Original Research Article

Clinical Profile and Outcome of Patients Admitted with Rodenticide Poisoning – A Single Centre Experience from Andhra Pradesh

B.S.V.V.Ratnagiri^{1*}, Lella Padmaja², Praveen Kasina³, M.Jagan Mohan⁴, Deepthi Madineni⁵, M.Alekhaya⁶, Srivani Reddy Anireddy⁷

¹Associate Professor, Department of Gastroenterology, Siddhartha Medical College, Vijayawada, A.P, India ²Tutor, Department of Obstetrics and Gynecology, Siddhartha Medical College, Vijayawada, A.P, India ³Intern, Siddhartha Medical College, Vijayawada, A.P, India

⁴Professor & HOD, Department of Gastroenterology, Siddhartha Medical College, Vijayawada, A.P, India ⁵Post Graduate, Department of General Medicine, Siddhartha Medical College, Vijayawada, A.P, India ⁶Intern, Siddhartha Medical College, Vijayawada, A.P, India

⁷Intern, Siddhartha Medical College, Vijayawada, A.P, India

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Abstract

Introduction: Rodenticides are easily available and inexpensive poisons found in almost every house in India to prevent rodent infestation. Rodenticide poisoning is the second most common type of poisoning in the Indian Subcontinent, and there is a limited amount of research on it. The aim of our study is to evaluate the clinical profile of patients admitted with rodenticide poisoning and to correlate various parameters like serum bilirubin, and serum creatinine with mortality. **Methodology:** A prospective hospital-based study of 100 consecutive cases diagnosed as Spontaneous Pneumothorax from January 2019 to September 2020. Every patient underwent comprehensive history taking, in-depth clinical examination, and investigations. Data were entered into Excel spreadsheets in 2019. Data was presented by using frequency and percentage and descriptive statistics were used. **Results:** A total of 100 patients were included in the study. The most commonly consumed poison was a phosphorus-based paste. The most common symptom reported was abdominal pain (90%). Warfarin-based baits were associated with a high incidence of bleeding manifestations. The mortality rate was 20%. Additionally, a delay in hospitalization was found to be a significant risk factor for mortality. Higher mortality rates and was accompanied by elevated bilirubin and SGPT levels. **Conclusion:** In this study, mortality was 20%, all due to phosphorus compound. Mortality was common in phosphorus compound, particularly in those who developed jaundice on day 4 of admission which is reflected by elevated bilirubin and serum creatine level.

Keywords: Rodenticide Poisoning, India, Self-Poisoning, Phosphorus Poisoning

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Introduction

Poisoning is a largely preventable cause of death, primarily resulting from suicide and rarely from accidents, in developing nations. In rural areas of India, poisoning is a major contributor to casualties and accounts for 20-25% of critical care admissions. Organophosphorus and rodenticide poisoning are the most common forms of poisoning in this region. Intentionally ingesting toxic substances which is known as Deliberate Self-Poisoning (DSP), is a major contributor to death and injury. Many individuals who engage in DSP commonly use rodenticides as the toxic substance.

Rodenticide poisoning can present with a wide range of clinical manifestations depending on the type and quantity of rodenticide ingested. Common symptoms include gastrointestinal disturbances (nausea, vomiting, abdominal pain), bleeding tendencies (hematuria, epistaxis), neurologic abnormalities (seizures, altered mental status), and cardiovascular instability. In severe cases, patients may develop multi-organ failure, requiring intensive care management. Rodenticides commonly available in three different forms: powder containing zinc phosphide, paste containing yellow phosphorus, and bait containing super warfarin. However, very limited research is available on the effects of consuming yellow phosphorus in the Indian subcontinent.

*Correspondence

Dr. B.S.V.V.Ratnagiri

Associate Professor, Department of Gastroenterology, Siddhartha Medical College, Vijayawada, A.P. India

Rodenticide poisoning can affect multiple systems within the body and there are no established treatment guidelines for managing such cases. Additionally, only limited clinical studies are available exploring the effects of phosphorus paste poisoning, which is considered the most toxic type of rodenticide poisoning. The clinical profile and outcome of patients admitted with rodenticide poisoning are crucial factors in understanding the magnitude of the problem, identifying associated risk factors, and improving patient care strategies.This study aims to investigate the clinical profile and outcome of patients admitted with rodenticide poisoning in a tertiary care hospital, in Andhra Pradesh.

Materials and methods

A Prospective longitudinal observational study was conducted in the department of General Medicine of a tertiary care teaching hospital from November 2019 to July 2021 (20 months). During the study period, a total of 108 patients were admitted to our emergency medical ward with a suspected history of ingesting rodenticide compounds. However, after carefully applying our inclusion and exclusion criteria, we selected only 60 patients who met all the necessary criteria, and they were included as the study subjects (n = 60).

Inclusion Criteria

• Patients who had ingested rodenticide compounds in the form of bait, paste, or powder.

Exclusion Criteria

- Patients who had initially been treated at another medical facility and were later referred to our hospital.
- Patients who presented to the hospital more than 24 hours after ingesting the rodenticide were also excluded.
- Patients who had consumed poison along with alcohol or other compounds.
- Patients with a history of chronic alcoholism or jaundice in the recent past were excluded.

Method of collection of data

The study subjects were patients who were admitted to the emergency medical ward with a history of ingesting a rodenticide compound. A comprehensive history, physical examination, and relevant laboratory tests were performed on these patients. After obtaining a detailed history, a thorough physical examination was performed, with a special emphasis on vital signs, jaundice, and bleeding symptoms, as outlined in the study's proforma. The patient was then closely monitored during their hospital stay for the development of jaundice or other adverse outcomes, and relevant laboratory test results were collected. Particular attention was paid to symptoms such as jaundice, bleeding tendencies, oliguria, seizures, or headache. On admission, on the 4th day, and subsequently, laboratory values such as serum bilirubin, INR, serum creatinine, and SGOT (serum glutamate oxido transferase) were collected. Standard decontamination methods were used to manage patients, including nasogastric lavage, and treatment was tailored to the specific compound ingested. N-acetyl cysteine was used in the ICU. Survivors were provided with counseling. Autopsies were performed on patients who died.

Statistical Analysis

Data was entered into EXCEL Spread Sheets 2019 and SPSS Version 20 was used for the Chi-square test one-way ANOVA 'f test.

Ethical Considerations

Ethical committee clearance was obtained before the commencement of the study. Informed consent was obtained from all study participants.

Results

A total of 60 patients were included in the study.

The study included 60 patients who ingested poison. Of these patients, 23.3% were less than 20 years old, 55% were between 20-30 years old, and 21.66% were over 30 years old. Males accounted for 63% of the patients while females accounted for 37%. Patients from low socioeconomic status accounted for 78.3% of the total while those from middle socioeconomic status accounted for 21.3%. None of the patients were from high socioeconomic status.

Of the patients, 43.3% were married while 56.7% were unmarried. In terms of poison consumption, 11.7% consumed bait, 70% consumed paste, and 18.3% consumed powder.

Nearly half of the patients (48.3%) presented within four hours of ingestion while 36.7% presented between four and eight hours after ingestion and 15% presented after eight hours.

Of the patients who ingested poison, 58.3% consumed less than 10 g of poison with most ingesting phosphorus compound while 35%

consumed between 15-20 grams and only 6.7% consumed more than 20 grams.

The most common symptoms were pain abdomen (88%), vomiting (46.6%), loose stools (40%), and thirst (33.3%). Jaundice was present in only 20% of patients but was associated with higher mortality rates. Most of the patients developed jaundice often on day 3-4 of ingestion. Almost all of these patients consumed paste (Phosphorus) and almost every patient died immediately which explains the leading cause of death and also the most dangerous compound. Out of 60 patients 7 Patients developed bleeding manifestations after consumption of poison. Out of which five patients had ingested bait (hydroxyl coumarins) and other 2 had ingested paste (phosphorus) this effect is due to acute hepato toxicity in patients in bait group, that had recovered after Inj.vitamin K. Out Of 60 patients ten patients developed decrease in urine output and all of them belongs to paste (phosphorus) category, and all of them were deceased which explains that toxicity was also due to renal damage.

Only four patients had headache without any further neurological features, and all these four are members of paste (phosphorus) category, and all these four patients had deceased. 23 patients had elevated bilirubin levels greater than 1.5 mg/dL, and out of these, 12 patients (52%) died. The remaining patients (48%) experienced a decrease in bilirubin levels over time and showed improvement in their condition. 24 patients had an ALT value greater than normal value (40 IU/L), which parallels with increased bilirubin level, 12 patients had values greater than 5 to 10 times the routine value, indicating fulminant liver failure that carries nearly 100% mortality. 60 patients 12 patients had elevated serum creatinine values, which implies the renal injury secondary to phosphorus compound. Out of all patients, 12 patients died. All of them had consumed paste (phosphorus), thus showing the lethality poison.

The death rate is the same for both male and female patients but males have a greater admission rate than females.

In this study, the paste group (phosphorus compound) had the highest number of deaths, accounting for 28.5% of deaths among those who consumed it. Compounds containing phosphide and warfarin did not cause any deaths. In this study, 13 individuals (21.6%) had jaundice with 12 patients (92.3%) had deceased. Only 1 patient survived, who had been treated with N-acetyl cysteine from the day of visit to the hospital. Out of 60 patients, 10 patients had oliguria out of which 9 patients had expired and only one patient survived. In this study, 23 patients had elevated bilirubin levels greater than 1.5 mg/dl, and out of these, 12 patients (52%) died. The remaining patients (48%) experienced a decrease in bilirubin levels over time and showed improvement in their condition. 13 patients (28.5%) had increased ALT values of greater than 80 IU/l, out of them 12 patients (92.3%) died and 1 patient survived 11 patients (18.3%) had levels of 40 to 80 IU /l, and 100% of them were recovered. Thereby ALT level of more than 5 timesnormal along with increased bilirubin and jaundice carries nearly full mortality. Out of 60 patients 12 patients had elevated creatine out of them 9 patients had expired.

The above table indicates that there is a significant difference between bilirubin on admission.

n, 4th day, >1 week, ALT, INR, creatine of the respondents, and their quality. Hence the calculated value is less than the table value (p<0.05)



Figure - 1: Type/Quality of Poison Ingested

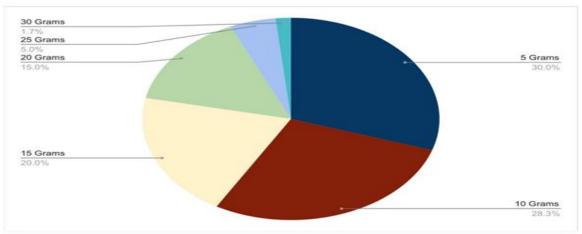


Figure - 2: Quantity of Poison Ingested

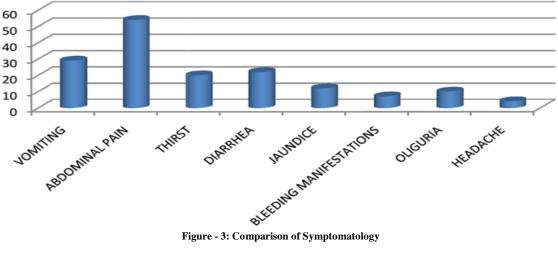


Figure - 3: Comparison of Symptomatology

Table – 1: Comparison of various parameters stratified by death and recovery								
Variable		Death	Recovery	Total				
	Less than 20	2 (16.7%)	12 (25%)	14 (23.3%)				
Age	21 to 30	7 (58.3%)	26 (54.2%)	33 (55%)				
	31 and greater	3 (25%)	10 (20.8%)	13 (21.7%)				
Gender	Male	6 (50%)	30 (62.5%)	38 (63.3%)				
	Female	6 (50%)	18 (37.5%)	22 (36.6%)				
Quality of the Poison	Bait	0 (0%)	7 (14.6%)	7 (11.7%)				
	Paste	12 (100%)	30 (62.5%)	42 (70%)				
	Powder	0 (0%)	11 (22.9%)	11 (18.3%)				
	Less than 4 hrs	1 (8.3%)	28 (58.3%)	29 (48.3%)				
Time Delay	4 to 8 hrs	4 (33.3%)	18 (37.5%)	22 (36.7%)				
	Greater than 8 hrs	7 (58.3%)	2 (4.2%)	9 (15.0%)				
Jaundice	Yes	0 (0 %)	47 (97.9 %)	47 (78.4%)				
	No	12 (100%)	1 (2.1%)	13 (21.6%)				
Serum Bilirubin (mg/dl)	Less than 1.5	0 (0.0%)	37 (77.1%)	37 (61.7%)				
	Above 1.5	12 (100%)	11 (22.9%)	23 (38.3%)				
	Below 40	0 (0.0%)	36 (75 %)	36 (60 %)				
ALT (IU/l)	41 to 80	0 (0.0%)	11 (23.5 %)	11 (18.3%)				
	Greater than 81	12 (100.0%)	1 (1.5%)	13 (21.7%)				

Table – 2: One way ANOVA difference between SGPT, bilurubin on admn, 4th day, >1 wk, INR, creatinine of the respondents and the quality of poison

Para	meter	Mean	S.D	SS	Df	MS	Statistical Inference
	Between groups			2.099	2	1.049	
On admission	Bait (n=7)	0.9143	0.21931				F=6.774
	Paste (n=42)	1.3357	0.44657				0.002
	Powder (n=11)	0.9364	0.19117				(p<0.05)
	Within groups			8.830	57	0.155	Significant
	Between groups			45.862	2	22.931	
	Bait (n=7)	0.9429	0.26992				F=6.184
On 4 th day	Paste (n=42)	2.5310	2.25318				0.004 (p<0.05) Significant
	Powder (n=11)	0.4545	0.52795				
	Within groups			211.374	57	3.708	
	Between groups			37.338	2	18.669	
>1 week	Bait (n=7)	0.000	0.0000				F=3.491 0.037
	Paste (n=42)	1.7214	2.72661				
	Powder (n=11)	0.0000	0.00000				(p<0.05)
	Within groups			304.811	57	5.348	Significant
	Between groups			4.164	2	2.082	
	Bait (n=7)	2.1914	0.55364				F=5.732
INR	Paste (n=42)	1.5493	0.67715				0.005
	Powder (n=11)	1.2073	0.08063				(p<0.05)
	Within groups			20.704	57	0.363	Significant
	Between groups			47.111	2	29.055	
SGPT levels	Bait (n=7)	0.9129	0.35336				F=6.050
	Paste (n=42)	2.6190	2.14607				0.04
	Powder (n=11)	0.4545	0.5323				(p<0.05)
	Within groups			213.489	57	3.623	Significant
	Between groups			1.469	2	0.734	
	Bait (n=7)	0.9429	0.69488				F=2.609
Creatinine	Paste (n=42)	1.1286	0.55887				0.0082
	Powder (n=11)	0.7273	0.18488				(p<0.05)
	Within groups			16.045	57	0.281	Significant

Discussion

Age

The majority of patients in this study (55%) were between the ages of 21-30, with 78.3% of patients being under 30 years old. This is consistent with the findings of P S Shankar et al[55] and Doshi et al[54] studies, which also reported a higher proportion of patients in the 21-30 age group. In Doshi et al[54] 44.44 % are in the age group of 21-30 years whereas in Shankar et al[55] 60% of patients are in 21-30 years age group.

Sex

Males accounted for 63.3% of the participants in this study, while

females accounted for 36.6%. This results in a male to female ratio of 1.5:1, which is similar to the findings of Shankar et al and Prakash et al[56] studies. In Shankar et al, 59.8% of the participants were male and 40.2% were female, with a male to female ratio of 1.48:1. In Prakash et al[56], 60% of the participants were male and 40% were female, with a male to female ratio of 1.5:1.

Socioeconomic Status

In this study, 78.3% of the participants are from lower socioeconomic backgrounds, while 21.7% are from the middle class. This is similar to the findings of Chatterjee et al[56] and Goel et al[57] studies. Chatterjee et al[56] reported that 88% of patients were from low

socio-economic status, 11% from middle socio-economic status and 1% from high socio-economic status. While in Goel et al[57] reported that 75.73% patients from low socioeconomic status, 24.27% from middle socio-economic status and 0% from high socio-economic status.

Type of poison ingested

The majority of patients in this trial (42 or 70%) consumed paste (phosphorus compound), followed by powder (11 or 18.3%), and bait (super warfarin) (7 or 11.7%). This is similar to the findings of Vijaya Shekar et al study, where 69% of patients consumed phosphorus and phosphide compounds and 31% consumed super warfarin.

Time interval between ingestion and hospitalization

In this study, patients who were admitted to the hospital more than 4 hours after ingestion had a much higher death rate (91.6%) compared to those who were admitted within 4 hours, which had a significantly lower mortality rate (8.3%). This is consistent with the findings of Goel et al study[57] where patients presenting within 4 hours are 14.5% and deaths among them are 20%, patients presenting in 4-8 hours are 54.4% and deaths occurring among them are 28.57%, and patients presenting after 8 hours are 31.1% and deaths among them are 53.12%.

Biochemical analysis

In this study, 23 patients (38.3%), who mostly consumed phosphorus compound, had a serum bilirubin level on the fourth day of admission that was higher than 1.5 mg/dL. Out of these 23 patients, 12 (52.2%) died. This is consistent with the finding of increased ALT levels on the fourth day of admission, which suggests acute liver toxicity from the phosphorus compound. 12 patients who consumed phosphorus also had increased serum creatinine.

Serum creatine with mortality

In this study, it was found that 20% of the patients had elevated creatine levels in their blood and 75% of them passed away. Additionally, out of the 60 patients admitted, 20% of them died, all of whom had consumed a paste containing phosphorus. Analysis using the Chi square test revealed no significant correlation between factors such as sex, age, socioeconomic status, marital status, abdominal pain, bleeding symptoms, length of hospital stay and patient outcome. However, there was a strong correlation between factors such as the quality, quantity, delay, thirst, diarrhea, jaundice, oliguria, head discomfort, bilirubin levels on the fourth day, ALT levels, and creatine levels with patient outcomes. These correlations were statistically significant with a p-value less than 0.05.

Oneway ANOVA difference between SGPT, bilurubin on admn, 4th day, >1 wk, INR, creatinine of the respondents and the quality of poison

Conclusion

Rodenticide poisoning is the second most commonly ingested poison in our area which is commonly available in three different forms, namely phosphorus compound (ratol paste),zinc phosphide (powder), super warfarin (bait).

In this study mortality was 20 %, all due to phosphorus compound. Mortality was common in phosphorus compound, particularly who developed jaundice on day 4 of admission which is reflected by elevated bilirubin, ALT and serum creatine level.

Hence phosphorus is considered as deadliest compound and must be banned to prevent mortality in young productive poor patients, who intend to ingest poison in a minute decision. N-acetyl cysteine can be tried in early stages of hepatotoxicity.

References

- 1. Hayes WJ: Pesticides studied in man. Baltimore, Williams & Wilkins, 1982.
- Public Health Study Team: Pest control and Public Health, Vol.
 S. Washington, DC, National Academy of Sciences, 1976, p81.
- Lisella FS, Long KR, Scott HG: Toxicology of rodenticides and their relation to human health. J Environ Health 1970;33:231-237.
- Goldfrank's Toxicologic emergencies 7th edn, 2002:90: 1379-1390.

- Fernandez OU, Canizares LL: Acute hepatotoxicity from ingestion of yellow phosphorus – containing fireworks. J Clin Gastroenterol 1995;21:139-42.
- 6. Ghoshal AK, Porta EA, Hartroft WS: Isotopic studies on the absorption and tissue distribution of white phosphorus in rats. Exp Mol Pathol 1971; 14:212-219.
- Cameron JM, Patrick RS: Acute phosphorus poisoning of toxic doses of yellow phosphorus in tissues of experimental animals. med sci law 1966;6:209-214.
- Warnet JM, Claude JR, Truhaut R: Experimental biological toxicity of white phosphorus. Eur J Toxicologic Hyg Environ 1973;6:57-64.
- 9. Diaz –Rivera RS, Collazo PJ, Pons ER: Acute phosphorus burn . Mil Med 2002;167:83-84.
- Marin GA, Montonya CA, Sierra JL, Senior JR: Evaluation of corticosteroid and exchange-transfusion treatment of acute yellow- phosphorus intoxication N Engl J Med 1971;284:125-128.
- 11. Rubitsky HJ, Myerson RM: Acute phosphorus poisoning Arch Intern Med 1949;83:164-178.
- 12. Bowen TE, Whelan TJ,Jr, Nelson TG: major death after phosphorus burns. Amm Srug 1971;174:779-784.
- McCarron MM, Gaddis GP, Trotter AT: Acute yellow phosphorus poisoning from pesticide pastes . Clin Tox 1981;18:693-711.
- Burnell JM, Dennis MB,Jr, Clayson KJ, et al:treatment of acute hepatic necrosis induced by yellow phosphorus .1976;71:827-831.
- 15. Ganote CE, Otis JB: Characteristic lesions of yellow phosphorus induced liver damage . Lab Invest 1969;21:207-213.
- Streliukhina NA: Effect of cysteine hydrochloride on morphological changes in liver with yellow phosphorus poisoning. Biull Eksp Biol Med 1984; 98: 111-115.
- Ben-Hur N,Giladi A, Neuman Z: Phosphorus burns : A Pathophysiological study. Br J Plastic Surg 1972;25:238-244.
- Mozingo DW Smith AA, McManus WF, et al: Chemcal burns. J Trauma 1988;28:642-647.
- 19. Simon FA, Pickering LK: Acute yellow phosphorus poisoning "smoking stool syndrome". JAMA 1976;235:1343-1344.
- 20. Klaser AE, Scalzo AJ, Blume C, et al. Ann Emerg Med 1996;28:713-718. 21.
- Van Mieghem C, Sabbe M, Knockaert D: The clicical value of the ECG in noncardiac conditions.chest 2004;125:1561-1576.
- 22. Fisher NG, Armitage A, McGongile RJ: Hypocalcemic cardiomyopathy.Eur J Heart Fail 2001;3:373-376.
- Koch A, Hofbeck M, Dorr HG, Singer; Hupocalcemia –induced heart failure as the intial symptom. Z Karidol 1999;88:10-13.
- Talley RC, Linhart JW, Trevino AJ, et al: Acute elemental phosphorus poisoning in man: cardiovascular toxicity. Am Heart J 1972;84:139-140.
- Riggs JE: Neurological manifestations of electrolyte disturbances. Neurol Clin 2002;20:227-239.
- Summerlin WT,Walder AI, Moncrief JA: White phophorus buns and massive haemolysis. Trama 1967;7:476-484.
- 27. Schellmann B, Zober A, Zink P: Suicide by phosphorus poisoning. Arch Tox 1979;42:303-309.
- 28. Konoyan TR: White phosphorus buns: Case report and liteature review.Mil Mad 1983; 148: 881-884.
- 29. Davis KG: Acute management of white phosporus bun. Mal Med 2002;167:83-84.
- Song ZY, Lu YP, Gu XQ: Management of yellow phosporus burs with silver nitrate. Scand J Work Environ Health 1985;11:33.
- Winek CL, Collom WD, Fusia EP: Yellow phosphorus ingestion. Clin TOX 1973; 6:541-545.
- Elizabeth J, Kelkar PN, Weishali G: Yellow phosphorus poisoning –an unusual presentation. J Physicins India 1995;43:371-372.
- 33. Stephenson JBP: Zinc phosphide poisoning . Arch Environ

Health 1967;15:83.

- Chugh SN, Aggarwal HK, Mahajan SK: Zincphosphide intoxication symptoms: Analysis . Int J Clin Pharmacol Ther 1998; 36: 406-407.
- Rodenberg HD, Chang CC, Watson WA: Zinc phosphide ingestion: a case report and review. Vet Hum Toxicol 1989;31:559.
- Johnson HD, Voss E: Toxicologic studies of zinc phosphide. J Am Pharm Assoc 1952;41:468.
- Watson WA, Litovitz TL, Rodgers GC Jr et al:2004 Annual report of the Ameri. Association of Poison control Centers Toxic Exposure Surveillance System Am J Emerg Med 2005;23:589-666.
- Babcock J, Hartman K, Pedersen A, et al: Rodenticide-induced coagulopathy in a young child. Am J Pediatr hematol oncol 1993;15:126-130.
- Lipton RA, Klass EM. Human ingestion of a "superwarfarin" rodenticide resulting in a prolonged anticoagulant effect. JAMA 1998;252:3004.
- 40. Lovejoy FH: Thallium. Clin Toxicol Rev 1982;5:1-2.
- 41. Ben-Assa B: Indirect thallium poisoning in a Bedoiun Family. Harefuah 1962;62:378-380.
- Chi CH, Chen KW, Chan SH, et al: Clinical presentation and prognostic factors in sodium fluoroacetate intoxication, J Toxicol 1996;34:707-712.
- Chenoweth MB: Monoflouroacetic acid and related compounds. Pharm Rev 1949;1:383-424.
- Heiser JM, Daya MR, Magnussen AR, Norton RL: Massive strychinine intoxication: Serial blood levels in a fatal case. J Toxicol Clin 1992;30:269-283.

- 45. PDR for Herbal Medicines, 2nd ed. Montvale, NJ, Medical Economics,2000.
- Shum S, Whitshead J, Vaughan L, et al: Chelation of organoarsenate. Vet Hum Toxicol 1995;37:239-242.
- 47. Wetherill SF, Guarino MJ, Cox RW: Barium chloride poisoning. Ann intern Med 1981; 95:187-188
- Herken H:Antimetabolic action of 6-amino-nicotinamide on the pentose phosphate pathway in the brain. In: Aldridge N, ed: Mechanism of Toxicity. London, St Martin's, 1970, p.189.
- 49. Prosser PR, Karm JH: Diabetes mellitus following rodenticide ingestion in man. JAMA 1978;239:1148-1150.
- Richter CP: The development and use of alpha-naphthylthiourea (ANTU) as a rat poison. JAMA 1945;129:927-931.
- 51. Tuncock Y, Kozan O, Caudar C, et al: urginea maritime (squill) toxicity . J Toxicol Clin 1995;33:83-86.
- Bova S, Travis L, Debetto P, et al: Vasorelaxant properties of norbromide, a selective vasoconstrictor agent. Br J Pharmacol 1996;117: 1041-1046.
- 53. Buller G, Heard J, Gorman S: Possible bromethalin-induced toxicity in humans. J Toxicol 1996;34:572.
- 54. Doshi et al: J Indian Acad Forensic Med. Apr- June, Vol- 33, NO 2.
- 55. P.S Shankar et al: Pulm edema in diazonion poisoning : Indian Manual of Chest disease 1967; 9:106-110.
- Chatterjee DC : Poisoning due to organophosphate insecticide , JIMA 1967;48:163.
- A Goel ,joseph S.Dutta TK: Organophosphorus poisoning, JAPI 1998;46:786-790