisoning was 0-5 yrs. Which was 93.1% (n=27) of total accidental poisoning excluding insect bites.



Graph – 6: Age Distribution in Accidental Cases

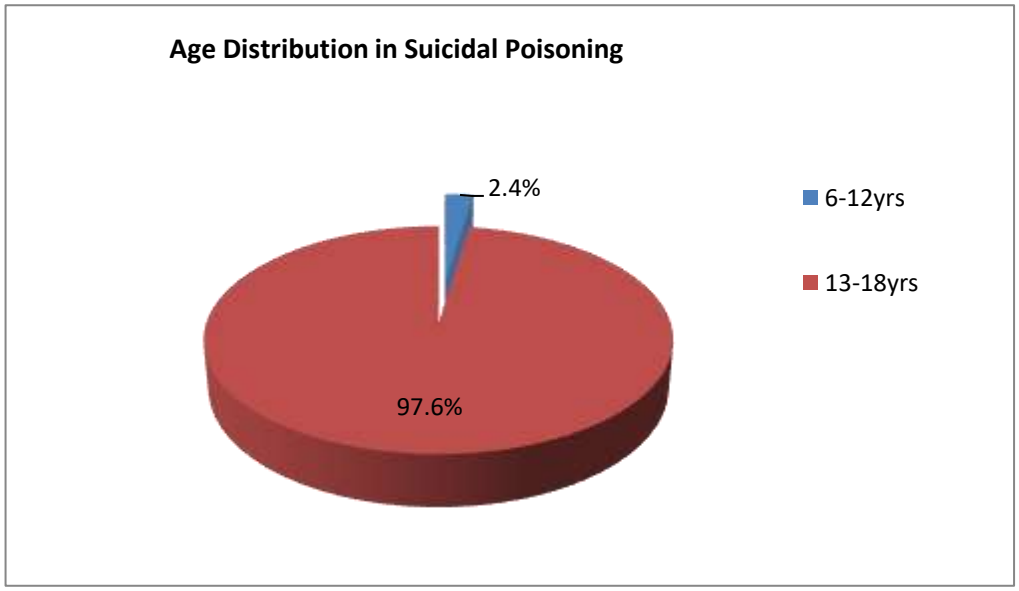
(Excluding Insect Bites)

IN SUICIDAL POISONING:

TABLE – 9: AGE DISTRIBUTION IN SUICIDAL POISONING

Suicidal poisoning		
Age Groups	**Frequency**	**%**
6-12yrs	1	2.4%
13-18yrs	41	97.6%
Total	42	100%

In this study suicidal poisoning more commonly seen in the age group of 13-18 yrs (97.6%), where as it was only 2.4% between 6-12 yrs age group and none in the age group of 0-5yrs.



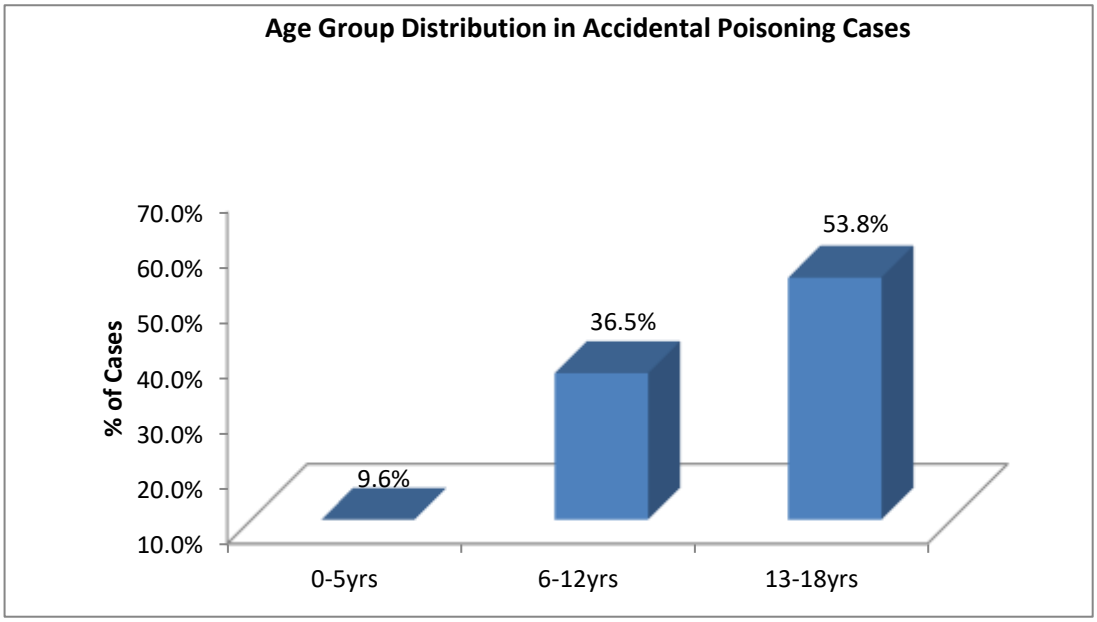
Graph – 7: Age Distribution in Suicidal Poisoning

IN INSECT BITES:

Table – 10: Age Distribution In Insect Bites

Insect bites		
Age Groups	**Frequency**	**%**
0-5yrs	5	9.6%
6-12yrs	19	36.5%
13-18yrs	28	53.8%
Total	52	100%

In this study cases due to insect bites increasing along with the age, reflecting as 9.6% (n=5) in the age group of 0-5 yrs, 36.5% (n=19) in 6-12 yrs age group and 53.85% (n=28) in the age group of 13-18yrs.



Graph – 8: Age Distribution in Insect Bites

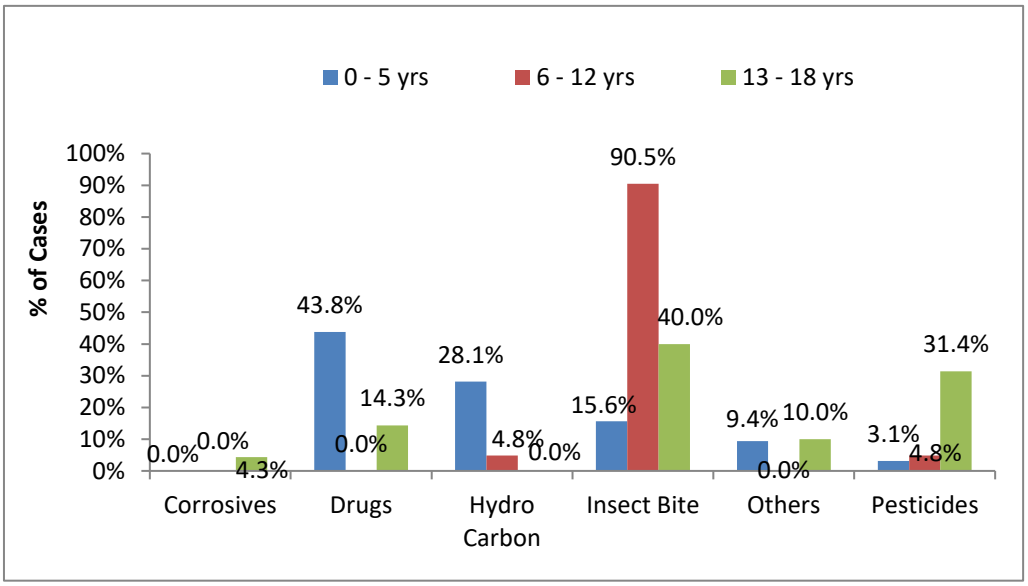
AGENTS INVOLVED IN DIFFERENT AGE GROUPS:

Table – 11: Agents Involved in Different Age Groups

Group	Total	Age Groups			P Value
		0-5yrs	6-12yrs	13-18yrs	
		Frequency (%)	Frequency (%)	Frequency (%)	
Corrosives	3	0 (0.0%)	0 (0.0%)	3 (4.3%)	<0.001
Drugs	24	14 (43.8%)	0 (0.0%)	10 (14.3%)	
Hydro Carbon	10	9 (28.1%)	1(4.8%)	0 (0.0%)	
Insect Bite	52	5 (15.6%)	19 (90.5%)	28 (40.0%)	
Others	24	3 (9.4%)	0 (0.0%)	7 (10.0%)	
Pesticides	10	1 (3.1%)	1 (4.8%)	22 (31.4%)	
Total	123	32 (100%)	21 (100%)	70 (100%)	

In this study most common agents involved in children up to 5yrs were drugs(n=14), followed by hydrocarbons (n=9)followed by insect bites (n=5). While in the age group of 6-12yrs out of

21 cases 19 cases due to insect bites (90.5%), and in the age group 13-18yr again insect bites (n=28) were more common followed by pesticides (n=22) and drugs (n=10).



Graph – 9: Agents Involved in Different Age Groups

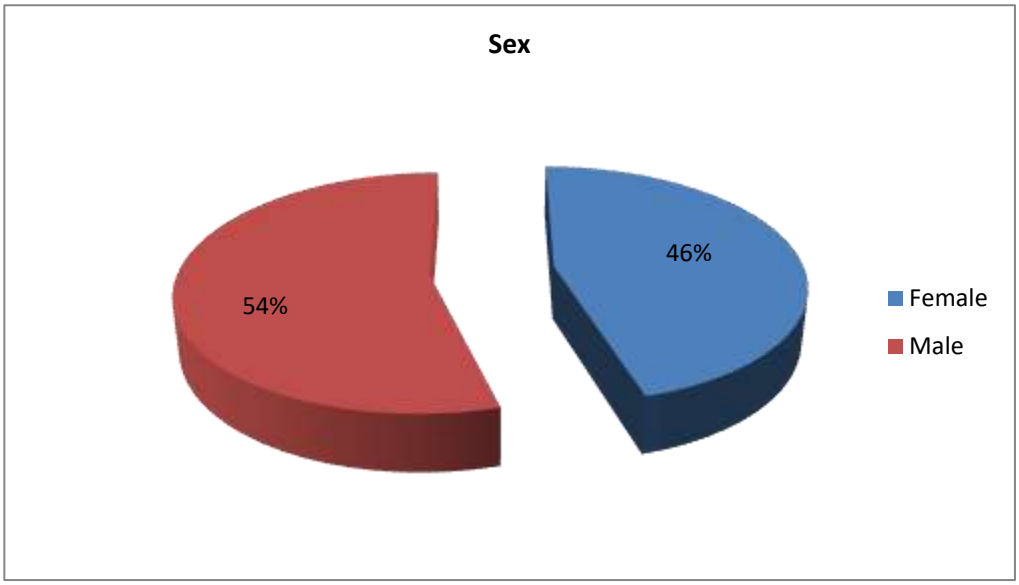
SEX:

In general:

Table – 12: Sex Distribution (In General)

Sex	Frequency	%
Female	56	45.5%
Male	67	54.5%
Total	123	100%

Males (n=67) effected more than females (n=56) making ratio of 1.2**:** 1.



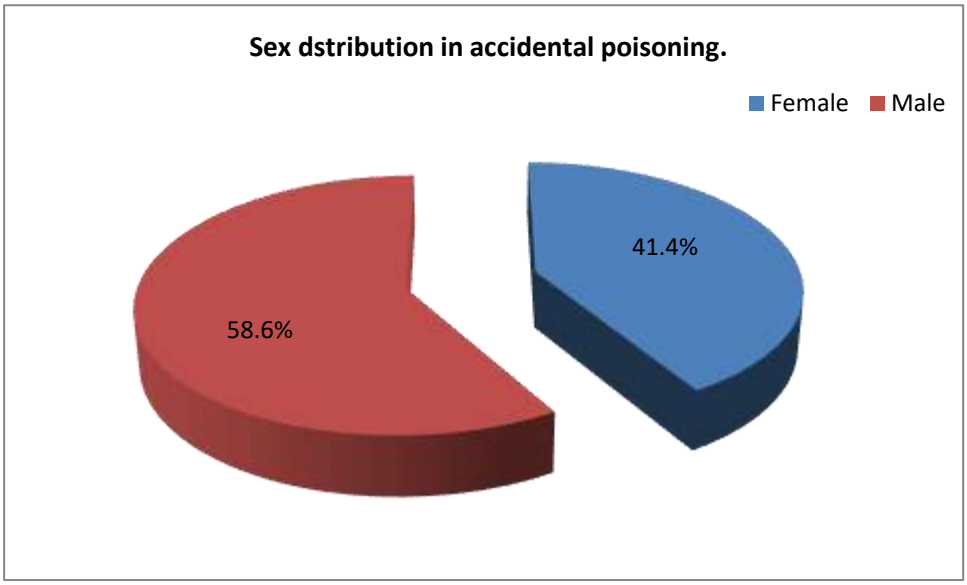
Graph – 10: Sex Distribution (In General)

IN ACCIDENTAL POISONING:

Table – 13: Sex Distribution (In Accidental Poisoning)

Accidental poisoning		
Sex	**Frequency**	**%**
Female	12	41.4%
Male	17	58.6%
Total	29	100%

In this study males (n=17) constitute 58.6% of total accidental poisoning, while females were (n=12) 41.4% of total accidental poisoning due oral ingestion.



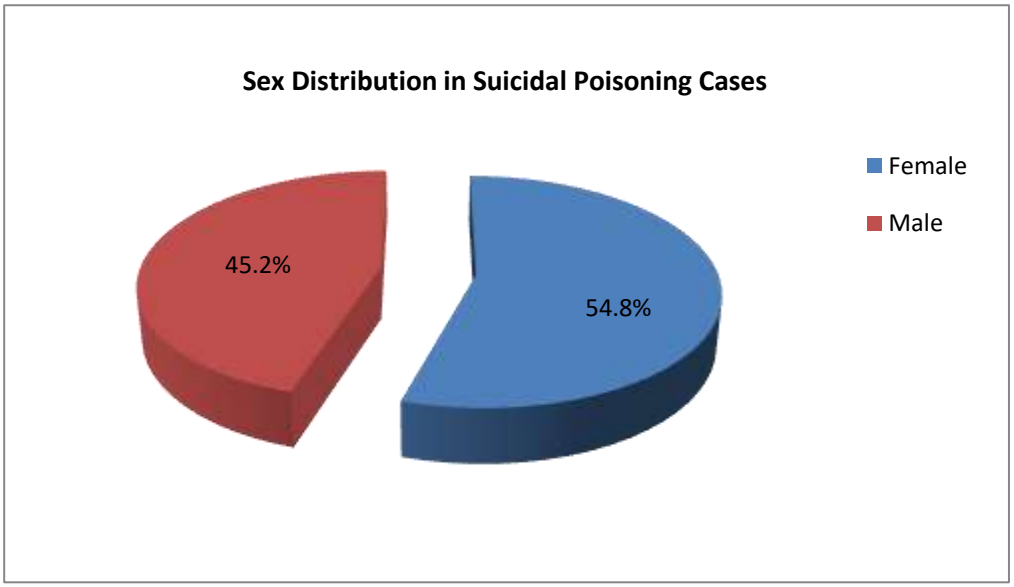
Graph – 11: Sex Distribution (In Accidental Poisoning)

IN SUICIDAL POISONING:

Table – 14 : Sex Distribution in Suicidal Poisoning

Suicidal poisoning		
Sex	**Frequency**	**%**
Female	23	54.8%
Male	19	45.2%
Total	42	100%

In this study females (n=23) were 54.8% and males (n=19) were 45.2% of total suicidal poisoning.

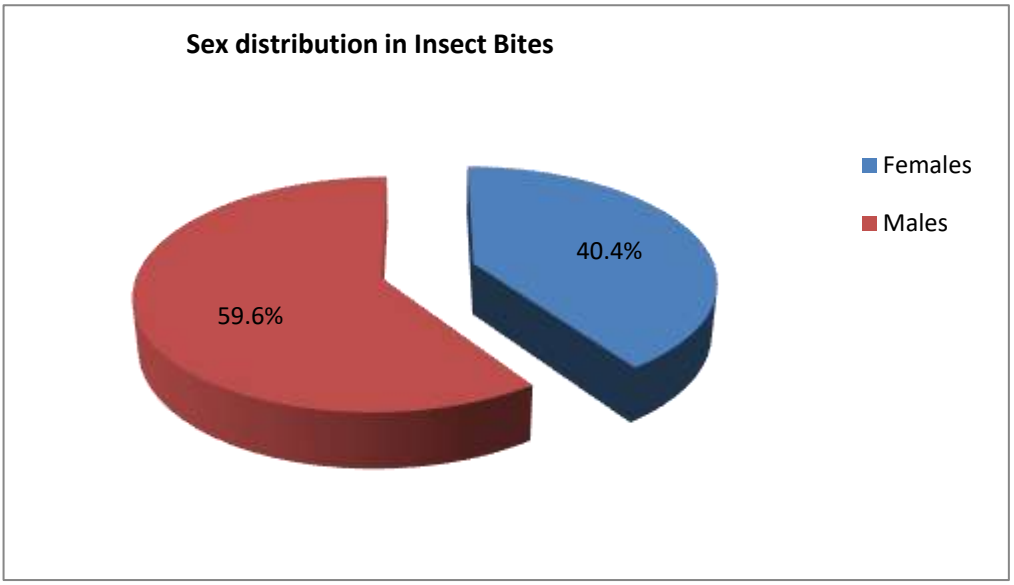


Graph – 12: Sex Distribution in Suicidal Poisoning

IN INSECT BITES:

Table – 15: Sex Distribution in Insect Bites

Insect bites		
Sex	**Frequency**	**%**
Females	21	40.4%
Males	31	59.6%
Total	52	100%



Graph – 13: Sex Distribution in Insect Bites

In this study males 59.6%males (n=31) were bitten by insects while it was 40.4% in females (n=21).

URBAN VS RURAL:

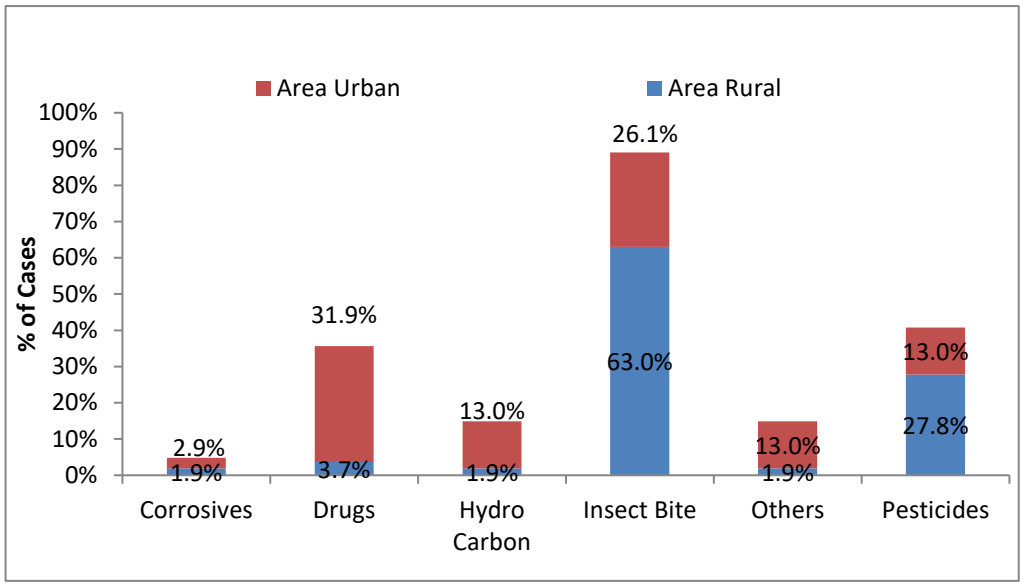
In general:

Agents involved in urban and rural areas:

Table – 16: Area Wise Distribution of Agents (In General)

Group	Total	Area		P Value
		Rural	Urban	
		Frequency (%)	Frequency (%)	
Corrosives	3	1 (1.9%)	2 (2.9%)	
Drugs	24	2 (3.7%)	22 (31.9%)	
Hydro Carbon	10	1 (1.9%)	9 (13.0%)	
Insect Bite	52	34 (63.0%)	18 (26.1%)	<0.001
Others	24	1 (1.9%)	9 (13.0%)	
Pesticides	10	15 (27.8%)	9 (13.0%)	
Total	123	54 (100%)	69 (100%)	

In rural region poisoning due to insect bites (n=34) and pesticides (n=15) were more common which was 63% and 27.8% respectively. While in urban region poisoning due drugs (n=22) followed by insect bites (n=18) and hydrocarbons (n=9) were more common making 31.9%, 26.1% and 13% respectively.

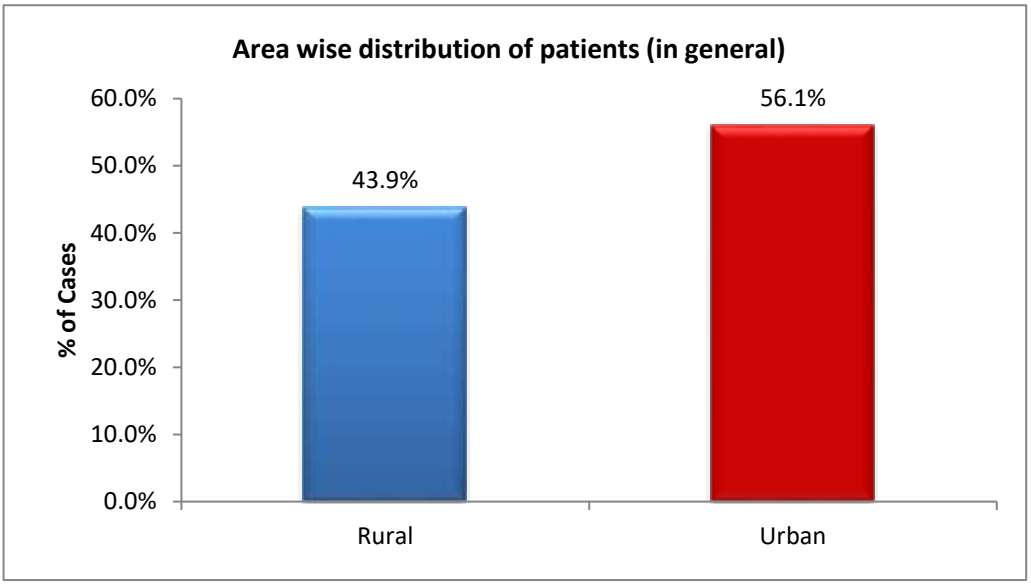


Graph – 14: Area Wise Distribution of Agents (In General)

Table – 17: Area Wise Distribution of Patients (In General)

Area	**Frequency**	**%**
Rural	54	43.9%
Urban	69	56.1%
Total	123	100%

Children in urban area (n=69) affected more than rural area (n=54) which was 56.1% and 43.9% respectively.



Graph – 15: Area Wise Distribution of Patients (In General)

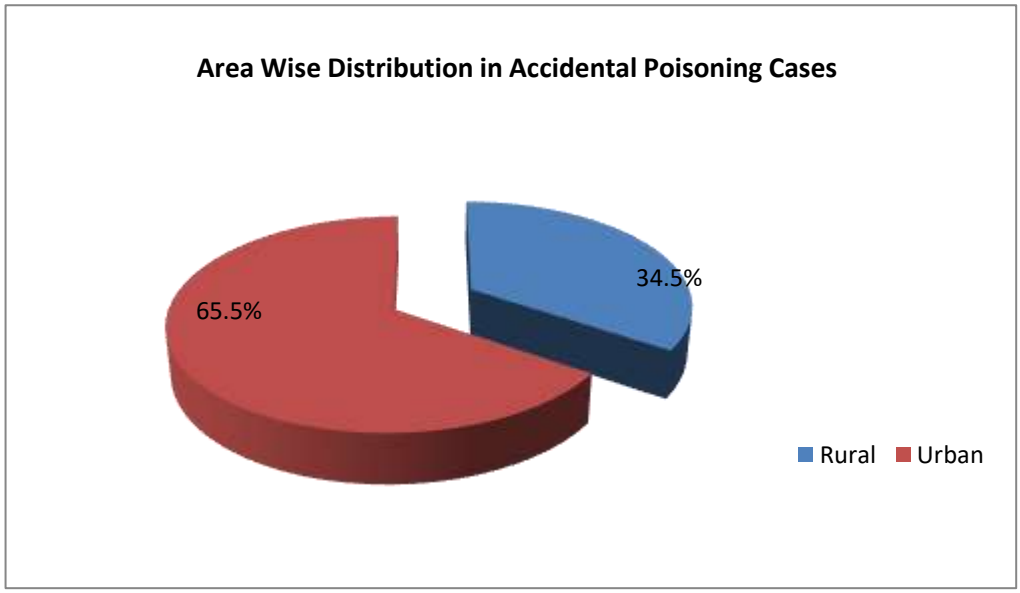
IN ACCIDENTAL POISONING AFTER EXCLUDING INSECT BITES:

Table – 18: Area Wise Distribution in Accidental Poisoning

(Excluding Insect Bites)

Accidental poisoning		
	Frequency	**%**
Rural	10	34.5%
Urban	19	65.5%
Total	29	100%

Accidental poisoning after excluding insect bites were more common in urban population (n=19, 65.5%) than rural population (n=10, 34.5%)



Graph – 16 : Area wise distribution in accidental poisoning

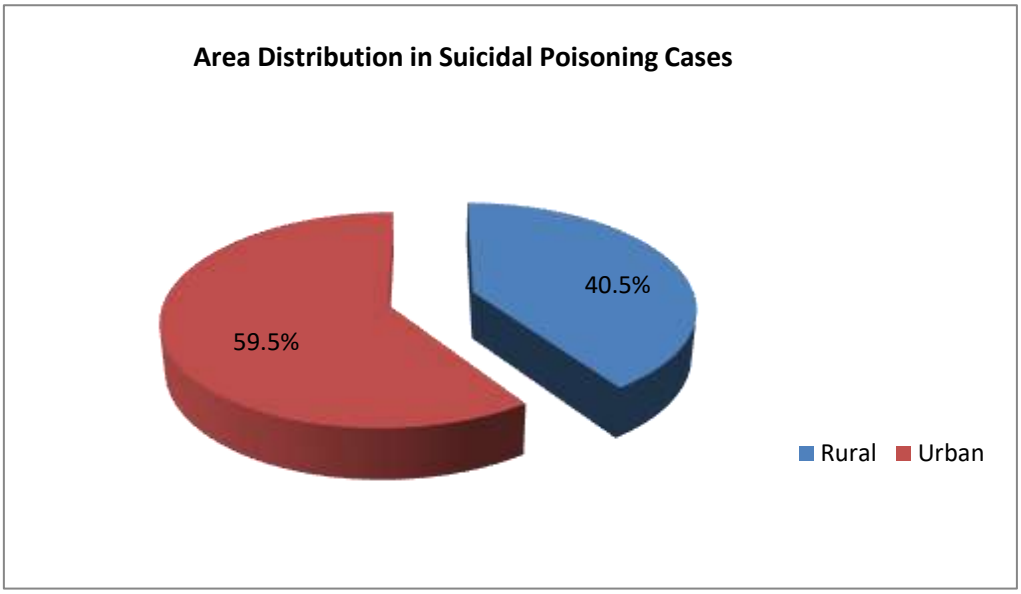
(Excluding insect bites)

IN SUICIDAL POISONING:

Table – 19: Area Wise Distribution in Suicidal Poisoning

Suicidal poisoning		
	Frequency	**%**
Rural	17	40.5%
Urban	25	59.5%
Total	42	100%

Suicidal poisoning was more common in urban population (n=25; 59.5%) than rural population (n=17; 40.5%).



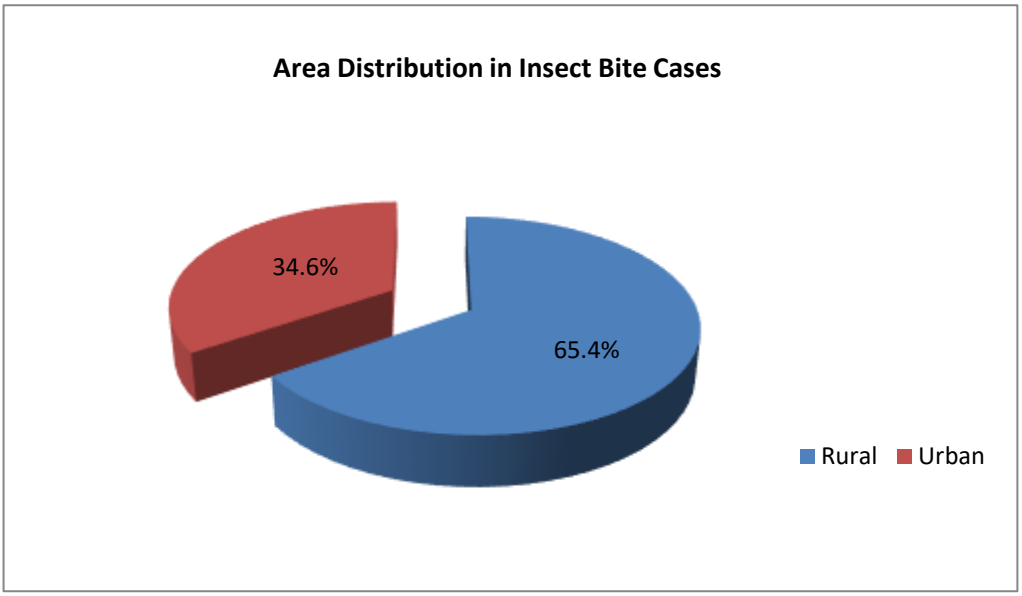
Graph – 17: Area Wise Distribution in Suicidal Poisoning

IN INSECT BITES:

Table – 20 : Area Wise Distribution in Insect Bites

Insect Bite		
	Frequency	**%**
Rural	34	65.4%
Urban	18	34.6%
Total	52	100%

Insect bites were more common in rural population than (n=34; 65.4%) than urban population (n=18; 34.6%).



Graph – 18 : Area Wise Distribution in Insect Bites

SOCIO-ECONOMIC STATUS:

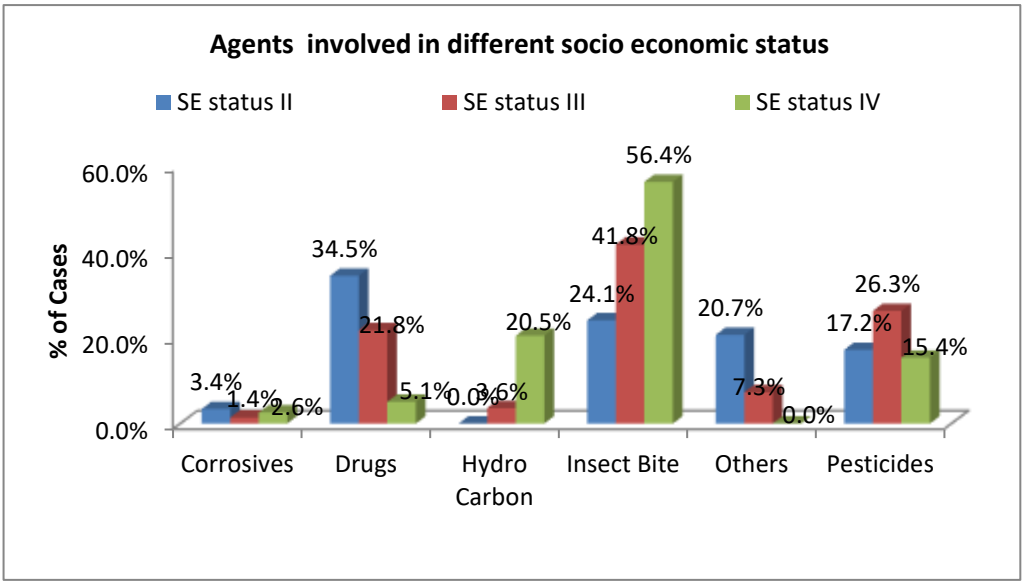
Agents in different SE-Status:

Table – 21: Agents Involved in Different Socio-Economic Status

Group	Total	SE status			P Value
		II	III	IV	
		Frequency (%)	Frequency (%)	Frequency (%)	
Corrosives	3	1 (3.4%)	1 (1.4%)	1 (2.6%)	
Drugs	24	10(34.5%)	12 (21.8%)	2 (5.1%)	
Hydro Carbon	10	0 (0.0%)	2 (3.6%)	8 (20.5%)	
Insect Bite	52	7 (24.1%)	23 (41.8%)	22 (56.4%)	<0.001
Others	24	6 (20.7%)	4 (7.3%)	0 (0.0%)	
Pesticides	10	5 (17.2%)	13(26.3%)	6 (15.4%)	
Total	123	29 (100%)	55 (100%)	41 (100%)	

In socio-economic status class IV insect bites (56.4%) more common followed by hydrocarbons (20.5%) and pesticides (15.4%). While in class III again insect bites (41.8%)

were more common followed by pesticides (26.3%) and drugs (21.8%) were common. Whereas in class II drugs (34.5%), insect bites (24.1%) others (20.7%) and pesticides (17.2%) were common. There was no hydrocarbon poisoning case in class II.



Graph – 19: Agents Involved in Different Socio-Economic Status

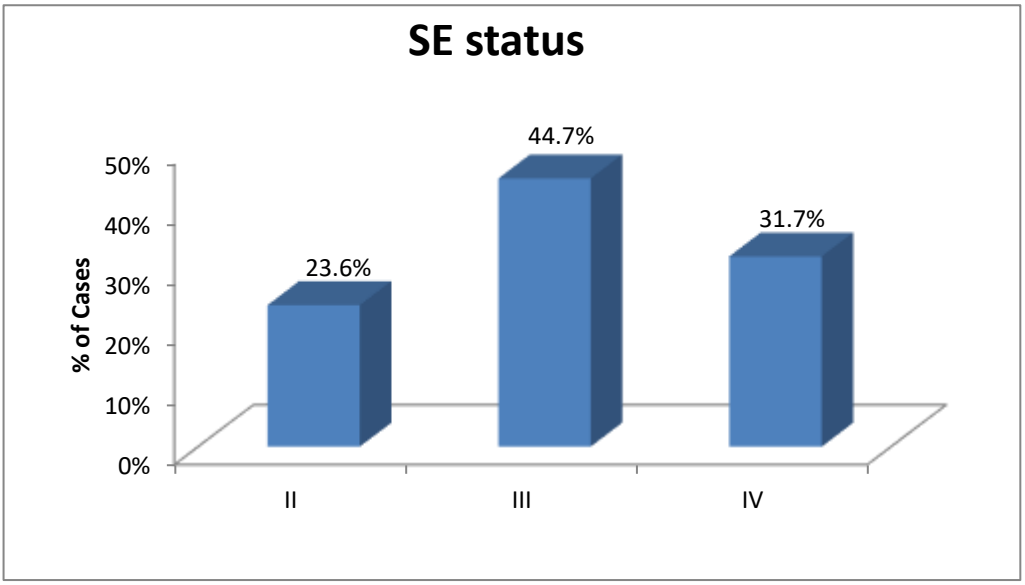
IN GENERAL:

Table – 22 : Se - Status Wise Distribution Of Patients

(In General)

SE STATUS	Frequency	%
II	29	23.6%
III	55	44.7%
IV	39	31.7%
Total	123	100%

Children belong to Socio-economic status class III more affected followed by class IV and class II according to Kuppuswamy socio-economic scale.



Graph – 20 : SE - status wise distribution of patients

(In general)

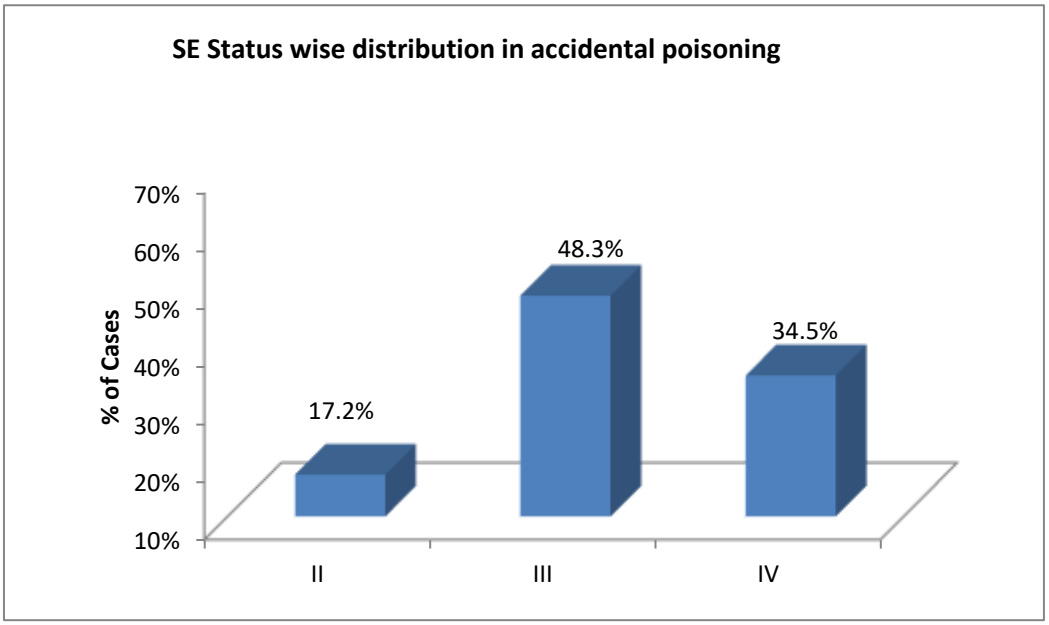
IN ACCIDENTAL POISONING AFTER EXCLUDING INSECT BITES:

Table – 23: SE - Status wise distribution in accidental poisoning

(Excluding insect bites)

Accidental poisoning		
SE Status	**Frequency**	**%**
II	5	17.2%
III	14	48.3%
IV	10	34.5%
Total	29	100%

More cases were belong to SE status class III (48.3%) followed by class IV (34.5%).



Graph – 21: SE - Status wise distribution in accidental poisoning

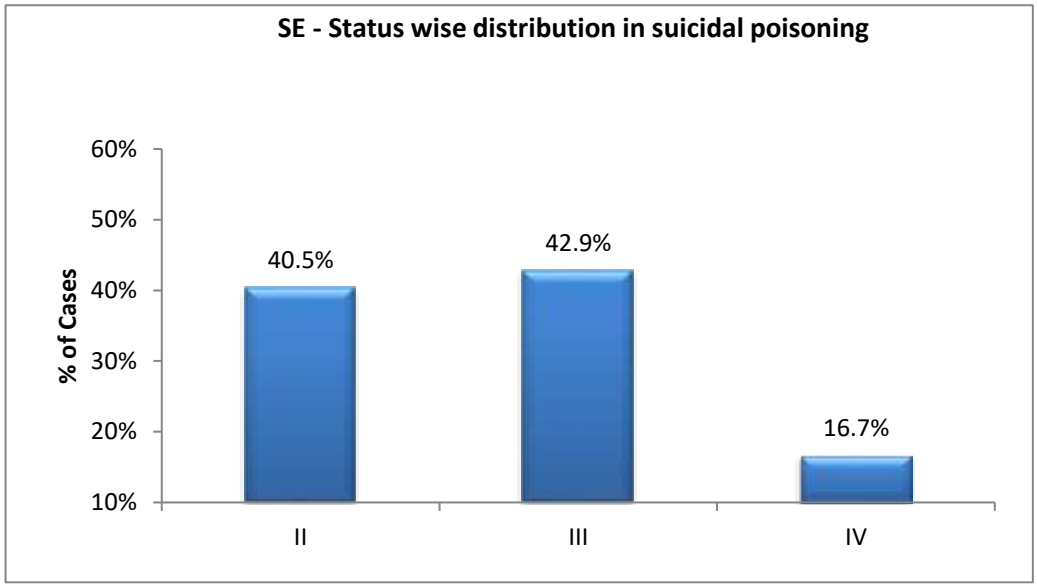
(Excluding insect bites)

IN SUICIDAL POISONING:

Table – 24 : Se - Status Wise Distribution in Suicidal Poisoning

Suicidal poisoning		
SE status	**Frequency**	**%**
II	17	40.5%
III	18	42.9%
IV	7	16.7%
Total	42	100%

In our study socio-economic status belongs to class II (40.5%) and class III (42.9%) most commonly involved in suicidal poisoning.



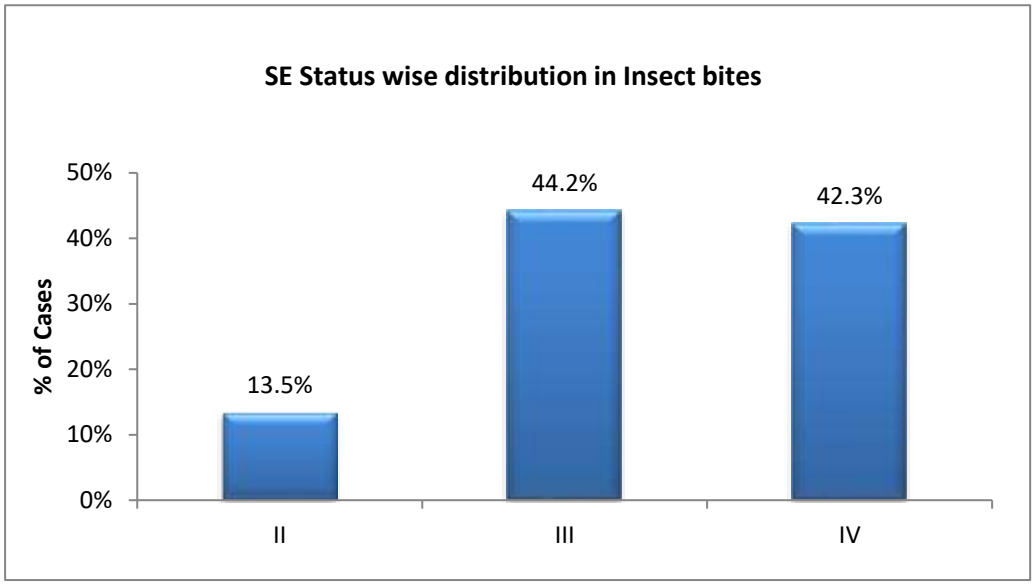
Graph – 22 : SE - Status Wise Distribution In Suicidal Poisoning

In insect bites:

Table – 25: SE - Status Wise Distribution In Insect Bites

Insect bites		
SE Status	**Frequency**	**%**
II	7	13.5%
III	23	44.2%
IV	22	42.3%
Total	52	100%

Most commonly involved SE status was class III (44.2%), followed by class IV (42.3%), which were lower middle and upper lower classes according to Kuppuswamy scale.



Graph – 23: SE - Status Wise Distribution In Insect Bites

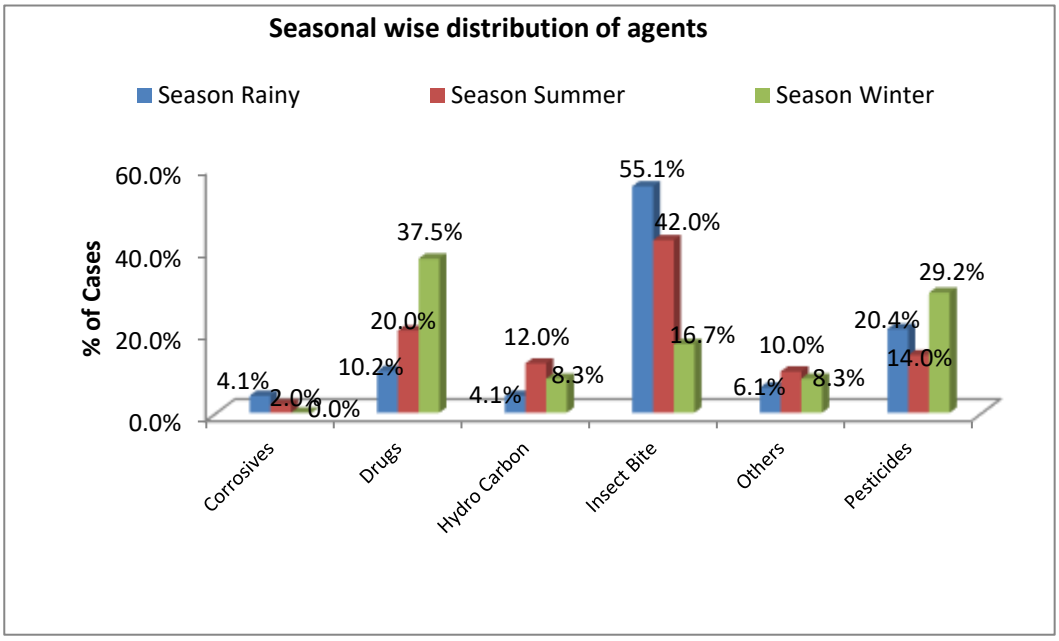
SEASONAL WISE DISTRIBUTION:

Agents involved in different seasons:

Table – 26: Seasonal Wise Distribution of Agents

Group	Total	Season			P Value
		Rainy	Summer	Winter	
		Frequency (%)	Frequency (%)	Frequency (%)	
Corrosives	3	2 (4.1%)	1 (2.0%)	0 (0.0%)	
Drugs	24	5 (10.2%)	10 (20.0%)	9 (37.5%)	
Hydro Carbon	10	2 (4.1%)	6 (12.0%)	2 (8.3%)	
Insect Bite	52	27(55.1%)	21 (42.0%)	4 (16.7%)	0.069
Others	24	3 (6.1%)	5 (10.0%)	2 (8.3%)	
Pesticides	10	10 (20.4%)	7 (14.0%)	7 (29.2%)	
Total	123	49 (100%)	50 (100%)	24 (100%)	

In rainy season insect bites (n=27) were more common followed by pesticides (n=10) which was 55.15 and 20.4% respectively. While in summer season again insect bites (n=21) were more common followed by drugs (n=10), pesticides (n=7) and hydrocarbons (n=6) making 42%, 20%, 14%, and 12% respectively. While in winter season drugs (n=9), pesticides (n=7) and insect bites (n=4) were more common making 37.5%, 27.2% and 16.7% respectively.



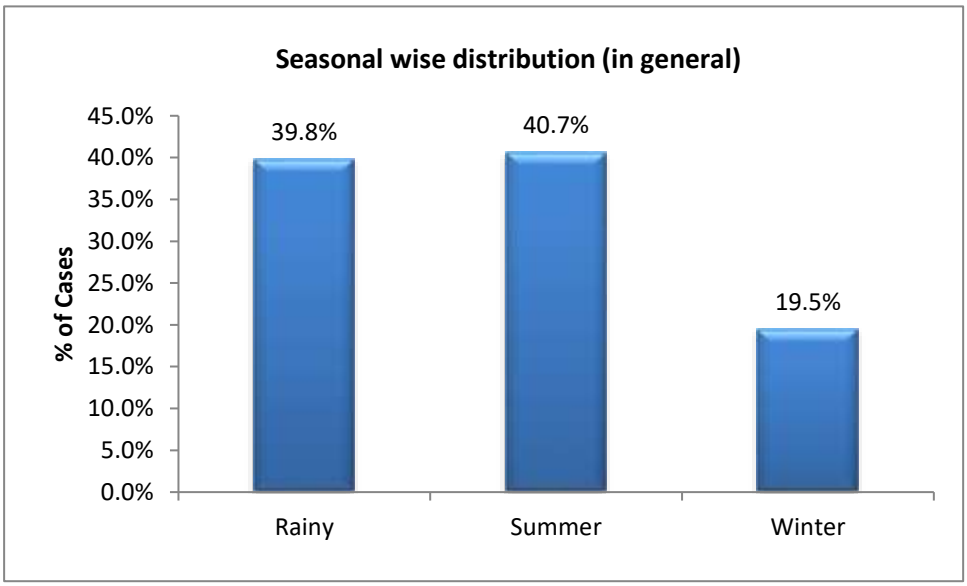
Graph – 24: Seasonal Wise Distribution of Agents

IN GENERAL:

TABLE – 27 : SEASONAL WISE DISTRIBUTION (IN GENERAL)

Season	Frequency	%
Rainy	49	39.8%
Summer	50	40.7%
Winter	24	19.5%
Total	123	100%

Most cases come in the summer season (40.7%) followed by in rainy season (39.8%) and least in winter season (19.5%).



Graph – 25: Seasonal Wise Distribution (In General)

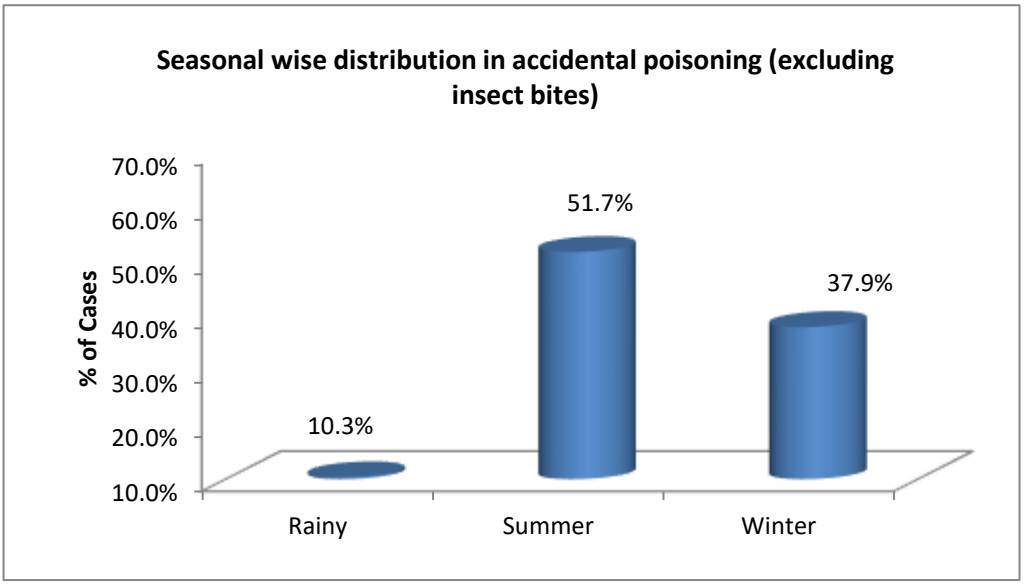
IN ACCIDENTAL POISONING AFTER EXCLUDING INSECT BITES:

Table – 28 : Seasonal Wise distribution in accidental poisoning

(Excluding insect bites)

Accidental poisoning		
Season	**Frequency**	**%**
Rainy	3	10.3%
Summer	15	51.7%
Winter	11	37.9%
Total	29	100%

In this study most of accidental cases come in summer season (51.7%) followed by winter (37.9%) followed by rainy season (10.3%).



Graph – 26 : Seasonal wise distribution in accidental poisoning

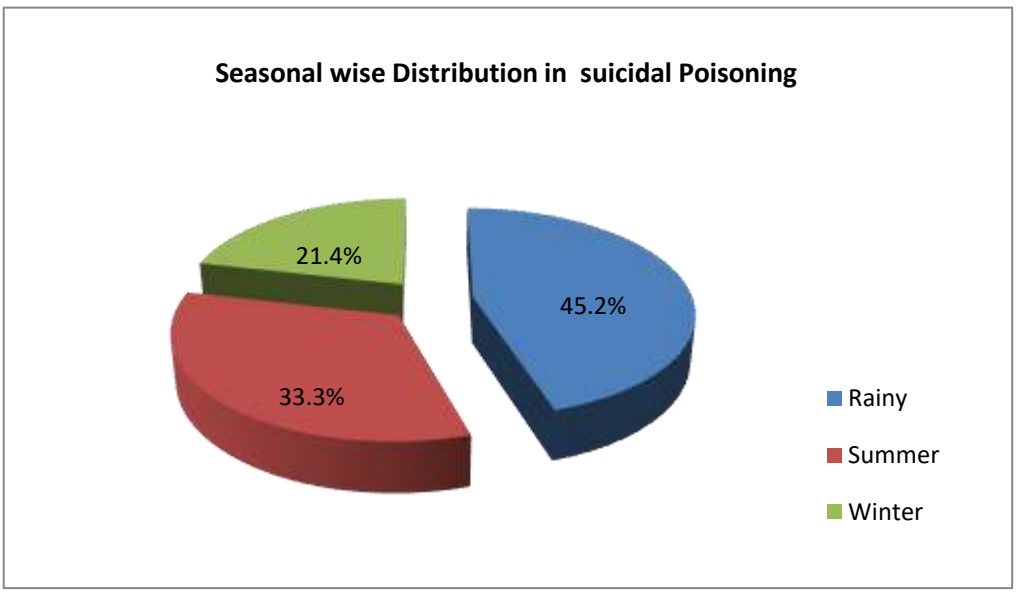
(Excluding insect bites)

IN SUICIDAL POISONING:

Table – 29 : Seasonal Wise Distribution in Suicidal Poisoning

Suicidal poisoning		
Season	**Frequency**	**%**
Rainy	19	45.2%
Summer	14	33.3%
Winter	9	21.4%
Total	42	100%

Suicidal poisoning was more commonly seen in rainy season (45.2%) and summer (33.3%) in this study.



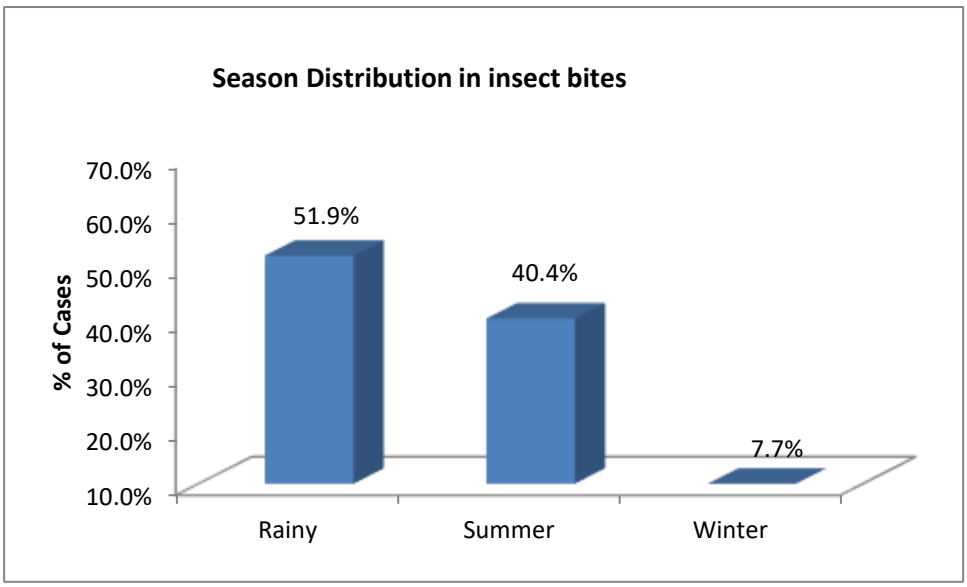
Graph – 27: Seasonal Wise Distribution in Suicidal Poisoning

IN INSECT BITES:

TABLE – 30 : SEASONAL WISE DISTRIBUTION IN INSECT BITES

Insect bites		
Season	**Frequency**	**%**
Rainy	27	51.9%
Summer	21	40.4%
Winter	4	7.7%
Total	52	100%

Insect bites were more common in rainy season (51.9%) followed by summer season (40.4%) and least in winter season (7.7%)



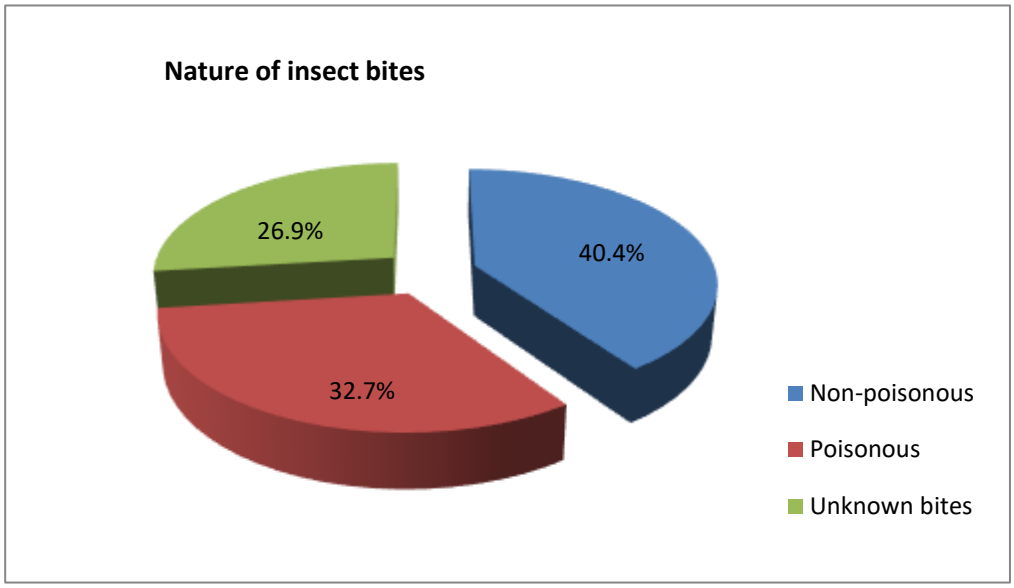
Graph – 28: Seasonal Wise Distribution in Insect Bites

NATURE OF THE INSECT BITES:

Table – 31: Nature of Insect Bites

Insect bites	Frequency	%
Non-poisonous	21	40.4%
Poisonous	17	32.7%
Unknown	14	26.9%
Total	52	100%

Among all insect bites non-poisonous were more common (n=21; 40.4%) than poisonous bites (n=17; 32.7%), rest were unknown bites (n=14; 26.9%).



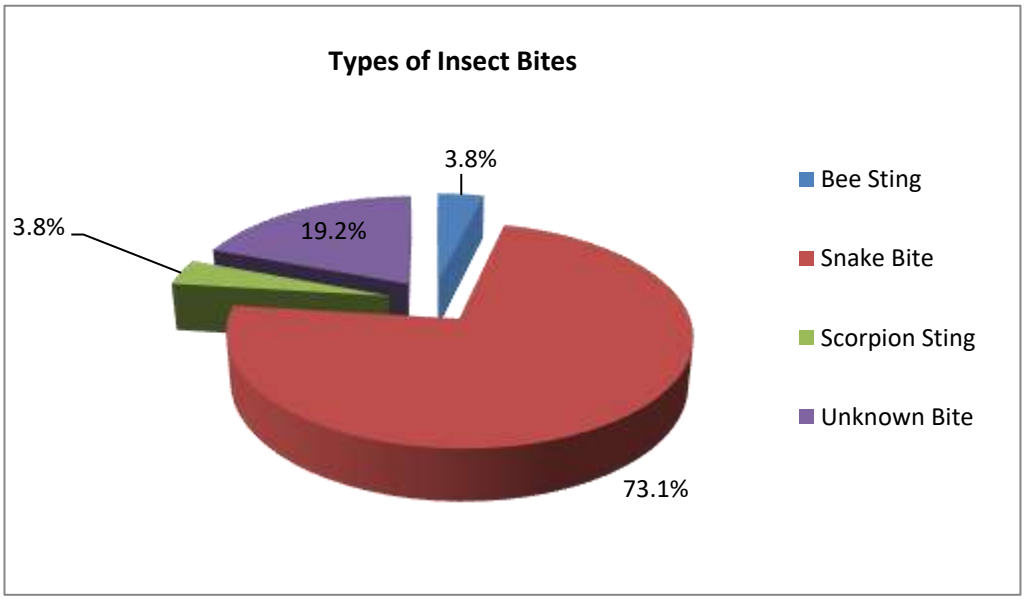
Graph – 29: Nature Of Insect Bites

TYPES OF INSECT BITES:

Table – 32: Types of Insect Bites

Insect Bite	Frequency	%
Bee Sting	2	3.8%
Snake Bite	38	73.1%
Scorpion Sting	2	3.8%
Unknown Bite	10	19.2%
Total	52	100%

In this study snake bites constitute 73.1%, unknown bites 19.2%, scorpion stings 3.8%, and bee stings caused 3.8% of total insect bites.



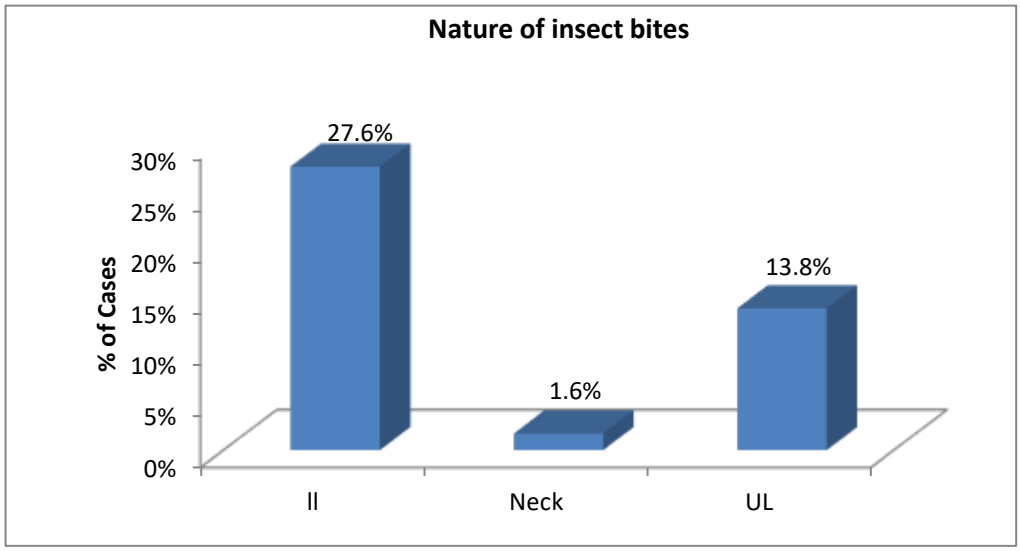
Graph – 30: Types of Insect Bites

SITE OF INSECT BITE:

Table – 33 : Nature of Insect Bites

Site of bite	Frequency	%
Lower limb	34	27.6%
Neck	2	1.6%
Upper limb	16	13.8%
Total	53	43%

Insect bites mostly on lower limbs (n=34) followed by upper limbs (n=16) and least in neck region (n=2). In neck region bites were caused by bee sting in one case and bite by unknown insect in second case.



Graph – 31 : Nature of Insect Bites

SUMMARY & CONCLUSION

Childhood poisoning is one of the important causes of the emergency department visits, which can be preventable with simple measures.

Though childhood poisoning is a worldwide problem there is significant reduction of morbidity mortality associated with it in developed countries by undertaking simple measures.

In developing countries like India though there is clear cut assumption of the problem, no significant reduction of the same in the last three decades, leading to increasing in emergency department visits with significant morbidity and mortality in children.

Although few studies were done, there is still lack of data from all regions of our country.

In the present study childhood poisoning was 5.45% of total pediatric emergency admissions in our hospital.

The most common childhood poisoning was due to insect bites followed by drug intake whether intentionally or unintentionally.

Children under 5yrs were more at risk for accidental poisoning with male predominance, mostly in the summer season most common agents involved were prescribed drugs and hydrocarbons.

While in older children and adolescents accidental poisoning due to insect bites and those due to intentional poisoning were more common, which can be largely preventable with simple measures.

Insect bites were more common from rural areas in the rainy season with slight male predominance. And also poisoning due to insecticides was more common in rural areas. Most of the cases belong to low socio-economic status.

In intentional poisoning drugs were more common in the urban areas with slight female predominance seen in the adolescent age group. The most common reasons was observed to be stress and psychological problems due to education related, family related, and financial related problems.

In insect bites childhood labor and poor socio economic status was common.

While in adolescents with intentional poisoning causes were due to low school performance and associated stress, family problems and financial problems were common. In most of the cases these were interrelated and difficult to separate.

DISCUSSION

MAGNITUDE OF POISONING:

In this study the incidence of poisoning was observed to be 5.45%. A study by Dar I et al, and an Indian authors of IAP text book Balasubramanian S et al based on Hospital data found that poisoning constitute 0.33%-7.6% of total hospital admissions[3,6]. A study by Mallesh K et al found that poisoning contributed 5.3% of total admissions[39]. And also according to world report on child injury by World Health Organization, 2008; India reported figures for fatal poisoning ranged between 0.65 and 11.6%[11]. So prevalence of our study was within range of the previous results. Another study by Brata GV et al found accidental poisoning formed 1.05% of all pediatric poisoning and it takes account only accidental cases of poisoning[30]. This wide range of variation in incidence due to under reporting, variation in study population and criteria they took into account.

Table 34: Studies on Magnitude of Poisoning

S. NO.	STUDY	RESULT
1	Present study	5.45%
2	Dar I et al,	0.33%-7.6%
3	IAP text book by Balasubramanian S et al,	0.33%-7.6%
4	Mallesh K et al,	5.3%
5	WHO	0.65 and 11.6%
6	Brata GV et al.	1.05%

Poisoning as over all:

In our study accidental poisoning (n=81) was more common than suicidal poisoning (n=42) which was 65.9% and 34.1% respectively. These were similar with studies done by Gupta SK et al[28], Azab SM et al[31], BachaT et al[32], Kohli U et al[33], Sharma J et al[34], Narayan P M et al[35], Manikyamba D et al[42], Halak V et al[47].

Table 35: Studies on Mode of Poisoning

Sl. No.	Study	Result
1	Present study	Accidental (65.9%)>suicidal (34.1%)
2	Gupta SK et al[28]	Accidental (79.9%)> suicidal (20.2%)
3	Azab SM et al[31]	Unintentional (68.5%)
4	Bacha T et al[32]	Unintentional (77.5%)> Intentional (15.5%)
5	Kohli U et al[33]	Accidental (96.9%)
6	Sharma J et al[34]	Accidental (92.8%)> Suicidal (7.8%)
7	Narayan PM et al[35]	Accidental (85.3%)
8	Manikyamba D et al[42]	Accidental (64%)
9	Halak V et al[47]	Accidental (98.59%)

Agents:

In this study overall poisoning by insect bites (n=52) were more common followed by drugs (n=24) and pesticides (n=24) which constituted 42.3%, 19.5% and 19.5% respectively. These were found similar in the study by Narayan PMet al[35], Rathore S et al[36].

A study by Manikyamba D et al[42] also reported snake bites were more common, and also they noticed poisoning by hydrocarbons were more common, this could be due to regional variation and high local usage of kerosene oil as fuel in that region.

In one study by Pratik VT et al[40] found that insecticides were more common followed by venomous bites, drug over dosage, kerosene oil consumption and corrosives, is in concordant with present study except venomous bites, the low incidence of venomous bites in this study could be due to under reporting.

In the present study while poisoning overall more commonly due to insect bites, whereas oral ingestion by agents causing accidental poisoning was more commonly due to drugs (n=14) followed by accidental poisoning by hydrocarbons (n=10) were 2nd most common.

In one study done by Gupta SK et al, at All India Institute of Medical Sciences found that household product was most common, followed by drugs and industrial chemicals and pesticides[28].

Another study by by Shoba Churi et al found that pesticides (n=111) followed by medicines(n=67) and house hold products(n=22) were most common[29]. This wide range of variation is due to factors like regional variation, different cultural customs and socio economic status of population, Local availability of these agents and their regular usage.

A study done by Brata GV et al found that although kerosene oil was most common, there was an increasing trend of poisoning with hydro chlorides (HCL) which is now most commonly used household agent(2nd) and decreasing trend of kerosene oil poisoning. Drugs constitute 3rd common agents[30]. This was reflected in our study as hydrocarbons like kerosene oil constituted as 2nd most common agent for accidental poisoning.

Table 36: Studies on Most Common Agents

Sl. No.	Study	Common agents	frequency
1	Present study	Insect bites, drugs and pesticides	42.3%,19.5%, 19.5%
2	Narayan PM et al[35]	Snake bites	27.7%
3	Rathore S et al[36].	Kerosene oil in summer Snake bites and op poisoning in rainy season	
4	Manikyamba D et al[42]	Bites and stings	44.26%
5	Pratik VT et al[40]	Insecticides > bites (16%)	
6	Gupta SK et al[28]	House hold products, drugs	47%, 21.8%
7	Shoba Churi et al[29]	Pesticides, medicines, household products.	N=111, 67, 22
8	Brata GV et[30]	Kerosene oil > HCL (17.3%)	

However, agents involved in suicidal poisoning was more commonly due to pesticides (n=23) 54.8%, drugs (n=10) 23.8%, others (n=6) 14.3% and corrosives (n=3) 7.1%. These were concordant with studies done by Azab SM et al[31], Bacha T et al[32].

Table 37: Studies on Agents Involved in Suicidal Poisoning

Sl. No.	Study	Common Agents (%)
1	Present study	Pesticides (54.8%), drugs (23.8%)
2	Azab SM et al[31]	Corrosives in preschool, pesticides in adolescents
3	Bacha T et al[32]	Drugs , Organophosphates

Age:

Poisoning overall

In this study most common age involved was 13-18yrs (n=70) constituted 56.9% of all poisoning cases, followed by 0-5yrs age group where it was 26% then 6-12yrs age group which was 17.1% of total poisoning cases. These results were similar to studies done by Sharma J et al[34], Annu J et al[38], Debata PK et al[44].

Accidental poisoning by oral ingestion

In this study most common age involved in accidental poisoning due to oral ingestion was 0-5 yrs (n=27) which is 93.1% of total accidental poisoning by oral ingestion. A study by Gupta SK et al[28], Brata GV et al[30], Kohli U et al[33], Sharma J et[34] al found that accidental poisoning was common in children aged less than 5 yrs, especially 18 months to 3yrs. Another study by Narayan PM et al[35], Rathore S et al[36] found that majority (43% and 62% respectively), were less than 5 years. In these studies insect bites also included in accidental poisoning reflecting low percentage poisoning in children aged less than 5yrs old than our study. A study by Annu J et al[38], Manikyamba D et al[42], Ram P et al[43],Basavaraj et al[46]found that accidental poisoning more common in children less than 5 yrs.

Suicidal poisoning

Most common age involved in suicidal poisoning was 13-18yrs (97.6%) of total suicidal poisoning. This was similar with studies done by Gupta SK et al[28], Azab SM et al[31], Bacha T et al[32], Sharma J et al[34], Narayan PM et al[35], Annu J et al[38].

Insect bites

In the present study an Insect bites were more common in the age group of 13 to 18 yrs (53.8%) followed by 6 to 12yrs (36.5%). This is concordant with the study by Kshirsagar VY[57] et al.

Table 38 : Studies on Age

Sl. No.	Study	Mode of poisoning	Age (%)
1	Present study	In general Accidental Suicidal Insect bites	13-18 yrs (56.9%) 0-5yrs (93.1%) 13-18yrs (97.6%) 13-18yrs (53.8%)
2	Sharma J et al[34]	- Accidental Intentional	11-18 yrs Below 5yrs Older children and adolescents
3	Annu J et al[38],	Accidental DSH	Median age 2yrs 16yrs
4	Debata PK et al[44].		11-19yr (74.5%)
5	Gupta SK et al[28],	Accidental Intentional	1-6yrs (79.9%) 12-18yrs (20.2%)
6	Brata GV et al[30]		1-5yrs (80.7%)
7	Kohli U et al[33],		1-3yrs

8	Narayan PM et al[35]	Accidental Suicidal	Below 5yrs (43%) After 11yrs (68%)
9	Rathore S et al[36]		Below 5 yrs (62%)
10	Manikyamba D et al[42],	Accidental	1-3yrs (64%)
11	Ram P et al[43],		1-5yrs (n=45)
12	Basavaraj et al[46]	Accidental	1-6yrs
13	Azab SM et al[31],	Accidental Suicidal	Below 6yrs (52%) After 12yrs (37%)
14	Bacha T et al[32]	Intentional	More than 5 yrs of age
15	Kshirsagar VY[57] et al.	Insect bites	More than 5 yrs

Sex:

Poisoning overall

In present study males (n=67, 54.5%) were more commonly involved than females (n=56, 45.5%) making ratio of 1.2:1. Which is in concordant with studies done by, Gupta SK et al[28], ChuriS et al[29],Sharma J et al[34], Narayan PM et al[35], Ram P et al[43], Kumar MR et al[45], Halak V et al[47],Ugwu GIM et al[41],and also with WHO world report on child injury prevention[11].

Accidental poisoning

In present study males were more commonly involved in accidental poisoning (n=17) which was 58.6% of total accidental poisoning. It is concordance with studies done by, Brata GV et al[30], Kohli U et al[33], Manikyamba D et al[42],Ram P et al[43].

Poisoning due to insect bites

In the present study accidental poisoning due to insect bites were more common among males (n=31, 59.6%) than females (n=21, 40.4%). It was found similar with studies by Mallesh K et al[39], Manikyamba D et al[42], Kshirsagar VY et al[57], Koirala DP et al[60].

Poisoning due to suicides

In the present study females (n=23) were more involved in suicidal poisoning than males(n=19), which was 54.8% and 45.2% respectively. This was concordant with studies by Bacha Tet al[32], Ram P et al[43].

Table 39 : Studies on Most Common Sex Involved

Sl. No.	Study	Mode of poisoning	Results (%)
1	Present study	In general Accidental Insect bites Suicidal	Males (n=67, 54.5%) > Females (n=56, 45.5%) Males (58.6%) Males (59.6%)>females(40.4%). Females (54.8%)<Males(45.2%).
2	Gupta SK et al[28]	-	Males (63.11%)>Females (36.88%)
3	Brata GV et al[30]	Accidental (1-5yrs)	Male> Female
4	Manikyamba D et al[42]	Accidental Insect bites	Male > female(1.56:1) Males>females
5	Ram P et al[43]	In general	Males(50.6%)>Females(49.4%) Males> females Females> males

		<5yrs 10-15yrs	
6	Kumar MR et al[45]		Males > females (2.5:1)
7	Halak V et al[47]		Males > females (1.17:1)
8	WHO		Boys> girls
9	ChuriS et al[29]	 Insect bites	Males > female (1.56:1) Males> females
10	Kohli U et al[33],	Accidental	Males > females
11	Sharma J et al[34].		Males>females (1.17:1)
12	Narayan PM et al[35],	In general Insect bites	Males(70%)
13	Ugwu GIM et al[41],		Males >females (2:1)
14	Mallesh K et al[39]	Insect bites	Males in 42 of 59 bites
15	Kshirsagar VY et al[57],	Insect bites	Males > females
16	Koirala DP et al[60].	Insect bites	Boys > girls
17	WHO		Boys >girls
18	Bacha T et al[32]	Suicides	Females (51.6%)> males

Socio- economic status:

Poisoning in general

In the present study most of the cases belongs to socio-economic status class III (n=55), followed by class IV (n=39) and class II (n=29) which was 44.7%, 31.7%, 23.6% respectively. In studies done by Sharma J et al[34], Manikyamba D et al[42] showing lower class more common. In this study more cases belong to upper lower and lower middle class, this variation could be due to our study area located in industrialized area and people comes from better SE-status than many other parts of country.

Present study showing accidental poisoning due to oral ingestion (48.3% from class III, 34.5% from class IV)and Insect bites(44.2% from class III, 42.3% from class IV)were also more common in the lower middle and upper lower class, whereas suicides(42.9% from class III, 40.5% from class II)showed that lower middle and upper middle classes were more involved. There was increasing trend towards higher socio-economic class. This was found to be similar with the studies by ChuriS et al[29], Manikyamba D et al[42]. It was also found to be similar with the **World report on child injury prevention** by **World Health Organization**[11].

Table 40: Studies on Socio-Economic Status

Sl. No.	**Study**	**Mode of poisoning**	**SE-status**
1	Present study	In general Accidental and insect bites Suicides	Lower middle (44.7%), upper lower (31.7%), upper middle (23.6%) Lower middle (48.3% & 44.2%), upper lower (34.5%&42.3%) Lower middle (42.9%), upper middle(40.5%)
2	Sharma J et al[34]	In general	Middle(20%) and low(75.7%)
3	Manikyamba D et al[42].		Lower (57%), upper lower (22%), lower middle (21%)
4	ChuriS et al[29],		Middle and lower SE-status
5	Ugwu GIM et al[41]	In General Drugs	Lower SE-class High income group
6	WHO[11]	Accidental (kerosene oil)	Lower to middle income countries (4 times)> high income countries

Seasonal wise distribution:

Poisoning in general:

In the present study more cases came in the summer and rainy season (n=50, and 49) making it 40.7% and 39.8% respectively. In rainy season most of cases due to snake bites (55.1%), and pesticides (20.4%). In summer season increasing cases were due to more accidental ingestion of hydrocarbons (12%), and accidental and suicidal ingestion by drugs (20%), along with insect bites (42%) and pesticides (14%). These were similar with studies done by RathoreS et al[36], Manikyamba D et al[42].

In accidental poisoning after excluding insect bites:

In the present study accidental poisoning more common in summer season(51.7%) followed by winter (37.9%) and rainy season (10.3%).

In suicidal poisoning:

In this study suicidal poisoning was more common in rainy season (45.2%) followed by summer season(33.3%). These were related to board exam conductance and results being declared (summer) and fresh start of new academic year (rainy season), reflecting stress related and psychological factors involved in these poisoning. This was in concordance with study done by Sharma J et al[34], Annu J et al[38].

In insect bites:

In this study insect bites were more common in rainy season (51.9%) followed by summer (40.4%) season, whereas least cases seen in winter season (7.7%). This was found similar with Kshirsagar VY et al[57].

Table 41 : Studies on Seasonal Variation

Sl. No.	Study	Mode of poisoning	Seasonal variation
1	Present study	In general insect bites accidental suicidal	Summer and rainy (40.7% & 39.8%) Rainy season (51.9%) Summer season (51.7%) Rainy season (45.2%)
2	Rathores et al[36]	Snake bites Op-Poisoning Kerosene oil	Rainy season Rainy season Summer season
3	Manikyamba D et al[42].	Accidental	Summer season
4	Annu J et al[38].	Suicidal	Psychiatric disorder
5	Sharma J et al[34]	Suicidal	Stress as a one of the factor
6	Kshirsagar VY et al[57]	Insect bites	Rainy season

Urban vs Rural:

In this study poisoning in general and accidental poisoning due to oral ingestion and suicidal poisoning was more common in urban population (56.1%, 65.5% and 59.5% respectively), this is similar with studies done by Kohli U et al[33], Rathore S et al[36], Basavaraj et al[46], Halak V et al[47]. While poisoning due to insect bites was more common in rural population (65.38%) which was in concordant with study by Manikyamba D et al[42]

Table 42 : Studies on Area Wise Distribution

Sl. No.	Study	Mode of poisoning	Area
1	Present study	Accidental Suicidal Insect bites	Urban> rural Urban > rural Rural > urban
2	Kohli U et al[33],	Accidental	Urban> rural
3	RathoreS et al[36]	Accidental	Urban(55%)>rural
4	Basavaraj et al[46]	Suicidal (52%)	Urban> rural
5	Halak V et al[47]	Accidental (98.59%)	Urban> rural
6	Manikyamba D et al[42].	Accidental Insect bites	Rural> urban Rural> urban

Poisoning due to insect bites:

In the present study 40.4% of cases were due to non-poisonous snake bites(n=21), while poisonous snake bites (n=17) were 32.7% and unknown bites (n=11), bites due to bee sting (n=1) and scorpion stings (n=2) were 26.9%. This was similar with the studies done by Manikyamba D et al[42], Vaiyapuri S et al[69].

Table 43 : Studies on Nature of Insect Bites

Sl. No.	Study	Poisonous/non-poisonous
1	Present study	Non poisonous (40.4%)>poisonous (32.7%)>unknown/ beesting/scorpion(26.9%)

2	Manikyamba D et al[42]	Non poisonous (68.1%)>poisonous(31.9%)
3	VaiyapuriS etal[69].	Majority were unknown origin

Most of the bites in lower limb (n=34, 65.4%) followed by upper limbs (n=16, 30.8%), and least in neck (n=2, 3.8%) reason these were caused by bee stings; this was found in similar with Kshirsagar VY et al[57].

Recommendations:

Although childhood poisoning was an important cause of emergency department visits there were very few studies with clear cut data available regarding causes and factors associated with it in India. It is recommended that there is need of further studies on childhood poisoning across all over India which will provide much needed data and emphasize preventable measures to help Government to make legislation to decrease the morbidity and mortality caused by childhood poisoning.

Childhood poisoning can be preventable with simple measures. Early detection and first aid measures at the site of poisoning need to be stressed.

Educating the Public and care givers to keep toxic substances out of reach of the children and making prescribed drugs and household products as child resistant packs can greatly reduce the accidental poisoning.

Making of common household products and agricultural based pesticides less toxic to humans can reduce the morbidity and mortality associated with it.

Wearing of shoes while playing and doing work on grounds and on fields can reduce snakebites and scorpion stings greatly. Similarly discouraging sleeping on floors especially in rainy seasons, strictly following and applying government legislations regarding child labor and human rights can reduce the number significantly.

More community-based studies are needed to provide precise data and reducing underreporting.

To prevent suicidal poisoning by training the teaching staff to identify high risk students and psychiatric consultation in at risk areas like schools and during periods of exams can reduce stress on students.

Early consultation of poison control centers can improve outcome in many severe cases so providing information regarding national poisoning control centers to primary health care providers and public in general is very important.

Making protocols for first aid and general measures, regarding common poisonings and their diagnosis and management, and proper circulation of these protocols to health care providers are also an important measure to reduce the problem. Making available of emergency medicines at primary health care centers will be useful.

Mass media such as television, radio and news papers should be harnessed for creating awareness about common poisons and their hazards and their prevention.

Regional poisoning control centers with well equipped laboratories to treat, guide and conduct research in the problem will greatly reduce and prevent the problem.

Poisoning control centers in India are;

National Poisons Information Centre (NPIC)

The National Poisons Information Centre (NPIC) was established in the Department of Pharmacology at AIIMS in 1995.

Welcome to the website of National Poisons Information Centre (NPIC) AIIMS, New Delhi, India.

Call 24 hrs a day, 7 days a week, 365 a year.

If you see someone has been poisoned, call NPIC on :

Toll Free No. - 1800 116 117

Tel No.- 26589391, 26593677

- Do not wait for the victim to look or feel sick.
- Do not treat the person yourself.
- Take the victim to nearby healthcare facility

All cases entered in accident register and Police information was given whenever necessary

REFERENCES

1. WHO/UNICEF: Children and poisoning. World report on child injury and prevention. Available from: http://www.who.int/violence_injury_prevention/child/injury/world_report/Poisoning_english.pdf. Accessed on 18, July 2015.
2. Kostic MA. Poisoning. In: Kliegman RM, Stanton BF, St Geme JW, Schor NF, Behrman RE, editor. Nelson Text book of Peditrics.20th edition.vol.1Philadelphia: Elsevier; 2016. p. 447-467.
3. Balasubramanian S, Jaydeep C. Intensive care and emergencies. Common poisonings in childhood. In: A Parthasarathy, editor. IAP Textbook of Pediatrics, 6th edition. New Delhi: Jaypee; 2016. P. 1032.
4. Dar I, Kamli M, Dar S, Wazir H. Unusual poisoning by oral ingestion of wild berries. The Internet Journal of Toxicology. 2008; 6(1): 1.
5. Justin S, Shobha C. Impact of Educational Intervention on Knowledge, Attitude and Practice among General Public Regarding Accidental Poisoning. Indian J o Pharm Practice.2014 mar 15; 7(1): 52.
6. Satish K V, Shivakumar P, Mandar R S, Sociodemographic profile of paediatric poisoning cases in Bangalore, India. Indian Journal of Forensic and Community Medicine, 2015;2(4):225-228.
7. Sahin S, Carman KB, Dinleyici EC. Acute Poisoning in Children; Data of a Pediatric Emergency Unit Iran J Pediatr. 2011dec; 21(4): 479–484.
8. Nowneet Kumar B, Minakshi D, Sohaib A, Vipan C. Profile of poisoning in children and adolescents at a North Indian tertiary care centre. JIACM. 2011; 13(1): 37-42. available from http://medind.nic.in/jac/t12/i1/jact12i1p37.pdf
9. Krishnakumar P, Geeta MG, Riyaz A. Deliberate self harm in children.Indian Pediatr. 2011 May;48(5):367-71.
10. Mrinal H, Mamata Devi H, Amarjyoti P. Death due to Poisoning in District of Kamrup, Assam A Medico-legal Study. J Indian Acad Forensic Med. 2013 jan;35(1):17. Available from http://medind.nic.in/jal/t13/i1/jalt13i1p17.pdf

11. WHO/UNICEF: World report on child injury and prevention 2008; chapter 6: p. 129. Available from: http://apps.who.int/iris/bitstream/10665/43851/1/ 9789241563574_ eng.pdf. Accessed on 16, September 2015.

12. Eddleston M.Patterns and problems of deliberate self-poisoning in the developing world.QJM. 2000 Nov; 93(11): 715-31. Available from http://www.ncbi.nlm.nih.gov/ pubmed/11077028

13. Sankar J, Nabeel R, Sankar MJ, Priyambada L, Mahadevan S. Factors affecting outcome in children with snake envenomation: a prospective observational study.Arch Dis Child. 2013 Aug;98(8):596-601.

14. Mohapatra B, Warrell DA, Suraweera W, Bhatia P, Dhingra N, Jotkar RM et al. Snakebite Mortality in India: A Nationally Representative Mortality Survey.PLoSNegl Trop Dis. 2011 Apr; 5(4): e1018.

15. Kasturiratne A, Wickremasinghe AR, de Silva N, Gunawardena NK, Pathmeswaran A, Premaratna R, et al. The global burden of snakebite: a literature analysis and modelling based on regional estimates of envenoming and deaths. PLoS Med. 2008nov;5:e218.

16. Gautam P, Sharma N, Sharma M, Choudhary S. Clinical and demographic profile of snake envenomation in Himachal Pradesh, India.Indian Pediatr. 2014 Nov;51(11):934-5.

17. Bhalla G, Mhaskar D, Agarwal A. A study of clinical profile of snake bite at a tertiary care centre.Toxicol Int. 2014 May;21(2):203-8.

18. Department of Health and Family Welfare, Ministry of Health and Family Welfare, Govt. of India: Health status indicators. National Health Profile(NHP) of India-2015; 3.2.114(A)(B): P 141-142. Available from: http://cbhidghs.nic.in/writereaddata/ mainlinkFile/NHP-2015.pdf. Accessed on 11,Aug 2015.

19. Markowitz G.The childhood lead poisoning epidemic in historical perspective. Endeavour. 2016 Jun;40(2):93-101.

20. Laios K, Tsoukalas G, Kontaxaki MI, Karamanou M, Androutsos G.Suicide in ancient Greece. Psychiatriki. 2014 Jul-Sep;25(3):200-7.

21. Reddenna L, Rama Krishna T. Management of poisoning: general protocol. International Journal of Pharmacology and Toxicology, 1 (2) (2013) 53-63. Available from:file:///C:/Users/home/Downloads/1366-4593-1-PB.pdf

22. Gella U, Shilpa N, Chandrababu S, Rathanshyam M, Venkatasubbiah M. A Prospective study on prevalence of poisoning cases- Focus on vasmol poisoning. International journal of pharmacy and pharmacoceutical sciences;2013: 5(4)

23. Bartrip P. A "Pennurth of arsenic for rat poison": the Arsenic Act, 1851 and the prevention of secrete poisoning. Med Hist. 1992 Jan; 36(1):53-69.

24. Jaswinder S, Jaspreet K, Vinod K, Shah. K.A, Tandon.R.N, Patil. V.R. Trends of Poisoning in a Tertiary Care Centre of North West Uttar Pradesh. J Indian Acad Forensic Med. 2015 oct-dec; 37 (4): 396. Available from: http://iafmonline.in/data/ publications/2015/JIAFM-37(4).pdf

25. Indu TH, Raja D, Ponnusankar S. Toxicoepidemiology of acute poisoning cases in a secondary care hospital in rural South India: A five-year analysis.J Postgrad Med. 2015; 61(3):159-62.

26. Singh SP, Aggarwal AD, Oberoi SS, Aggarwal KK, Thind AS, Bhullar DS et al. Study of poisoning trends in north India--a perspective in relation to world statistics(abstract).J Forensic Leg Med. 2013 Jan; 20(1): 14-8.

27. Mahalakshmy T, Dongre AR, Kalaiselvan G. Epidemiology of childhood injuries in rural Puducherry, South India.Indian J Pediatr. 2011 Jul;78(7):821-5.

28. Gupta SK, Peshin SS, Srivastava A, Kaleekal T.A study of childhood poisoning at National Poisons Information Centre, All India Institute of Medical Sciences, New Delhi. J Occup Health. 2003 May; 45(3): 191-6.

29. Churi S, Ramesh M, Bhakta K, Chris J. Prospective assessment of patterns, severity and clinical outcome of Indian poisoning incidents. Chem Pharm Bull (Tokyo). 2012; 60(7): 859-64.

30. Brata G V, Jhamb U, Singhal R, Krishnan R. Common childhood poisonings and their outcome in a tertiary care center in Delhi. Indian J Pediatr (abstract). 2013 Jun;80(6):516-8.

31. Azab SM, Hirshon JM, Hayes BD, El-Setouhy M, Smith GS, Sakr ML et al. Epidemiology of acute poisoning in children presenting to the poisoning treatment center at Ain Shams University in Cairo, Egypt, 2009-2013.Clin Toxicol (Phila). 2016; 54(1): 20-6.

32. BachaT, Tilahun B. A cross-sectional study of children with acute poisoning: A three-year retrospective analysis World J Emerg Med. 2015; 6(4): 265–269.

33. Kohli U, SreedharKuttiat V, Lodha R, Kabra S.K. Profile of Childhood Poisoning at a Tertiary Care Centre in North India. Indian journal of pediatrics; 2008 aug: 75.

34. Sharma J, Kaushal RK. Profile of poisoning in children. Pediatric oncall journal; 2014 apr-jun: 11(2). Available from http://www.pediatriconcall.com/Journal/Article/FullText.aspx?artid=828&type=J&tid=&imgid=&reportid=439&tbltype=

35. Narayan P M, Braja K D, Smita S, Anil K M. Trends of Acute poisoning cases in a tertiary care hospital in Odisha, India: a prospective study. IOSR Journal of Dental and Medical Sciences; 2014 nov: 13(11): 12-17. Available from: http://www.iosrjournals.org/iosr-jdms/papers/Vol13-issue11/Version-5/C0131151217.pdf

36. Rathore S, Verma AK, Pandey A, Kumar S. Pediatric Poisoning Trend in Lucknow district, India. J Forensic Res. 2013; 4(1): 1-3. Available from: http://www.omicsonline.org/pediatric-poisoning-trend-in-lucknow-district-india-2157-7145.1000179.php?aid=12784

37. Rukiye U S, Medine A T, İlknur B, Yurda Ş, Yıldız B D.Characteristics of Children with Acute Carbon Monoxide Poisoning in Ankara: A Single Centre Experience. J Korean Med Sci.2015 dec; 30(12): 1836-40.

38. Annu J, Sivanandam S, Matthai J. Poisoning in Children from an Educationally and Economically Advanced Urban Area of South India. Asian Journal of Epidemiology.2012; 5(4): 123-129. Available from http://scialert.net/fulltext/?doi=aje.2012.123.129&org=11

39. Mallesh K, Asha B, Anil K K, Rakesh S R. Spectrum of poisoning in children: Study from tertiary care hospital in South India. JEBMH. 2015 aug; 2(33): 4989-4999.

40. Pratik V T , Shankar M B , Manjunath S , Vikram P , Pradeep K G, Mahabalesh S. Trends of poisoning among children at Kasturba Hospital, Manipal. NUJHS. 2013 jun; 3(2): 25-28. Available from: http://nitte.edu.in/journal/june2013/TOPAC.pdf

41. Ugwu GIM, Okperi BO, Ugwu EN, Okolugbo NE. Childhood poisoning in Warri, Niger Delta, Nigeria: A ten year retrospective study. Afr J Prm Health Care Fam Med.

2012; 4(1): 1-5. Available from: http://www.phcfm.org/index.php/phcfm/article/viewFile/321/404

42. ManikyambaD, Madhavi N. Clinical Profile of Poisoning in Children Admitted in a Tertiary Care Centre. International Journal of Science and Research (IJSR). 2015 feb; 4(2): 975-978.Available from: http://www.ijsr.net/archive/v4i2/SUB151280.pdf
43. Ram P, Kanchan T, Unnikrishnan B. Pattern of acute poisonings in children below 15 years--a study from Mangalore, South India. J Forensic Leg Med. 2014 Jul; 25: 26-9.
44. Debata PK, Deswal S, Kumath M. Causes of unnatural deaths among children and adolescents in northern India - a qualitative analysis of postmortem data.J Forensic Leg Med. 2014 Aug; 26: 53-5.
45. Kumar MR, Kumar GP, Babu PR, Kumar SS, Subrahmanyam BV, Veeraprasad M et al.A retrospective analysis of acute organophosphorus poisoning cases admitted to the tertiary care teaching hospital in South India. Ann Afr Med. 2014 Apr-Jun; 13(2): 71-5.
46. Basavaraj, Pushpalatha K. Clinical profile and outcome of acute pediatric poisoning in urban tertiarycare hospital. JEBMH. 2015; 2 (5): p. 459-463.
47. Vasavada DH, Desai DP. Clinical Profile And Outcome Of Children Presenting With Poisoning (A Hospital Based Study). NJIRM. (2013), [cited June 23, 2016]; 4(4): 1-7. Available from: http://www.scopemed.org/?mno=41434
48. Warner M, Chen LH, Makuc DM, Anderson RN, Miniño AM. Drug poisoning deaths in the United States, 1980-2008. NCHS Data Brief. 2011 Dec; (81): 1-8.
49. Tyrrell EG, Orton E, Tata LJ. Changes in poisonings among adolescents in the UK between 1992 and 2012: a population based cohort study. Inj Prev. 2016 May 16.
50. Gururaj G. Injury prevention and care: an important public health agenda for health, survival and safety of children.Indian J Pediatr. 2013 Mar.
51. Patel V, Ramasundarahettige C, Vijayakumar L, Thakur JS, Gajalakshmi V, Gururaj G. Suicide mortality in India: a nationally representative survey.Lancet. 2012 Jun 23; 379(9834): 2343-51.
52. Singh SP, Aggarwal AD, Oberoi SS, Aggarwal KK, Thind AS, Bhullar DS et al. Study of poisoning trends in north India--a perspective in relation to world statistics.J Forensic Leg Med. 2013 Jan; 20(1): 14-8.

53. Sheriff A, Rahim A, Lailabi MP, Gopi J. Unintentional injuries among children admitted in a tertiary care hospital in North Kerala.Indian J Public Health. 2011 Apr-Jun; 55(2):125-7.

54. Venkatesh C, Sriram P, Adhisivam B, Mahadevan S. Clinical profile of children with kerosene aspiration. Trop Doct. 2011 Jul; 41(3): 179-80.

55. Girish KS, Kemparaju K. Overlooked issues of snakebite management: time for strategic approach. Curr Top Med Chem. 2011;11(20):2494-508.

56. World Health Organization 2010:Guidelines for the management of snake bites. chapter 5.7: p. 40-41. Available from: http://apps.searo.who.int/PDS_ DOCS/B4508.pdf. Accessed on 13, nov 2015.

57. Kshirsagar VY, Ahmed M, Colaco SM. Clinical profile of snake bite in children in rural India.Iran J Pediatr. 2013 Dec;23(6):632-6.

58. Onyiriuka N. Snake bite poisoning in childhood: Approach to diagnosis and management. Paediatrics Today 2012; 8(1): 11-21. Available from: http://www.paediatricstoday.com/index.php/pt/article/view/29

59. Mukherjee S. Characteristics of snake envenomation in eastern India, a study of epidemiology, complications and interventions. Int J Sci Rep. 2015 Aug;1(4):190-195. Available from: http://www.sci-rep.com/index.php/scirep/article/view/61

60. Koirala DP, Gauchan E, Basnet S, Adhikari S, BK G. Clinical Features, Management and Outcome of Snake Bite in Children in Manipal Teaching Hospital Nepal Journal of Medical sciences 2013 July-Dec; 2(2): 119-124. Available from: http://www.nepjol.info/index.php/NJMS/article/viewFile/8954/7351

61. Punde DP. Management of snake-bite in rural Maharashtra: a 10-year experience. Natl Med J India. 2005 Mar-Apr; 18(2): 71-5.

62. Narayan Reddy KS, Murty OP, General considerations. In: Narayan Reddy KS, Murty OP. The Essentials of Forensic Medicine and Toxicology. 33rd edition. New Delhi: Jaypee; 2014.p.498-519.

63. Pillay VV. Diagnosis of poisoning. In: Pillay VV. Modern medical toxicology.4thed. New Delhi: Jaypee; 2013. P 7.

64. Daly FF, Little M, Murray L. A risk assessment based approach to the management of acute poisoning. Emerg Med J. 2006 May; 23(5):396-9.

65. Gupta S, Taneja V. Poisoned child: emergency room management. Indian J Pediatr. 2003 Mar; 70Suppl 1:S2-8.

66. Ramasubbu B, James D, Scurr A, Sandilands EA.Serum alkalinisation is the cornerstone of treatment for amitriptyline poisoning. BMJ Case Rep. 2016 Apr 11;2016. Available from: http://casereports.bmj.com/content/2016/bcr-2016-214685.abstract

67. McGregor T, Parkar M, Rao S. Evaluation and management of common childhood poisonings.Am Fam Physician. 2009 Mar 1;79(5):397-403.

68. Calello DP, Henretig FM. Pediatric toxicology: specialized approach to the poisoned child.Emerg Med Clin North Am. 2014 Feb;32(1):29-52.

69. Vaiyapuri S, Vaiyapuri R, Ashokan R, Ramasamy K, Nattamaisundar K, Jeyaraj A et al. Snakebite and its socio-economic impact on the rural population of Tamil Nadu, India.PLoS One. 2013 Nov 21;8(11). Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3836953/

70. Rietjens SJ, de Lange DW, Donker DW, Meulenbelt J. Practical recommendations for calcium channel antagonist poisoning.Neth J Med. 2016 Feb;74 (2): 60-7.

71. Greenwald PW, Farmer BM,O'Neill M, Essner RA, Flomenbaum NE. Increasing frequency and fatality of poison control center reported exposures involving medication and multiple substances: data from reports of the American Association of Poison Control Centers 1984-2013.ClinToxicol (Phila). 2016 May 23:1-7.

72. Sakai N, Hirose Y, Sato N, Kondo D, Shimada Y, Hori Y. Late Metabolic Acidosis Caused by Renal Tubular Acidosis in Acute Salicylate Poisoning.Intern Med. 2016;55(10):1315-7.

73. Mark Little. Emergency management and resuscitation of poisoned patients: perspectives from "down under".Scand J Trauma ResuscEmerg Med. 2009; 17: 36.

74. Murray L, Daly F, Little M, Cadogan M. Aproach to the poisoned patient. In: Murray L, Daly F, Little M, Cadogan M. Toxicology Handbook. 2nd ed. Australia: Elsevier; 2011. P 2-30.

75. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. *Lancet*. 2008;371(9612):597-607.

76. World Health Organization (internet): Clinical management of acute pesticide intoxication: prevention of suicidal behaviours. Management of mental and brain disorders Department of mental health and substance abuse 2008: p 9-14. Available from: http://www.who.int/mental_health/prevention/suicide/pesticides_intoxication.pdf?ua=1. Accessed on 13, Jun 2015.

77. Barry JD. Diagnosis and management of the poisoned child. Pediatr Ann. 2005 Dec; 34(12):937-46.

78. Frithsen IL, Simpson WM Jr. Recognition and management of acute medication poisoning.Am Fam Physician. 2010 Feb 1;81(3):316-23.

79. Meyer S, Eddleston M, Bailey B, Desel H, Gottschling S, Gortner L. unintentional household poisoning in children. KlinPadiatr 2007; 219 (5): 254 – 270.

80. Shivbalan S, Sathiyasekeran M, Thomas K. Therapeutic misadventure with paracetamol in children.Indian J Pharmacol. 2010 Dec; 42(6):412-5.

81. Özkaya AK, Güler E, Karabel N, Namlı AR, Göksügür Y. Datura stramonium poisoning in a child.Turk J Pediatr. 2015 Jan-Feb;57(1):82-4.

82. Kraut JA, Approach to the Treatment of Methanol Intoxication.Am J Kidney Dis. 2016 May.

83. Gumber MR, Kute VB, Shah PR, Vanikar AV, Patel HV, Balwani MR et al. Successful treatment of severe iron intoxication with gastrointestinal decontamination, deferoxamine, and hemodialysis. Ren Fail. 2013; 35 (5): 729-31.

84. Jayashree M, Singhi S. Changing trends and predictors of outcome in patients with acutepoisoning admitted to the intensive care. J Trop Pediatr. 2011 Oct; 57 (5): 340-6.

85. Bonastre Blanco E, Domingo Garau A, Cols Roig M, PanzinoOcchiuzzo F, VilarEscrigas P. Swallowed iron Accidental iron poisoning.AnPediatr (Barc). 2010 Dec; 73 (6): 373-5.

86. St-Onge M, Dubé PA, Gosselin S, Guimont C, Godwin J, Archambault PM et al. Treatment for calcium channel blocker poisoning: a systematic review.ClinToxicol (Phila). 2014 Nov;52(9):926-44.

87. Woodward C, Pourmand A, Mazer-Amirshahi M. High dose insulin therapy, an evidence based approach to beta blocker/calcium channel blocker toxicity.Daru. 2014 Apr 8;22(1):36.

88. Müller D, Desel H. Common causes of poisoning: etiology, diagnosis and treatment. DtschArztebl Int. 2013 Oct;110(41):690-9.

89. Chan BS, Isbister GK, O Leary M, Chiew A, Buckley NA. Efficacy and effectiveness of anti-digoxin antibodies in chronic digoxinpoisonings from the DORA study (ATOM-1).ClinToxicol (Phila). 2016 Jul; 54(6): 488-94.

90. Smith SW. Drugs and pharmaceuticals: management of intoxication and antidotes.EXS. 2010;100:397-460.

91. Erdmann A, Werner D, Hugli O, Yersin B. Focused use of drug screening in overdose patients increases impact on management. Swiss Med Wkly. 2015 Dec 28;145. available from: http://www.smw.ch/content/smw-2015-14242/

92. Krieter P, Chiang N, Gyaw S, Skolnick P, Crystal R, Keegan F et al. Pharmacokinetic Properties and Human Use Characteristics of an FDA Approved Intranasal Naloxone Product for the Treatment of Opioid Overdose. J ClinPharmacol. 2016 May.

93. Feinstein DL, Akpa BS, Ayee MA, Boullerne AI, Braun D, Brodsky SVet al. The emerging threat of superwarfarins: history, detection, mechanisms, and counter measures.Ann N Y Acad Sci. 2016 May 31.

94. Freixo A, Lopes L, Carvalho M, Araujo F.Superwarfarine Poisoning.Acta Med Port. 2015 May-Jun; 28(3): 389-92.

95. King N, Tran MH. Long-Acting Anticoagulant Rodenticide (Superwarfarin) Poisoning: A Review of Its Historical Development, Epidemiology, and Clinical Management. Transfus Med Rev. 2015 Oct; 29(4): 250-8.

96. Schoen JC, Cain MR, Robinson JA, Schiltz BM, Mannenbach MS. Adolescent Presents With Altered Mental Status and Elevated Anion Gap After Suicide Attempt by Ethylene Glycol Ingestion. PediatrEmerg Care. 2016 Jan.

97. Megarbane B, Borron SW, Baud FJ. Current recommendations for treatment of severe toxic alcohol poisonings. Intensive Care Med. 2005 Feb; 31(2):189-95.

98. McMartin K, Jacobsen D, Hovda KE. Antidotes for poisoning by alcohols that form toxic metabolites.Br J ClinPharmacol. 2016 Mar;81(3):505-15.

99. American College of Medical Toxicology. Guidance Document: Management Priorities in Salicylate Toxicity. J Med Toxicol. 2015 Mar; 11(1): 149–152.

100. Dougherty PP, Lee SC, Lung D, Klein-Schwartz W. Evaluation of the use and safety of octreotide as antidotal therapy for sulfonylurea overdose in children. PediatrEmerg Care. 2013 Mar; 29(3):292-5.

101. Llamado R, Czaja A, Stence N, Davidson J. Continuous octreotide infusion for sulfonylurea-induced hypoglycemia in a toddler.J Emerg Med. 2013 Dec;45(6):e209-13.

102. Halesha BR, Harshavardhan L, Lokesh AJ, Channaveerappa PK, Venkatesh KB. A Study on the Clinico-Epidemiological Profile and the Outcome of Snake Bite Victims in a Tertiary Care Centre in Southern India. J ClinDiagn Res. 2013 Jan; 7(1): 122–126.

103. Alirol E, Sharma SK, Bawaskar HS,Kuch U, ChappuisF. Snake Bite in South Asia: A Review.PLoSNegl Trop Dis. 2010 Jan; 4(1). Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2811174/

104. Ariaratnam CA, Sheriff MH, Theakston RD, Warrell DA. Distinctive epidemiologic and clinical features of common krait (Bungaruscaeruleus) bites in Sri Lanka.Am J Trop Med Hyg. 2008 Sep; 79 (3): 458-62.

105. Herrera C, Macedo JK, Feoli A, Escalante T, Rucavado A, Gutiérrez JM et al. Muscle Tissue Damage Induced by the Venom of Bothrops asper: Identification of Early and Late Pathological Events through Proteomic Analysis.PLoSNegl Trop Dis. 2016 Apr 1; 10(4). Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4818029/

106. Takeda S. ADAM and ADAMTS Family Proteins and Snake Venom Metalloproteinases: A Structural Overview.Toxins (Basel). 2016 May; 8(5). Available from: http://www.mdpi.com/2072-6651/8/5/155

107. Trummal K, Tonismagi K, Aaspollu A, Siigur J, Siigur E.Viperalebetina venom nucleases.Toxicon. 2016 May 11;119:39-45.

108. Sunitha K, Hemshekhar M, Thushara RM, Santhosh MS, Sundaram MS, Kemparaju K et al. Inflammation and oxidative stress in viper bite: an insight within and beyond.Toxicon. 2015 May; 98: 89-97.

109. Golay V, Roychowdhary A, Pandey R, Singh A, Pasari A, Abraham A. Acute interstitial nephritis in patients with viperine snake bite: single center experience of a rare presentation.Saudi J Kidney Dis Transpl. 2012 Nov;23(6):1262-7.

110. Vinod KV, Dutta TK. Snakebite, dysautonomia and central nervous system signs.QJM. 2013 Sep; 106(9): 865-6.

111. Bawaskar HS, Bawaskar PH, Punde DP, Inamdar MK, Dongare RB, Bhoite RR. Profile of snakebite envenoming in rural Maharashtra, India.J Assoc Physicians India. 2008 Feb; 56: 88-95.

112. Tan CH, Tan NH, Tan KY, Kwong KO.Antivenom cross-neutralization of the venoms of Hydrophisschistosus and Hydrophiscurtus, two common sea snakes in Malaysian waters.Toxins (Basel). 2015 Feb 16; 7(2): 572-81.

113. Ahmed SM, Ahmed M, Nadeem A, Mahajan J, Choudhary A, Pal J. Emergency treatment of a snake bite Pearls from literature. Journal of Emergencies, Trauma Shock. 2008; 1(2): 97–105.

114. Directorate General of Health Services, Ministry of Health and Family Welfare, government of India: Snakebite treatment protocol: treatment phase. National Snakebite Management Protocol. 2009; p. 17-27. Available from: http://164.100.130.11:8091/nationalsnakebitemanagementprotocol.pdf. Accessed on 19, Oct 2015.

115. Abroug F, Souheil E, Ouanes I, Dachraoui F, Fekih-Hassen M, OuanesBesbes L. Scorpion-related cardiomyopathy: Clinical characteristics, pathophysiology, and treatment.ClinToxicol (Phila). 2015 Jul; 53(6):511-8.

116. Chippaux JP, Goyffon M. Epidemiology of scorpionism: a global appraisal. Acta Trop. 2008 Aug; 107(2):71-9.

117. Kumar PM, Krishnamurthy S, Srinivasaraghavan R, Mahadevan S, Harichandrakumar KT. Predictors of Myocardial Dysfunction in Children with Indian Red Scorpion (Mesobuthus tamulus) Sting Envenomation. Indian Pediatr. 2015 Apr; 52(4):297-301.

118. Agrawal A, Kumar A, Consul S, Yadav A.Scorpion bite, a sting to the heart. Indian J Crit Care Med. 2015 Apr;19(4):233-6.

119. Bawaskar HS, BawaskarPH.Scorpion envenoming,a step ahead.Indian Pediatr. 2015 Apr;52(4):289-90.

120. Natu VS, Kamerkar SB, Geeta K, Vidya K, Natu V, Sane S et al. Efficacy of anti-scorpion venom serum over prazosin in the management of severe scorpion envenomation. J Postgrad Med. 2010;56(4):275-80.

121. Krishnamurthy S, Mahadevan S. Efficacy of scorpion antivenom in children.Indian Pediatr. 2014 Jun; 51(6):499-500.

122. Rana SK, Nanda C, Singh R, Kumar S. Management of Snake Bite in India-Revisited. Jk Science. 2015; 17(1): 3-6. Available from : http://www.jkscience.org/archives/2-Re