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AN EPIDEMIOLOGICAL STUDY OF ALUMINIUM PHOSPHIDE POISONING AT ALLAHABAD.

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Abstract

During one-year study period, out of a total 13,100 admissions, 301 suspected poisoning cases (2.3%) were admitted to the Swarup Rani Nehru Hospital, Allahabad. 120 poisoning cases (39.9%) were recorded in the summer season (March to June) and 235 (78.1%) were in the age group of 11-30 years. Male to female ratio was 2:1. Out of 204 males, 136 belonged to the rural area and 56 to the urban area, while in 13 cases, residential status could not be traced.

Out of the 1752 medicolegal autopsies conducted during the study period in the mortuary, 205 were suspected poisoning deaths and 83 of these were circumstantially established cases of aluminum phosphide poisoning. Most vulnerable age group was 21-30 years accounting for 40.5% of poisoning deaths. Male female ratio was 2:1. Maximum incidence of poisoning deaths was observed in the rainy season (41.95%). 61.5% of victims were of rural background.

On gross examination, almost all vital organs were found to be congested. A variety of microscopic changes were observed in different organs. Easy availability of this cheap, highly toxic substance was responsible for an increased incidence of aluminum phosphide poisoning or deaths.

INTRODUCTION

Poisoning is as old as our society. Of late, the incidence of poisoning cases is increasing steadily with each passing year. By definition, anything which when used internally or on the body surface in a dose or in repeated

doses, if acts chemically and physiologically, causing disturbances of body functions and leads to disease or death is a poison.1 As per law, poison is a substance which when administered is injurious to health or life.2 Poisoning, both intentional or accidental, significantly contributes to mortality and morbidity throughout the world. According to WHO, 3 million acute poisoning cases with 2,20,000 deaths occur annually.3 Of these, 90% of fatal poisoning occur in developing countries, particularly amongst the agricultural workers. Developing countries such as India and Sri Lanka have reported alarming rates of toxicity and deaths due to poisons.4 In United States, deaths due to poisoning number more than 775 per year. Most of the people who die from poisoning are adults and deaths often results from intentional rather than accidental exposure.5

Both homicidal and suicidal cases of poisoning are more common in India than in western countries, owing to easy availability of poison in the Indian markets. Insecticides and pesticides account for majority of these poisoning cases. More recently, Aluminium phosphide, because it is cheap, easily available, highly toxic, and has no antidote, has emerged as the most common suicidal agent.6 The toxic effects of Aluminium phosphide are due to deadly phosphine gas liberated when it reacts with water or hydrochloric acid in the stomach. Organophosphorus compounds are other more common cause of insecticide poisoning in India.7

The present study was been designed to assess the epidemiological features both in suspected cases of poisoning and in cases of poisoning deaths and to study the histopathological changes in different organs due to Aluminium phosphide poisoning.

MATERIAL AND METHODS

The material for the present study comprised of all suspected poisoning cases (SPC) brought to the emergency ward of the Swarup Rani Nehru. Hospital, attached to the M.L.N. Medical College, Allahabad as well as dead bodies of suspected poisoning deaths (SPD) brought for medico-legal examination to the mortuary from various police stations and nearby rural areas of Allahabad district.

The period of study was one year (Jan. 2003 to Dec. 2003). In SPC, data was retrieved from the emergency ward. Whereas in cases of poisoning deaths, both suspected or confirmed, data was collected from police inquest report, by interviewing the police personnel accompanying the dead body and relatives, friends, neighbors and co-workers of the deceased. In selected urban cases, investigation at the scene of crime was also conducted.

Of a total 13,100 admissions in the SRN Hospital, 301 SPC (2.3%) were admitted. Whereas, out of 1752 SPD medico legal autopsies conducted, a total of 205 poisoning deaths were observed.

RESULTS

Among all admissions, SPC constituted 2.3% of the total admissions whereas a prevalence of 11.7% was noted in SPD. Out of 205 poisoning deaths, 83 cases were established due to Aluminium phosphide poisoning. (Table-1)

Both for SPC and SPD male: female ratio was 2:1 (Table - 2). Age distribution study (Table - 3) showed peak prevalence in the age group 11 - 30 years for SPC (235 cases i.e. 78.1%) and 21-40 years for SPD (120 cases i.e. 58.5%) respectively. In both SPC and SPD cases aged below 10 years, the majority of poisoning was accidental in nature, while in adolescents and adults, the poisoning was mostly of suicidal intention.

Table 4 and 5 details the month wise and season wise breakup of SPC and SPD. Maximum incidence of suspected poisoning admissions was recorded in the summer season comprising of 120 cases (39.9%) followed by monsoon season (32.6%), while for poisoning deaths, a total of 86 cases were recorded in the rainy season (41.9%), followed by summer (36.6%). Both in SPC and SPD cases, a minimum incidence was noted in the winter months. In April 2003, a maximum of 34 cases of suspected poisoning were admitted whereas in August 03, a maximum of 28 poisoning deaths were recorded.

Table-6 shows incidence of SPC and SPD in relation to habitat. Of SPC, 196 cases (65.1%) belonged to the rural area whereas 92 cases (30.6%) belonged to the urban areas. Out of 205 cases of SPD, 126 cases (61.5%) belonged to the rural area and 31.7% belonged to the urban area.

Table-7 depicts the incidence of circumstantially proven Aluminium phosphide poisoning deaths. The presence of classical symptoms and signs of Aluminium phosphide poisoning, wrappers or dying statement of the deceased was taken as circumstantial evidence. In the present study, 83 cases (40.5%) of poisoning deaths due to Aluminium phosphide were recorded.

The naked eye examination of visceral organs in cases of Aluminium phosphide poisoning deaths exhibited congestion of brain, lungs liver, kidneys and stomach to varying degrees. Lungs were oedematous and there was no sloughing of gastric mucosa. Out of 83 Aluminium phosphide-poisoning deaths, tissue specimens for histopathological examinations were collected from 53 cases. On histology, brain showed congestion in 100% cases and degeneration in 60.4%. Lungs showed congestion in 100%, alveolar thickening 98.1%, oedema 92.5% besides dilated capillaries, collapsed alveoli and hemorrhages in descending percentage. Liver showed congestion in 100%, central venous congestion in 92.5%, degeneration, hemorrhage, sinusoidal dilatation, bile stasis, centrilobular necrosis etc. Kidneys showed congestion in 100% cases, tubular degeneration and necrosis in 73.6%, infiltration in 62.3%. Stomach was found congested in 100% cases followed by hemorrhages in 35.9%, and in none of the case oedema and necrosis of mucosa or round cell infiltration was noticed. (Table-8)

DISCUSSION

Suicide is among the top 10 causes of deaths in India. The commonest mode of committing suicide in India is by the ingestion of poison. In the present study, the admissions due to poisoning accounted for 2.3% of total patient admissions. It has been observed that the Aluminium phosphide poisoning was mainly used for suicide. An attempted suicide is an unsuccessful suicidal act with non-fatal outcome. However, some of the cases of attempted suicide may accidentally die during the act. Ahuja et al.8 reported that attempted suicide was more common in women, while completed suicide is 2-4 times more common in men. In the present study, the numbers of attempted suicides were more than successful sucides. Sagar et al,9 observed a steady increase in the number of suicidal deaths. These constituted an average of 7.9% of total fatalities during 1977-80, which increased sharply during 1988-91. In the present study an incidence of 11.7% of SPD out of total postmortems conducted was observed. (Table-1) It was further observed that most of suicides due to poisoning were easy to detect since the findings at the scene often make the issue clear, if actual scene was not disturbed. Since there was no facility to detect the exact nature of a poison in our hospital, hence the nature of poison could not be worked out, except where the victim or the family members or the relatives had voluntarily disclosed the causative agent A preponderance of males over females (M: F ratio 2:1) both in SPC as well as in SPD was a striking feature of the present study (Table-2) Higher prevalence in males may be explained on the basis of unemployment, lack of desired job avenues, rapidly changing stressful lifestyles, family conflicts and problems arising out of them. Similar findings were reported by Siwach et al,10 who observed males, out numbered females by about 1.6 times while attempting suicide by poisoning. Sinha et al.11 also noted that males out number females in cases of poisoning. Our findings are in general agreement with other workers in the field.12-15 However, Siwach et al,14 reported a higher M/F ratio of 4:1.

In this study, the maximum SPC belonged to the younger age group and that the age group of 21-30 years accounted for 41.2% of cases. Similarly, an involvement of younger age group was also recorded in cases of SPD, where, 21-30 years age group accounted for maximum of 40.5% of cases. Moreover, least number of cases was observed in persons beyond 50 years of age in both SPC and SPD. (Table-3). A possible explanation could be that younger persons were easily excited, enraged, depressed and attempted drastic steps like suicide; on the other hand, the more elderly individuals took more mature decisions. Our findings regarding involvement of the younger age group in poisoning were in consonance with those of others.10,11,15,16 Katira et al,12 have reported in their series of 90 Aluminium phosphide cases, involvement of younger age group 20-29 years in 38 cases, whereas Arora et al,16 observed maximum involvement of 10-29 years age group in SPD. However, Srivastava et al15 reported highest incidence of poisoning in children (36.5%). Sinha et al, 17 reported maximal homicidal deaths in younger age group (21-30 years) and it appears that there is a remarkable similarity between age group involved in suicidal poisoning deaths and homicidal deaths.

Out of 301 SPC, 120 cases (39.87%) were recorded in summer season. April (34 cases) and March (31 cases) recorded maximum admissions whereas out of 205 SPD the rainy season accounted for maximum of 86 cases (41.95%). August (28 cases) and July (27 cases) recorded maximum poisoning deaths. Most Aluminium phosphide poisoning deaths occurred in the rainy season. This difference in the seasonal incidence in SPC and SPD cannot be explained. Only scanty literature is available regarding seasonal variations in poisoning. Furthermore in both SPC and SPD, winter season recorded minimal number of poisoning. It is interesting to mention that Sinha et al.17 and Sheikh et al 18 have reported highest number of homicidal deaths in rainy season followed by winter and summer.

In the present study, a preponderance of rural habitat over urban habitat was been observed, both for SPC and SPD, moreover, males of the rural areas were predominantly affected in both situations. This could be explained by the fact that the males were more involved in outdoor activities, earned livelihood for the family and were subjected to stressful living conditions, while women were mostly confined to household work. A greater incidence in rural populace may be due to the fact that India is an agricultural economy and more people live and earned livelihood in rural areas in this part of the country. Besides in the Allahabad region, industries are very few. Our findings are in general agreement with those of others.19 Singh et al,20 have also observed that males of rural background outnumbered females and urban habitat in their series of poisoning cases. In the present study, one important observation is that incidence of poisoning suicidal deaths (11.7%) were fairly high compared to homicidal deaths.

In all the 205 cases of SPD, viscera was preserved and sent for chemical examination. However, the results of chemical analysis regarding exact poison could not be included because of procedural delays in sending the viscera to forensic laboratory, and due to secret communication, of chemical analysis repost directly to concerned courts. The presence of classical symptoms and signs of Aluminium phosphide poisoning, empty wrappers at the site of death or dying statement of the deceased was taken as circumstantial evidence of etiology of poisoning.

A naked eye examination during postmortem, of all the 83 cases of Aluminium phosphide revealed that almost all the vital organs were congested to varying degrees including brain, lungs, liver, kidneys and stomach. The heart appeared to be normal on gross examination. Sloughing or necrosis of gastric mucosa was not observed even in a single case, contrary to the findings of Siwach et al.10 and Arora et al.15 Further, Hendricks et al.21 in contrast to our findings, did not observe any changes in suprarenal glands, kidneys and stomach on gross examination.

A striking feature about microscopic changes due to Aluminium phosphide poisoning is that all vital organs

showed congestion (100%). Our findings were in conformity with those of Singh et al22 who also observed congestion in all organs. Brain exhibited degeneration in 60.9 % cases. Liver showed a variety of microscopic features especially central venous congestion in 92.4%, degeneration 73.9% and hemorrhage 56.6%. Changes present in the liver included alveolar thickening 98.11%, oedema 92.4%, and dilated capillaries 81.1% etc. Kidneys showed tubular degeneration and necrosis 73.6%, infiltration 62.3% and no regeneration whereas changes in the stomach included hemorrhages in 35.35%. In the stomach, in none of the cases oedema and necrosis of mucosa or sound infiltration was noted. Our findings related to histopathological changes were in general agreement with those of others.10,15,23 however, some of the features reported earlier could not be confirmed in this study. Tubular regeneration was not seen in any case of the kidney and no sloughing of gastric mucosa and leukocyte infiltration was observed in contrast to Siwach et al.10 Our findings were not in agreement with those of Singh et al, who on histopathology who did not report any specific change except congestion and patchy necrosis of the liver.22

In summary, in the present study, 83 cases of Aluminium phosphide poisoning deaths have been established because of left over Aluminium phosphide tablets or due to the presence of the wrapper at the scene or voluntary disclosures by the parents or the relatives, along with other circumstantial evidences and clinical findings in respect to poison. It has been observed that Aluminium phosphide poisoning is fairly common. There is an increasing trend in prevalence of suicide attempts. Poisoning is the most prevalent method for suicide and of late, Aluminium phosphide has emerged as an agent of choice to commit suicide, especially amongst younger age group of rural background, because it is cheap, easily available and highly lethal.

Year	Total admissions	Reported poisoning cases		Total autopsies	Suspected poisoning deaths	
		No.	%	1752	No.	%
2003	13100	301	2.30		205	11.70

Table-1 Incidence of suspected poisoning cases, and poisoning deaths.

Poisoning cases				Total		Poisoning deaths					
Male		Females				Males		Females		Total	
No	%	No.	%	No.	%	No.	%	No.	%	No.	%
204	67.77	97	32.23	301	100	137	66.83	68	33.17	205	100

Table 2. Sex distribution in suspected poisoning cases and poisoning deaths.

Age group (years)	Poisoning cases		Poisoning deaths	
	No.	%	No.	%
0-10	3	1.00	2	0.98
11-20	111	36.88	35	17.07
21-30	124	41.20	83	40.49
31-40	44	14.61	37	18.05
41-50	16	5.31	22	10.73
51-60	3		13	6.34
61-70		1.00	10	4.88
71 & above			3	1.46
Total	301	100	205	100

Table 3. Showing age group involved in suspected poisoning cases and poisoning deaths.

Months	Poisoning cases		Poisoning death	
	No.	%	No.	%
Jan.	25	8.31	17	8.29
Feb.	19	6.31	11	5.37
March	31	10.30	14	6.83
April	34	11.30	20	9.76
May	28	9.30	21	10.24
June	27	8.97	20	9.76
July	21	6.98	27	13.17
Aug.	21	6.98	28	13.66
Sep.	27	8.97	15	7.32
Oct.	29	9.63	16	7.80
Nov.	18	5.98	9	4.39
Dec.	21	6.98	7	3.41
Total	301	100	205	100

Table 4. Month wise breakup of suspected poisoning cases and poisoning deaths.

Seasons	Poisoning cases		Poisoning deaths	
	No.	%	No.	%
Summer (March-June)	120	39.87	75	36.59
Rainy (July-Oct.)	98	32.56	86	41.95
Winter (Nov.-Feb.)	83	27.57	44	21.46
Total	301	100	205	100

Table 5.Seasonwise breakup of suspected poisoning cases and poisoning deaths.

Habitat	Suspected poisoning cases				Suspected poisoning deaths			
	Male	Female	Total	%	Male	Female	Total	%
Rural	136	61	196	65.12	84	42	126	61.46
Urban	56	36	92	30.56	43	22	65	31.71
Unknown	13	0	13	4.32	10	4	14	6.83
Total	204	97	301	100	137	68	205	100

Table.6: Habitat of suspected poisoning cases and poisoning deaths

Year	Poisoning deaths		Aluminium phosphide poisoning	
	No.	%	No.	%
2003	205	100	83	40.49

Table.7: Incidence of Aluminium phosphide poisoning deaths.

Liver			Lungs			Kidneys			Stomach		
Changes	No.	%	Changes	No.	%	Changes	No.	%		No.	%
Congestion	53	100	Congestion	53	100	Congestion	53	100	Congestion	53	100
Central venous congestion	49	92.45	Alveolar thickening	52	98.11	Tubular degeneration & necrosis	39	73.58	Haemorrhage	19	35.85
Degeneration	39	73.58	Oedema	49	92.45	Infiltration	33	62.26	Oedema & necrosis	0	0
Hemorrhage	30	56.60	Dilated capillaries	43	81.13	Tubular dilatation	20	37.74	Round cell infiltration	0	0
Sinusoidal dilation	29	54.72	Collapse alveoli	35	66.04	Cloudy swelling	9	16.98	Brain	No.	%
Bile stasis	26	49.06	Haemorrhage	31	58.49	Regeneration	0	0	Congestion	53	100
Centrilobular necrosis	23	43.40							Degeneration	32	60.38
Kupffer cell hyperplasia	15	28.30									
Mononuclear infiltration	13	24.53									
Fatty changes	11	20.75									

Table 8: Microscopic changes of Aluminium phosphide poisoning in different organs.

REFERENCES

1. Nandy A. Forensic toxicology. Principles of Forensic Medicine. 2nd Ed. pp. 438-54.

2. Glaister. Law relating to poisoning: Med Juris Toxicol. 1957; 10: 466-67.

3. WHO. Public health impact of pesticides used in agriculture. Geneva, WHO 1990.

4. Senanayke NPH. Mortality due to poisoning in developing agricultural country over 20 years. Hum Exp Toxicol 1995; 14: 437-45.

5. Klaassen CD. Principles of toxicology and treatment of poisoning. In: Goodman & Gilmans'. The Pharmacological Basis of Therapeutics. 10th International Ed, McGraw Hill, New York. PP 67-80.

6. Jayaram KS. Death pills from pesticides. Nature 1991; 353: 177.

7. Parikh CK. Parikh's Textbook of Medical Jurisprudence, Forensic Medicine and Toxicology. 6th Ed. CBS Publishers, New Delhi 2000; 10.41-10.49.

8. Ahuja N. Emergency Psychiatry: A Short Textbook of Psychiatry. 1999, PP. 204-05.

9. Sagar MS, Sharma R.K., Dogra TD. Analysis of changing patterns of unnatural fatalities in South Delhi (comparative study of 1977-80 and 1988-91) J Forensic Med Toxicol 1993; 10: 21-25.

10. Siwach SB et al. Acute Aluminum phosphide poisoning: An epidemiological, clinical and histopathological study. J API 1988; 36: 594-96.

11. Sinha US, Kapoor AK. et al. A Profile of poisoning cases admitted in SRN Hospital, Allahabad. Proceedings of the International Conference and Workshop on Recent Advances in Medical Toxicology, Legal medicine, Forensic cytology, Forensic histopathology; 1998.

12. Katira R, Elhence GP et al. A study of Aluminium phosphide poisoning with special reference to electrocardiographic changes. JAPI 1983; 38: 471-73.

13. Chugh SN et al. Incidence and outcome of aluminum phosphide poisoning in a hospital study. I J M R 1991; 94: 232-35.

14. Siwach SB, Singh H, Jagdish, Katyal VK, Bhardwaj G. Cardiac arrhythmias in aluminium phosphide poisoning studied on continuous holter and cardioscopic monitoring. JAPI 1998; 46: 598-601.

15. Srivastava A, Peshin SS, Kaleekal T, Gupta SK. An epidemiological study of poisoning cases reported to the National Poisons Information Centre, All India Institute of Medical Sciences, New Delhi. Hum Exp Toxicol 2005; 24: 279-85.

16. Arora B, Punia RS, Kalra R. Histopathological changes in aluminium phosphide poisoning. J IMA1994; 93:380-81.

17.Sinha US, Kapoor AK, Pandey SK. Pattern of homicidal deaths in SRN Hospital mortuary at Allahabad. J. Forensic Med Toxicol 2003; 20: 33-36.

18.Sheikh I, Subramanyam BV. Study of homicide in Surat with special reference to changing trends. J Forensic Med Toxicol 1995; 12: 5-8.

19.Aggarwal BK, Aggarwal MP, Jain S. Aluminum phosphide poisoning: a study of 40 cases. JAPI 1989; 37: 66-68.

20.Singh D, Jit I, Tyagi S. Changing trends in acute poisoning in Chandigarh zone: a 25 years autopsy experience from a tertiary care hospital in Northern India. Am J Forensic Med Pathol 1999; 20: 203-10.

21.Hendriecks A et al. A double fatality with children due to fumigant wheat. Eur J Toxicol 1976; 9: 113-18.

22.Singh S, Singh D, Wig N, Jit I, Sharma BK. Aluminium phosphide ingestion--a clinicopathologic study. J Clin Toxicol 1996; 34: 703-06.

23.Siwach SB, Das A et al. Tissue magnesium content and histopathological changes in no survivors of aluminum phosphide poisoning. JAPI 1994; 43: 676-78.