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Quantitative Risk Assessment in Toxicology Methodology and Application

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ABSTRACT

Quantitative Risk Assessment (QRA) is a systematic approach to evaluating and quantifying risks associated with complex systems, processes, or environments. It integrates statistical analysis, mathematical modelling, and probabilistic techniques to estimate the likelihood and potential consequences of adverse events. Poison is defined as the substance or Natural or a Man – made origin that through its chemical action usually kills, injures or impairs the living organism, if ingested , breathed in, injected in to the body or absorbed through the skin. The objective of QRA is to provide decision-makers with actionable insights to minimize risks, enhance safety, and optimize resource allocation. This methodology is widely applied across industries, including energy, manufacturing, transportation, and healthcare, to address uncertainties and ensure regulatory compliance. By leveraging data-driven models, QRA enables the identification of high-risk scenarios, the prioritization of mitigation strategies, and the development of contingency plans to safeguard operations and public welfare. These abstract outlines the core principles, methodologies, and applications of QRA, emphasizing its role in proactive risk management and resilience building. The assessment was conducted for 1 year with 918 population and this data is extracted from the Government General Hospitals of Guntur and Tenali and also from Retrospective data which is designed according to the WHO and Questionaries collected from studies of Authors. The result obtained from the Assessment was that the pesticides poison source is common among age groups of 20 – 40 years of males due to marital discord. This study cherishes about the risk analysis within the different category of individuals after consumption of poison.

Key words: Quantitative, Toxicological, Decision making, Consequence Analysis, Risk Assessment, Sensitivity Analysis, Risk Modelling, Hazardous, (EMP) Emergency Mitigation Plan.

INTRODUCTION

It is a systematic method used to identify and understand the hazards and risk associated with natural gas exploration and handling. This section focuses on analysing the major risks involved in converting 37 exploratory wells into developmental wells. The QRA process provides a rational evaluation of these risks, outlining preventive and mitigation measures. The results can assist in project planning and decision-making, particularly for emergency situations such as blowouts, and help develop an emergency response plan (ERP) to minimize damage to personnel, **Risk study for onshore driving activities.** The study covers all aspects of drilling rig operations and related activities during the exploration phase. Major hazards include loss of well control, blowouts, and process leaks.

Objectives of the QRA Study

Analyse the likelihood and frequency of risk scenarios via historical accident data from the oil and gas industry. Predict the consequences of potential risk scenarios, confirming high- consequence events through quantitative simulations. Recommend preventive and risk mitigation measures and provide inputs for an Emergency Management Plan (EMP).

Risk Assessment Methodology

The risk assessment is based on the likelihood of risks and their consequences evaluated through hypothetical accident scenarios. Major risks such as blowouts, process leaks, and fires are assessed via a risk matrix, that combines risk severity and likelihood. Risks are ranked semi quantitatively, resulting in classifications of high, medium, low, or very low, guiding appropriate mitigation strategies [1].

Hazard identification

Hazard identification includes a review of project-related information and guidance from industry platforms such as OGP, ITOPF, EGIG, and DNV. Key hazards include process leaks, fires, and non-process explosions.[4][8]

Frequency Analysis

Frequency analysis estimates the likelihood of failure cases identified during hazard identification. This analysis uses historical accident frequency data, event tree analysis, and judgemental evaluation to determine the probability of each hazard's occurrence.[6]

Consequence Analysis

Consequence analysis evaluates the impact of accidents on personnel, infrastructure, and the environment. The analysis considers aspects such as environmental and community impact, occupational health and safety, property damage, corporate image, and restoration timelines and costs. [3]

Risk evaluation

Risk significance is calculated as the product of likelihood and consequence. Risks are classified as high, medium, low, or very low, guiding necessary actions and mitigation measures.

Risk Assessment for Identical Hazards

Two major risk categories are associated with exploratory drilling activities: process leaks and fires and non-process fires/explosions. A comprehensive risk assessment was used to evaluate these risks, considering the high percentage of methane in natural gas for jet fires and explosion modelling.[2]

Simulation Scenarios and Consequences

The consequences of fire/heat waves and overpressure events are analysed for their impact on equipment and people. Radiation intensity and overpressure damage are quantified to assess potential injuries and structural damage.

Weather Conditions for Consequence Analysis

The weather conditions considered include day and night temperatures, the wind speed, and the stability class, which ingested, inhaled, injected, or absorbed through the skin in sufficient quantities, can cause illness or death.

Similarly, the Greek word "Toxicon", meaning poison, conveys the same concept. Essentially, a poison is any harmful substance to the body under these conditions. Every substance can become poisonous if it is consumed in large enough quantities.[5]

Poisoning

Poisoning is a significant health issue worldwide, arising from various sources, such as occupational exposure to industrial chemicals and pesticides, accidental or intentional contact with household or pharmaceutical products, and poisoning from venomous animals, toxic plants, and food contamination.[29]

Organophosphate poisoning:

Organophosphate poisoning is one of the most common types of poisoning due to the high availability of these compounds. Among the organophosphorus compounds, methyl parathion (metacid) is the most frequently used, followed by dichlorovos (nuvan). This poisoning is particularly prevalent in southern India. Where farmers, who make up a significant portion of the population. Commonly use organophosphorus compounds such as parathion as Insecticides. The accessibility of these compounds has led to a high number of suicide cases in this region.[23]

Toxicity of organophosphate compounds

- Highly toxic compounds: endosulphan, methyl parathion, malathion, oleander seeds (in high doses). Datura, zinc sulfide and phosphide.
- Less toxic compound: Gammahexene, pyrethroids and superwarfarin.[35]

Mechanism of organophosphates

Organophosphate Compound Cause poisoning through irreversible inhibition of the enzyme acetylcholinesterase (AChE) by phosphorylating its active state. This inhibition leads to the accumulation of acetylcholine, resulting in overactivation of cholinergic receptors at neuromuscular junctions and within the autonomic and central nervous system. The extent and speed of AChE inhibition vary depending on the structure of the organophosphate compounds and their metabolites.[26]

Generally, parathion compounds in their original form are not significant AcheE inhibitors. They require metabolic activation(oxidation) in vivo to convert to their active Oxon forms. For example, parathion must be metabolized into paraoxon in the body to effectively inhibit AChE. The toxic mechanism of organophosphate pesticides differs from that of a carbamates which inhibits the same enzyme reversibility and are sometimes used as medicines(example neostigmine pyridostigmine) or insecticides (E.g.. Carbaryl).[34]

After the initial inhibition and formation of the acetylcholinesterase organophosphate (AChE-OP) complex, to further reactions can occur:[40]

- **Spontaneous reactivation:** The enzyme may slowly reactive itself, but this process is much slower than the initial inhibition, taking hours to days. The rate of this regeneration depends on the type of OP compound. Generally, AChE dimethyl OP complexes reactive spontaneously in less than one day. whereas AChE diethyl OP complexes may take several days. During this period, reinhibition of the newly activated enzymes can occur.
- **Ageing**: The enzyme- OP complex progressively loses an alkyl group over time, rendering it unresponsive to reactivating agents. This process, known as ageing depends on factors such as pH, temperature,, and the type of OP compounds. Dimethyl OPs have an aging half-life of 3.7 yrs. Where diethyl OPs have an aging half-life of 3.3 slower the spontaneous reactivation is the greater the quantity of inactive AChE available for ageing.

To counteract this, nucleophilic reagent such as oximes can hasten reactivation by liberating more active enzymes, thus acting as antidotes in Organophosphate poisoning. Since ageing occurs more rapidly with dimethyl OPs. Oximes are theoretically most effective within the first 12 hours of poisoning. By catalyzing the regeneration of active AChE from the enzyme-OP complex oxime reduce the quantity of inactive AChE availability for ageing. However, in the case of diethyl OP intoxication, oximes may remain effective for several days.

A mode of toxic action refers to a common set of physiological and behavioral signs that characterize a specific type of adverse biological response. This should not be confused with the mechanism of action, which pertains to a biochemical process underlying a given mode of action.

Modes of toxic action are important and widely use tools in ectotoxicology and aquatic toxicology because they classify toxicants according to their type of toxic action. There are two major types of modes of toxic action: Nonspecific acting toxicants and specific acting toxicants. Nonspecifically acting toxicants produce narcosis, whereas specific acting toxicants are non-narcotic and exert specific action at a specific target site

Specific drug levels, as discussed below, may be helpful in confirming the diagnosis and in making management decisions. [37]

Mechanism of toxicity

There are several specific modes of toxic action

- **Acetylcholinesterase (AChE) inhibitors:** AChE is an enzyme found in nerve synapses that regulates nerve impulses by breaking down the neurotransmitter acetylcholine (Ach) when toxicants bind to AChE they inhibit the breakdown of Ache resulting in continuous nerve impulses across synapses. This prolonged signaling can eventually cause nervous system damage. Examples of AChE inhibitors include organophosphates and carbamates, which are commonly found in pesticides.
- **Irritant:** These chemicals have an inflammatory effect on living tissue through chemical action at the site of contact. The effect of irritants is an increase in the volume of cells due to hypertrophy (an increase in cell size) or hyperplasia(an increase in number of cells). Examples of irritants include benzaldehyde, acrolein, zinc sulfate and chlorides.[15]

Pesticides are classified on the basis of various criteria such as the following

- Toxicity (hazardous effects)
- Best organism they kill and pesticide function
- Chemical composition
- Mode of entry
- Mode of action
- How or when they work
- Formulations and sources of origin

Classification of pesticides on the basis of toxicity

The toxicity of pesticides mainly depends on two factors namely dose and time. Hence how much of the substance is involved(dose)and how often the exposure to the substances (time) give rise to two different types of toxicity acute and chronic.[24]

Acute toxicity:

Acute toxicity refers to how poisonous a pesticide is to a human, Animal or plant after a single short-term exposure. A pesticide with high acute toxicity is deadly even when a very small amount is absorbed. Acute toxicity may be measured as acute oral toxicity, acute dermal toxicity or acute inhalation toxicity.

Chronic toxicity:

Chronic toxicity is a delayed poisonous effect from exposure to a pesticide. The chronic toxicity of pesticides concerns the general public as well as those working directly with pesticides because of potential exposure to pesticides on or in food products, water in the air.

WHO class	LD 50 for Human being (Mg/kg body wt.)	Examples	US.F.D. value
	Oral Dermal		
Extremely Hazardous	<5 < 50	Parathion, Dieldrin, Phorate	700-3500 mg 3.5-14 mg
Highly hazardous	5-50 50 – 200	Aldrin, Dichlorvo	350-35000gms 700-3500 mg
Moderately hazardous	50-2000 200-2000	DDT, Chlordan	21-35 mg
Slightly hazardous	Over2000 Over 2000	Malathion	7-35gms
Unlikely to present acute hazard	5000 or higher	Carbetamide, Cyclosporin	70-350 gms 3.5-14gms

- **Classification of Pesticides the Perspective of Forensic Medicine and Toxicology**
  - Pesticides may be described as physical, chemical or biological that kill an undesirable or troublesome animal, plant or microorganism. Pesticides are generic names for a variety of agents that may be classified more specifically on the basis of patterns of use and organism death. Pesticides Insecticides.
  - Compounds that kill or repel insects and release species
  - Eg: organophosphates, organochlorine, carbamates.[27]
- **Herbicides**

– Compounds that kill weeds or prevent the growth of undesirable herbs or weeds in the field. e. g. parquet, atrazine etc.

- **Fungicides**
  - Compounds that kill fungi and moulds. e. g. Captan, Captofol, etc. Rodenticides – Compounds which kill rats, mice, moles and other rodents. For example, anticoagulants, arsenic, and strychnine are used.
- **Acaricides**
  - Compounds that kill mites, ticks and spiders. e. g. azobenzene, chlorobenzene, etc., Nematicides – Compounds that kill nematodes. e.g. Ethylene bromide Molluscicides – Compounds which kill the molluscs such as snails and slugs. e. g. Metaldehyde. Miscellaneous Pesticides – Compounds of lead, copper, mercury, nicotine, etc. Eg. azobenzene, chlorobenzene etc.
- **Nematicides**
  - Compounds that kill nematodes. e.g., Ethylene bromide
- **Molluscicides**
  - Compounds that kill mollusc such as snails and slugs. e. g. Metaldehyde.
- **Miscellaneous Pesticides**
  - Compounds such as lead, copper, mercury, and nicotine.
- **Classification on the basis of sources of origin :** Pesticides are natural or biological agents that are used to kill unwanted plants or animal pests.

pesticides may be classified into biopesticides and chemical pesticides on the basis of their source. Biopesticides act on target pests and strongly linked organisms. Biopesticides include microbial pesticides (containing a live bacterium, fungus, virus, protozoan or alga as the active ingredient), and chemicals derived from animals, bacteria, fungi and plants. They are less toxic, decompose easily and are required in small quantities. There are three major classes of biopesticides.

Microbial pesticides

The active ingredient is microorganisms such as bacteria or fungi. For example, bacterial toxins produced by *Bacillus thuringiensis*, and Bacillus sphaerius act on mosquito larvae and black fly larvae.

Plant incorporated protectants

Pesticides produced naturally by plants and genetic material introduced together are termed plant--incorporated protectants. Biochemical pesticides-pesticides, which include natural materials that have nontoxic mechanism to control pests. Eg: Insect sex pheromones

Chemical pesticides-

These wide ranges affect a large group of non-target organisms. Chemical pesticides are quite toxic and not always biodegradable. Chemical pesticides are further divided into carbamates and pyrethroids. Organochlorine, organophosphate, Table 3: Pesticide classification on the basis of the pest organism they kill and pesticide function are called modes of entry.

Pesticide classification on the basis of the pest organism they kill and pesticide function are called modes of entry.

Classification of pesticides on the basis of mode of action

Pesticides are also classified according to their mode of action which is shown in Table 5.

Pesticide formulations are mixtures of technical grade pesticides with inert diluents and auxiliary chemicals. Pesticide formulations can be divided into three types: solids. Liquids and gases. Some formulations are ready to use whereas others must be mixed before use.

Classification of pesticides on the basis of mode of action:

S.no	Type of pesticide	Mode of action	Example	UFD (usual fatal dose)
1.	Physical poison	Exertion of physical effect	Activated clay	100 mg /kg
2.	Protoplasmic poison	Precipitation of protein	arsenicals	70-180 mg/kg
3.	Respiratory poison	Inactivates the respiratory enzymes	Hydrogen cyanide	50 mg/kg
4.	Nerve poison	Inhibits impulse conduction	malathion	100 mg/kg

Table 5: Classification according to mode of action

CLASSIFICATION

Acid and alkaline poisoning: Caustics and corrosives cause tissue injury via a chemical reaction. The vast majority of caustic chemicals are acidic or alkaline substances that damage tissue by accepting a proton (alkaline substance) or donating a proton (acidic substance) in an aqueous solution. Toilet bowl-cleaning. Soldering flux containing zinc chloride, Swimming pool-cleaning products, Cement –cleaning products [.32]

DEFINITION: A corrosive substance is a substance that fixes, destroys and erodes the surface with which it comes into contact.

CLASSIFICATION OF CORROSIVE POISON:

Types	**Examples**	**UFD (usual fatal dose)**
Mineral salts	Sulphuric acid Nitric acid Hydrochloric acid	30ml 10-15ml 2-5ml
Organic acids	Oxalic acid Carboxylic acid Acetic acid Salicylic acid	15-20g 10-15ml 5-10ml 150 mg/kg
Vegetable acids	Hydrocyanic acid	0.5 mg/kg
Alkalis	Caustic soda Ammonium hydroxide	5-10ml 5-10ml

METHODS AND MATERIALS

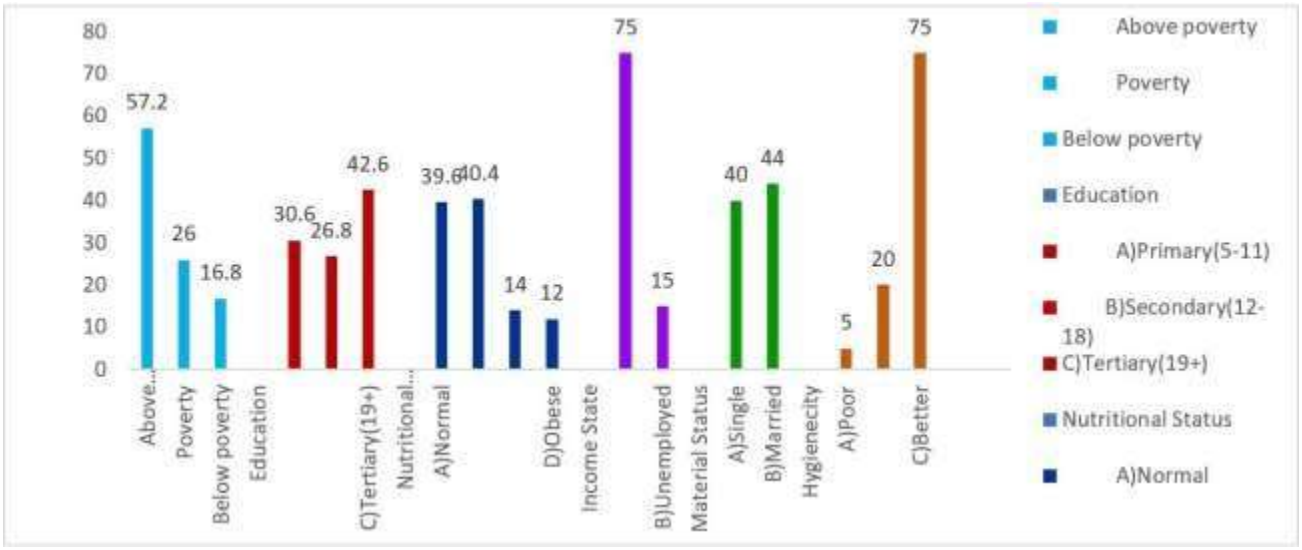
Study Site:

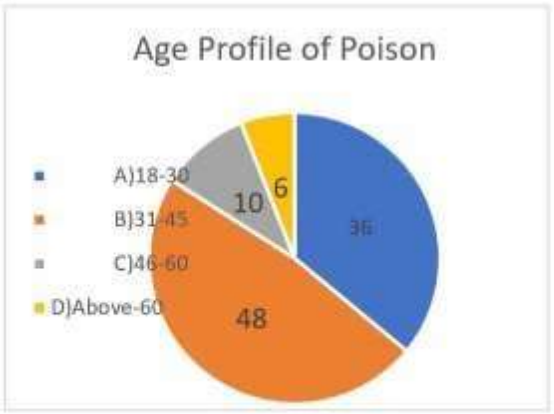
Government general hospital of Tenali and Guntur, Retrospective data.

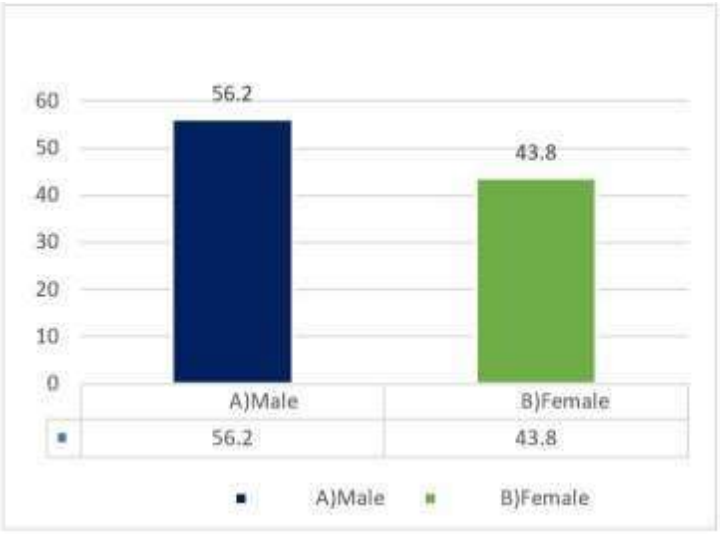
- **Study Duration:** 1Year
- **Study Population:** Total population 917
- **Study Design:** A well design questionnaires preparation according to WHO and various studies of different Authors
- **Study Criteria**
  - Inclusion
  - Exclusion
- **Inclusion**
  - Patients who are willing to provide information
  - Age (18-60) are included
  - Who are suffered with poison admitted
  - Who consumed poison admitted
- **Exclusion**
  - Paediatrics
  - Lactating women
  - Who are not providing information
  - Age above 60
- **Study procedure**
  - Data collected from the patient by using questionaries , data were evaluated and entered into MS excel sheet and prepared results from the study.

Demographics	No. of patients	Percentage%
Age		
18-30	180	36
31-45	240	48
46-60	50	10
Above-60	30	6
Sex		
Male	281	56.2
Female	219	43.8
Family history		
Above poverty	288	57.2
Poverty	128	26
Below poverty	84	16.8
Education		
Primary (5-11)	153	30.6
Secondary (12-18)	134	26.8
Tertiary (19+)	213	42.6
Nutritional Status		
Normal	198	39.6
Malnourished	202	40.4
Overweight	70	14
D)Obese	60	12
Income State		
Employed	375	75
Unemployed	75	15
Material Status		
Single	220	40
Married	279	44
Hygiene city		
Poor	25	5
Average	100	20
Better	375	75

Table1: Shows demographics details of the patient consumed poison and this data collected from questionaries.



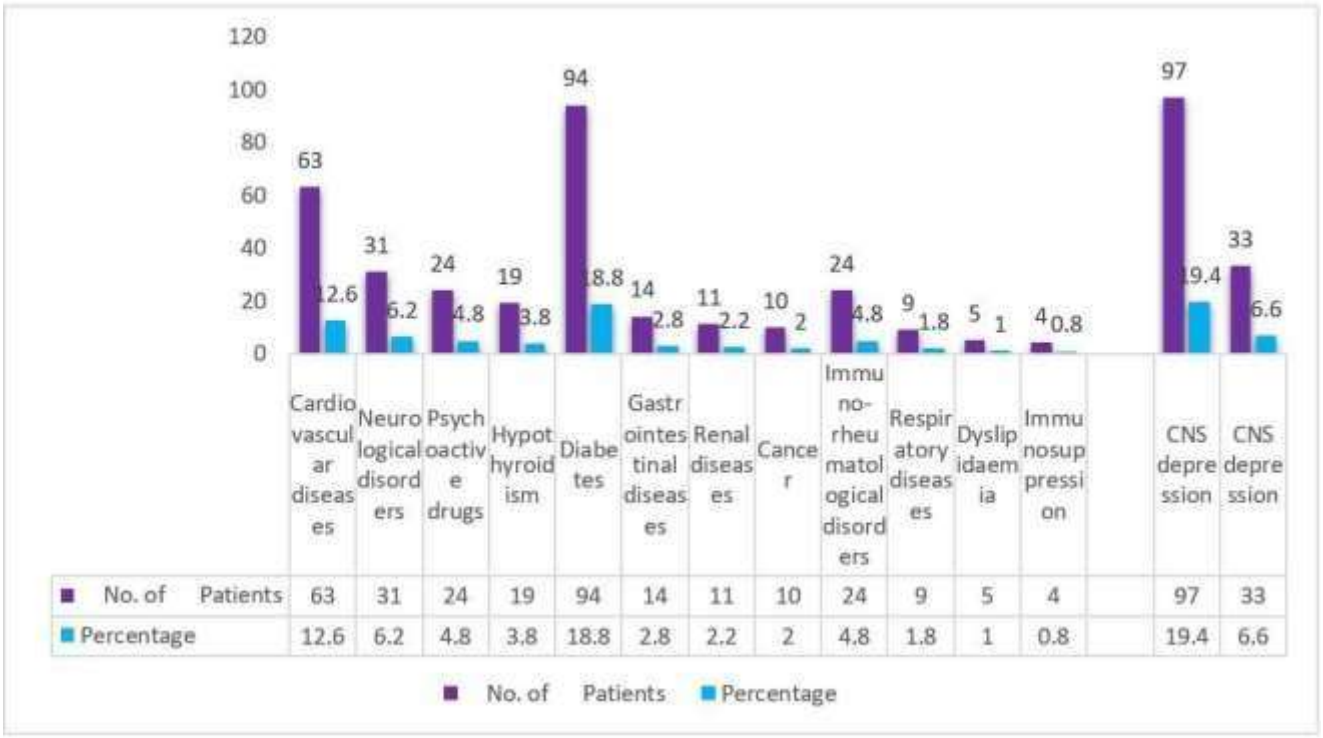




COMPLICATIONS	No. of Patients	Percentage
Cardiovascular diseases	63	12.6
Neurological disorders	31	6.2
Psychoactive drugs	24	4.8
Hypothyroidism	19	3.8
Diabetes	94	18.8
Gastrointestinal diseases	14	2.8
Renal diseases	11	2.2
Cancer	10	2
Immuno-rheumatological	24	4.8
disorders	9	1.8
Respiratory diseases	5	1

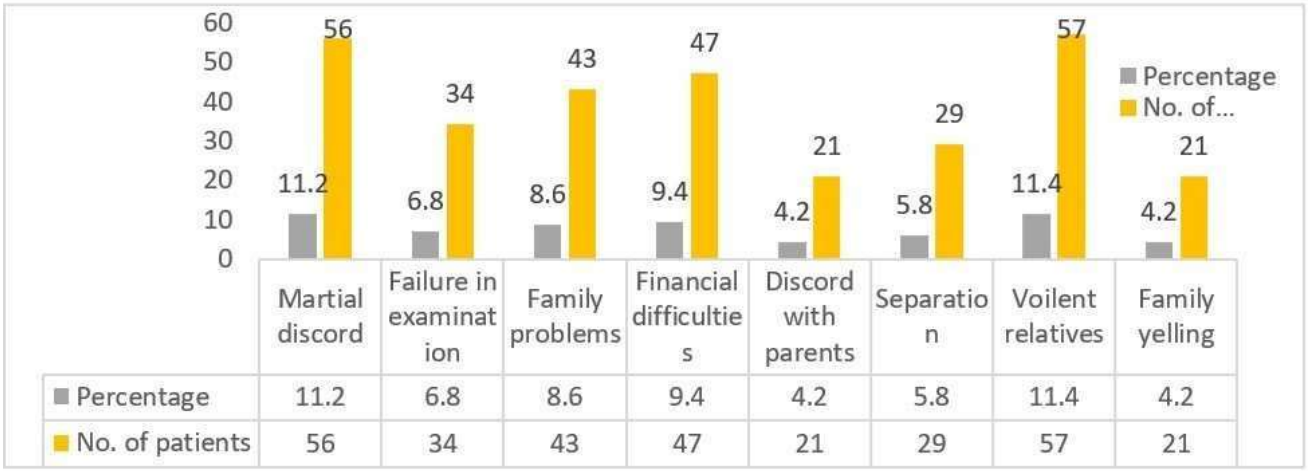
Dyslipidaemia	4	0.8
Immunosuppression	97	19.4
CNS excitation (Obtundation, Stupor, Coma, Agitation)	33	6.6
CNS depression		

Table2: Complications observed after poision consumption in the patient and data was analysed



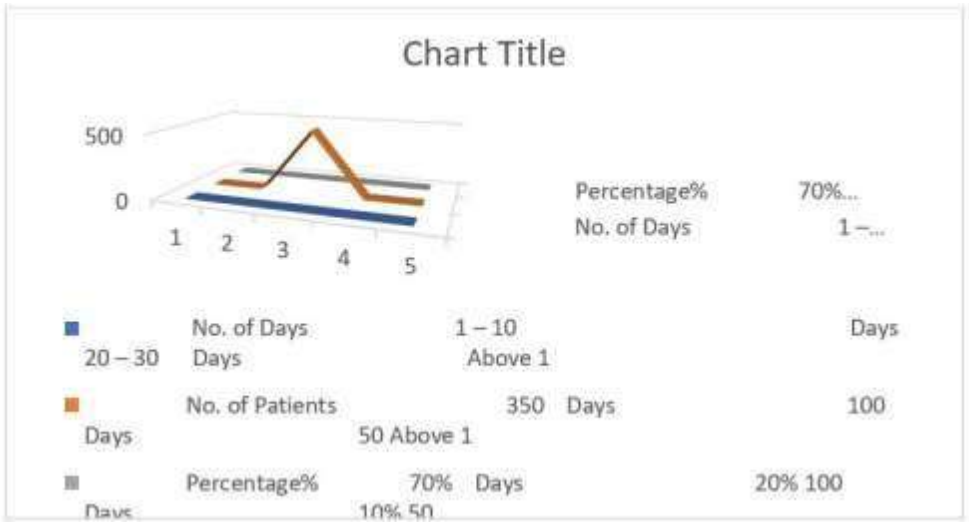
Causes of poisoning	No. of Patients	Percentage%
Marital discord	56	11.2
Failure in examination	34	6.8%
Family problems	43	8.6%
Financial difficulties	47	9.4%
Discord with parents	21	4.2%
Separation/death of family member	29	5.8%
Loss of job	32	6.4%
Violent relatives	57	11.4%
Beating in the family	29	5.8%
Family insults	36	7.2%
Family yelling	18	3.6%
Family indifference	21	4.2%

Table 3: Reasons for the administration of Poison and this data collected from questionaries



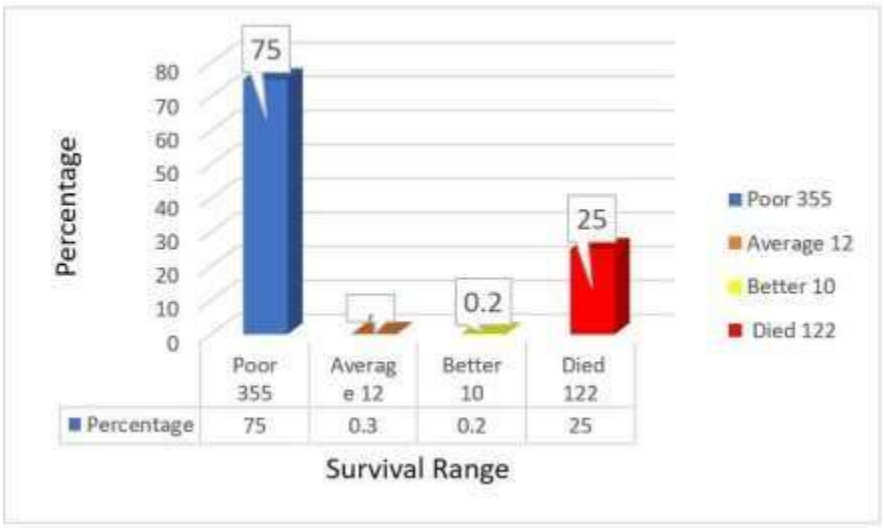
No. of Days	No. of Patients	Percentage %
1 – 10	350	70%
20 - 30	100	20%
Above 1 month	50	10%

Table 4: Duration of hospital stay with no. of patients with their percentage and this data was obtained



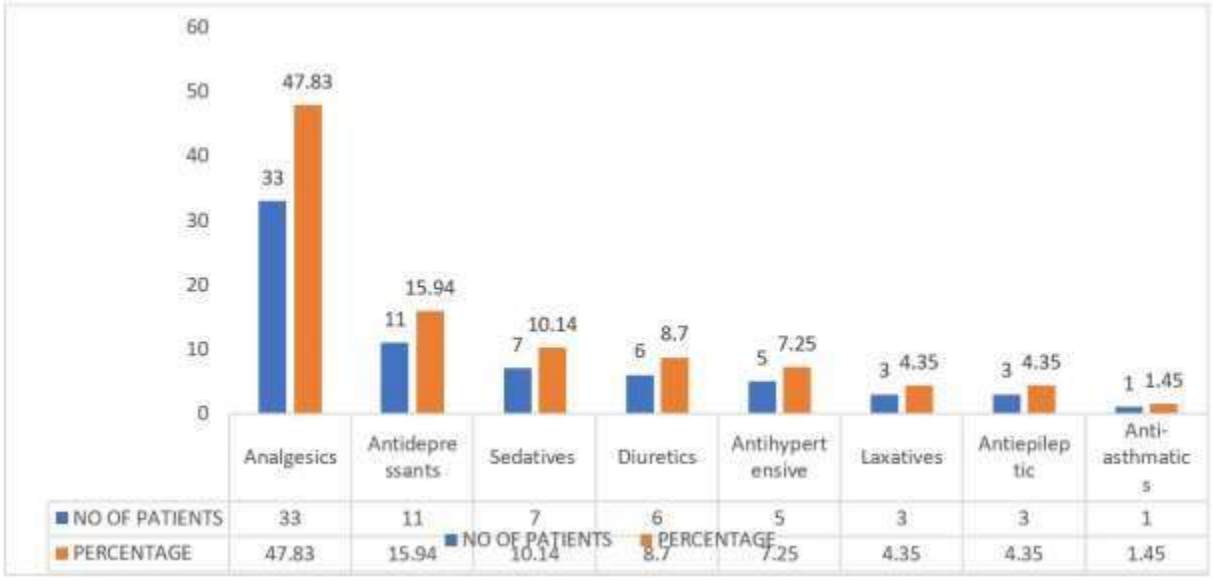
Survival Rate	No. of Patients	Percentage %
Poor	355	75%
Average	12	0.3%
Better	10	0.2%
Died	122	25%
Total	500	100%

Table 5: Survival rate of the Patient admitted in the hospital and their outcome and this data was collected from Questionares.



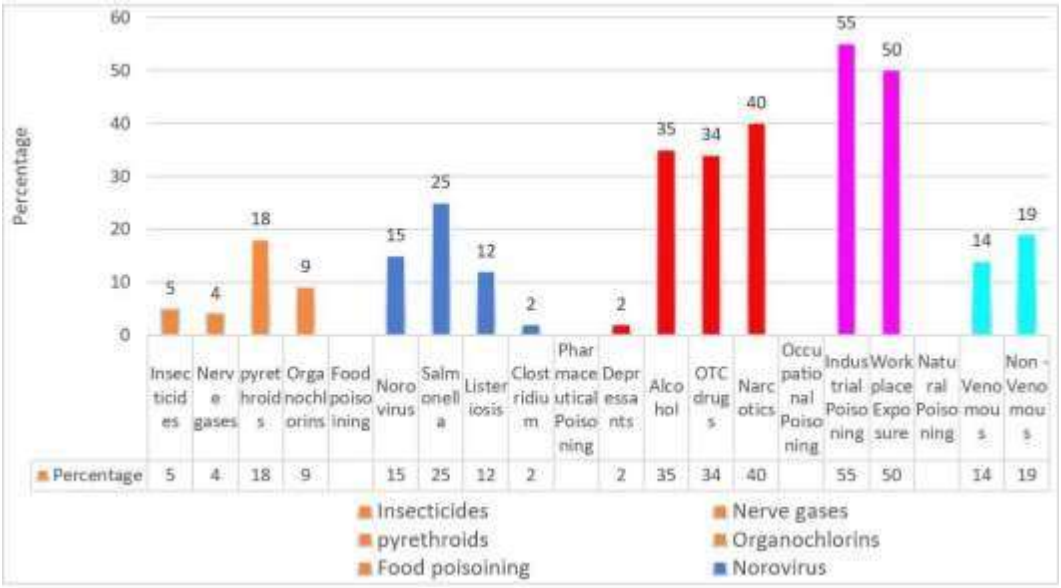
Class of drug	No. of patients	Percentage%
Analgesics	33	47.83
Antidepressants	11	15.94
Sedatives	7	10.14
Diuretics	6	8.7
Antihypertensive	5	7.25
Laxatives	3	4.35
Antiepileptic	3	4.35
Anti- asthmatics	1	1.45
Total	69	100

Table 6: Different class of drugs used in the treatment of Poison and this data was collected from patients for the study

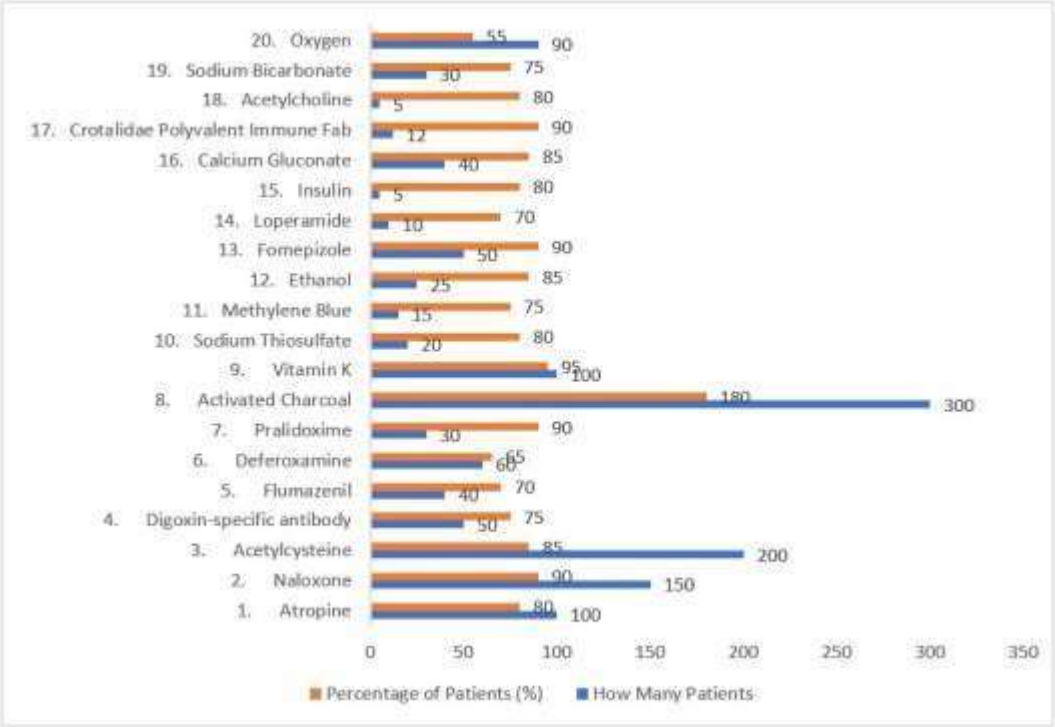


Types of Poisoning	No. of Patients	Percentage %
Organophosphates		
A) Insecticides	25	5
B) Nerve Gases	20	4
C) Pyrethroids	30	18
D) Organochlorines	15	9
Food Poisoning		
A) Norovirus	29	15
B) Salmonella	35	25
C) Listeriosis	20	12
D) Clostridium	10	2
Pharmaceutical Poisoning		
A) Depressants	10	2
B) Alcohol	40	35
C) OTC drugs	34	34
D) Narcotics	50	40
Occupational Poisoning		
A) Industrial Poisoning	60	55
B) Work place exposure	55	50
Natural Poisoning		
A) Venomous	28	14
B) Non-Venomous	31	19
	14	19
	19	25

Table 7 :Different types of Poisonous Substance are consumed by the people and admitted in hospitals, extracted from the questionares.



Name of Antidote	How Many Patients	Percentage of Patients (%)
1. Atropine	100	80
2. Naloxone	150	90
3. Acetylcysteine	200	85
4. Digoxin-specific antibody	50	75
5. Flumazenil	40	70
6. Deferoxamine	60	65
7. Pralidoxime	30	90
8. Activated Charcoal	300	60
9. Vitamin K	100	95
10. Sodium Thiosulfate	20	80
11. Methylene Blue	15	75
12.Ethanol	25	85
13 Fomepizole	50	90
14. Loperamide	10	70
15. Insulin	5	80
16. Calcium Gluconate	40	85
17. Crotalid Polyvalent Immune Fab	12 5	90 80
18. Acetylcholine	30	75
19. Sodium Bicarbonate	400	95
20. Oxygen	90	55
21. Antivenin	10	85
22. Dantrolene	20	90
23. Hydroxocobalamin	30	80
24. Etaracizumab	15	75



Discussion

This study analyses trends in pharmaceutical and non-pharmaceutical poisoning cases based on data collected retrospectively and prospectively from 997 poisoning cases referred to two government Hospitals between December 2023 and November 16, 2024. The study reveals that 85% of the patients were admitted to medicine departments, with a significant number of cases involving pesticide poisoning, particularly in developing countries. This trend is consistent with findings from earlier studies (Bobst, 2017) which highlighted the predominant role of pesticide exposure in developing nations, contrasting with pharmaceutical abuse as a leading cause in developed regions. The Monte Carlo data further supports the observation that repeated large doses of pharmaceuticals are a significant cause of poisoning. The incidents of poisoning, whether intentional or accidental, are on the rise, despite efforts by legislative, punitive, and social education mechanisms aimed at mitigation (Sharma et al., 2007).

An increasing trend in poisoning-related deaths was observed, rising from 18.59% in 1996 to 25% in 2004, with the highest incidence (48.4%) in the 16–25 age group. This pattern aligns with findings from Bhullar et al. (1996) and Bajaj and Wasir (1988), emphasizing the vulnerability of younger populations due to societal pressures, materialistic aspirations, and the inability to cope with competitive environments. An analysis of poisoning trends in the USA from 1920 to 2001, extending to 2023 in 18 states, showed a 56% increase in deaths due to poisoning, rising from 5% per 100,000 in 2001 to 11% in 2023. Unintentional and undetermined poisoning cases contributed significantly to this increase, while homicides remained stable, and suicides showed a decline.

In developing countries, organophosphate poisoning remains a critical issue, accounting for significant mortality and disability due to pesticide exposure (Eddleston et al., 1998). Our study revealed that 78% of victims were from rural backgrounds, highlighting the compounded challenges of limited resources, larger family sizes, and lower literacy rates compared to urban areas. Similarly, a study in Nepal (Maskey et al., 2012) found organophosphate poisoning to be more prevalent among males, often linked to marital discord, consistent with research by Jesslin et al. (2010). The most affected age group was 20–30 years, aligning with findings by Evelise Barboza et al. (2021)

and others. Additionally, poisoning cases predominantly involved married individuals, likely due to the added family responsibilities, societal customs, and resource constraints, as observed in studies conducted in Lalitpur (2008).

Intentional poisoning emerged as the most common cause, reflecting the prevalence of parasuicide through pharmaceutical and non-pharmaceutical substances in Asia, particularly in China, India, Pakistan, Bangladesh, and Sri Lanka (Eddleston, 2007). Non-pharmaceutical poisonings frequently presented with central nervous system excitation and cardiovascular manifestations, causing severe multi-organ damage compared to pharmaceutical poisonings (Rottmann and Greenla, 2008). Notably, pesticide poisoning exhibited a higher mortality rate, consistent with findings by Gasparrini et al. (2015). According to the World Health Organization, nearly 1 million suicides occur annually worldwide, with 185,961 from India. Self-poisoning accounts for 16%–49% of all suicide cases globally, underscoring its longstanding role as a common method of suicide.

The study also observed that most poisoning cases involved individuals with lower educational attainment. Middle and primary school-educated patients accounted for 32.7% and 23.7%, respectively, which aligns with findings by Jesslin et al. (2010). Additionally, nuclear families represented 53.8% of cases, while joint families accounted for 40.62%, reflecting evolving familial structures (Subhadip Bharati et al.). These findings highlight the need for region-specific epidemiological updates to identify risk factors and tailor prevention strategies. The study underscores the critical importance of addressing underlying socio-economic and psychological determinants of poisoning while fostering regular data collection to inform public health policies and interventions.

Conclusions

Acute poisoning remains a significant and urgent medical issue, particularly in low-income and underdeveloped countries, where the incidence is substantially higher than in developed nations. This study analysed a poisoning case, emphasizing risk assessment through questionnaires and a review of prior research. The findings revealed that while specific antidotes and treatments exist for certain poisons, many toxic agents still lack effective antidotes or standardized therapeutic protocols, posing a challenge to healthcare systems in resource-limited settings. To mitigate this crisis, governments and manufacturers should enforce stricter regulations on the sale of pesticides, herbicides, and fertilizers, complemented by farmer education on the safe use and toxic effects of these substances. Awareness campaigns are essential to minimize unnecessary usage and reduce the risk of poisoning. Furthermore, the mortality and morbidity associated with poisoning can be significantly reduced by implementing educational programs, conducting audio-visual presentations on prevention strategies in rural areas, and providing counselling and poison information services. Health professionals must remain vigilant about the patterns of common poisoning agents and be trained in emergency management, including household interventions. This study underscores the urgent need for global efforts to enhance preventive measures, improve treatment protocols, and raise awareness about poisoning, particularly in vulnerable regions (Lall SB et al., [41]; Bhattarai MD et al., [27]).

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