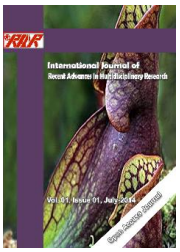

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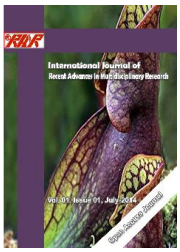

Research Article

A STUDY OF HISTOPATHOLOGICAL CHANGES IN VISCERA IN ALUMINIUM PHOSPHIDE POISONING

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ABSTRACT

A prospective study to observe thehistopathological findings in Aluminium PhosphidePoisoning was undertaken on 70 autopsy cases conducted at S. S. Medical College, Rewaand associated Gandhi Memorial Hospital. Aluminium Phosphide poisoning hasbecome the commonest poisoning in central India over the last few years. Aluminium Phosphide is a solid fumigant bulk grain preservative and is available in the market under various trade names like Celphos, Quickphos, Phostoxin, Phostek, Gastoxin, Zedesa, Li Fumesets. It is marketed either in Tablet or pellet form. Mortality is high as mechanism of action is not clearly understood and no specific antidote is available against this poison. Samples of tissues fromliver, lungs, kidneys and spleen were preserved in 10% Formal Saline, processed for histopathology, stained by HaematoxylinandEosin method and studied microscopically. Observations made in similar previous studies are compared with findings of present study and discussed.

INTRODUCTION

Aluminum phosphide has been used as a pesticide since 1940s (Jain *et al*., 2005). It is a lethal poison. The incidence of the poisoning has been increasing steadily and it is now the commonest poisoning in Northern and Central regions of the country (Jain *et al*., 2005; Siwach *et al*., 1988; Singh Dalbir *et al*., 1985; Bajaj and Wasir, 1988; Saraswat *et al*., 1985 and Ram 1988). Isolated accidental cases of fatal exposure of phosphine gas liberated from Aluminum phosphide have been reported in the literature in 1967 and 1980 from bulk shipment of wheat using Aluminum Phosphide as pesticide. (Ziipf *et al*., 1967 Wilson *et al*., 1980). In India, this poisoning was not known before 1980. The first case in India was reported in 1981 from M.G.M. Medical College, Indore. (Kabra and Narayanan, 1988). In our center, over the last twenty years, Aluminium phosphide poisoning remains major cause of mortality of patients brought to our tertiary care level hospital. Present study was undertaken keeping the magnitude of this poisoning in mind.

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MATERIALS AND METHODS

In the present study, 70 medicolegal autopsy cases of Aluminium phosphide were included. Detailed and accurate history regarding the freshness of Tablets and quantity of ingestion was obtained. Viscera were preserved for Chemicalanalysis in saturated solution of common salt.Small tissues from lungs, spleen, liver and kidneys were taken during autopsy and preserved in 10% Formol Saline solution for further processing. Histopathological examination of lungs, spleen, liver and kidneys was doneby staining the slides with H and E staining.

Observations

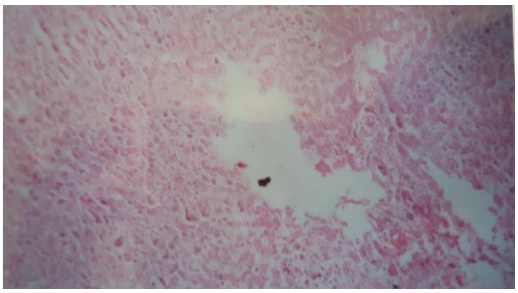
Table1. Showing age incidence of cases

Age Groups (years)	No of males	No of females	Total numbers
10 - 20	1	3	24 (34.2%)
21 - 30	5	7	32 (45.7%)
31 - 40	1	6	7 (10%)
41 - 50	2	4	6 (8.5%)
51 - 60	0	1	1 (1.4%)
Total	29 (41.4%)	41 (58.5%)	70

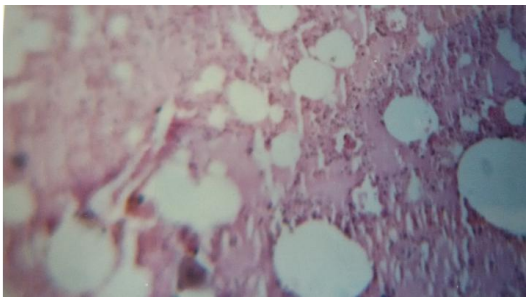
Table 2. Showing number of Tablets of Aluminium Phosphide consumed

No of Tablets	No of cases	Percentage
½	2	2.8%
1	35	50%
2	19	27.1%
3	10	14.2%
4	3	4.2%
5	1	1.4%

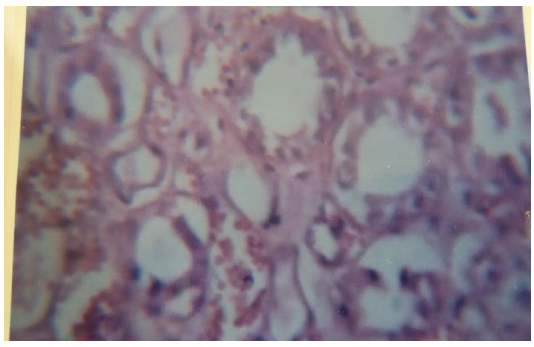
(as per history 68 cases consumed unexposed Tablets- 02 shows cases consumed exposed Tablets)



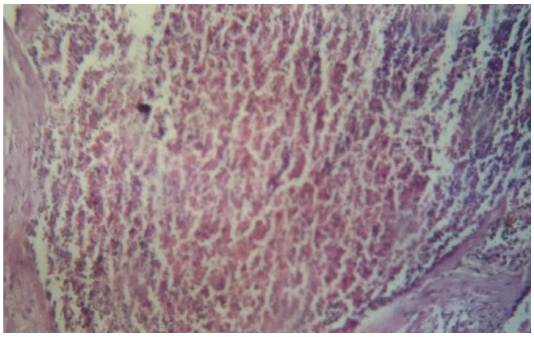
H and E stained section from Liver showing necrosis of Liver cells around Portal tract. Magnification Low power 6x10



H and E stained section from Lung showing interstitial and alveolar edema with congestion. Magnification low power 6x10



H and E stained section from Kidney showing congestion, tubular degeneration and necrosis Magnification High power 6x40



H and E stained section from Spleen showing sinusoidal congestion. Magnification low power 6x10

DISCUSSION

Incidence of Aluminium phosphide poisoning has been rising steadily ever since first case was detected in the year 1980. It is marketed in India asTablets of Celphos, Quickphos, etc. It is available insmall and large packs containing grayish-whiteTablets weighing about 3 gmseach, containing 56%Aluminium phosphide and 44% aluminiumcarbonate, capable of releasing 1 gm of phosphine. (Jain *et al*., 2005). Fatal dose of Aluminium phosphide is stated to bein the range of 150-500 mg/70 Kg. (Chugh *et al.,* 1988) Mortality rate in clinical reports is stated tovary between 37-100% by different authors. (Siwach *et al*., 1988; Saraswat *et al*., 1985; Ram *et al*., 1988; Kabra *et al*., 1988; Sepaha *et al*., 1985; Chopra *et al*., 1986; Siwach *et al*., 1994; Chugh *et al*., 1991; Chugh, 1992; Chugh, 1995; Khosla *et al*., 1986 and Mishra *et al*., 1989). First study of series of this poisoning wasundertaken by S. Singh *et al.,* 1982-83 and included 6 medicolegal autopsies with limited histopathological study. A series of 80 medico-legalautopsies of the poisoning were studied by Siwach *et al* 1985-86 and histopathological cases. In thepresent study, 70 medicolegal autopsies of Aluminium phosphide poisoning were studied andhistopathological examination was done inall the cases (Table 1). Maximum incidence of 45.7% was found in the age group of 20-30 years. Similar high incidence in this age group was reported by Khosla *et al*. (1988) 80%, Katira *et al*. (1990) and Chug *et al*. (1991) 40.22%.

High incidence in females similar to present study (58.5%) is reported by Rastogi *et al.* (1989), Puranim *et al.,* 1989 and Singh *et al.,* 1990. This is in contrast to higher incidence in males reported by Singh Dalbir *et al*. (1985), Siwach *et al*. (1988), Khosla *et al*. (1988), Chug *et al*. (1989) and Siwach *et al*. (1995). Majority of cases reported in present study had consumed unexposed Tablets (97.1%). Similar observation has been made by Siwach *et al*. (1988) and Gupta *et al*. (1995). Present study reports that condition of Tablets consumed (exposed or unexposed) is more important than the number of Tablets consumed with regards to mortality. Deaths are caused by even half unexposed Tablets. This fact is also supported by various studies such as ½ -3 Tablets by Singh Dalbir *et al*., 1985, ½ -2 Tablets by Siwach *et al*. (1988), ½ -6 Tablets by Chug *et al*. (1989), 1.5 -3 Tablets by Chug *et al*. (1991) and 1-5 Tablets by Siwach *et al*. (1997).

The manner of death was reported to besuicidal in100% of the cases by Jain *et al*. (2005), 87% of the cases by Singh Dalbir *et al*., 198576% in a study by Chugh *et al* in a clinical study on 418 patients of this poisoning. It was stated by Jain *et al*. (2005), that the survivaltime after ingestion of Aluminium phosphideingestion depended mainly on the availability of themedical facility. Average survival time inhospitalized cases (56%) was 12.8 hours incontrast to 2.6 hours in non-hospitalized cases (44%). On external examinationduring autopsy, face was reported to be livid in 39 cases, out of which 11 showed a distinct bluishdiscoloration. Garlicky pungent od our wasperceived close to the body in 50% of the cases. Froth around the mouth and/or nose was reported in 72% of the cases. They reported that typical odour of Aluminium phosphide wasnoticed when the lungs were sectioned in 56% ofthe cases.

Table 3. Showing Comparison of Histopathological findings of various organs in the present study with the previous studies on cases with Aluminium Phosphide poisoning

S.No.	Findings	Present study (1998-99) N= 70	A K Jain et al (1998-99) N= 50	Dalbir Singh et al (1989-94) N=25	Siwach et al (1985-86) N=25	S. Singh et al (1982-83) N=6	Katira et al 1989 N=57	Chugh et al 1990 N=15	Siwach et al 1994 N=30
Liver	Congestion	100%	88%	98%	100%	-	-	100%	72%
	Mild fatty infiltration	23%	38%	16%	100%	50%	-	Some	64%
	Centrizonal necrosis	18%	20%	40%	100%	17%	-	Some	8%
	Cirrhosis	1.5%	-	-	-	-	-	-	-
	Peripheral Necrosis	15%	-	-	-	-	-	-	-
	Small granuloma	-	-	-	-	17%	-	-	-
	Marked sinusoidal dilatation, Portal triaditis& focal necrosis	-					77%	-	-
	Edema	-	-	-	-	-	-	100%	-
	Diffuse sinusoidal infiltration by acute inflammatory cells	-	-	-	-	-	-	-	64%
	Portal triad infiltration by Round cells	-	-	-	-	-	-	-	40%
Kidney	Congestion	100%	100%	97%	76%	-			
	Necrosis, degeneration and regeneration of tubular epithelium	78%	78%	-	76%	17%			
Lungs	Congestion	100%	100%	99%	100%	34%	74%	100%	
	Interstitial & alveolar edema	100%	92%	48%	100%	50%	74%	100%	35%
	Thickening of alveoli by haemolysed RBC and dilated capillaries	54%	54%	-	100%	-	74%	100%	
	Red hepatisation	12%	18%	-	100%	-	-	40%	
	Round cell infiltration	-	12%	-	100%	-	74	100%	
Spleen	Congestion	82%	82%	100%	-	-			
	Necrosis	32%	40%	-	-	-			

Comparison of different histopathological changes in various organs in aluminium phasphide poisioning by various auther

Grey to greyish brown fluid orpasty material was seen in the gastric cavity in 56% cases. They reported that the mucosa of the stomach was relativelypale in cases, which were not hospitalized. Sloughing ofthe gastric mucosa was reported to be more common in the fundalregion and fundal thinning was reported in 72% of the cases. Theypostulated it to be due to the vapors of phosphine, which rise and get accumulated in the fundal regioncausing marked mucosal sloughing in this region. Authors recommend further elaborate clinical and autopsy studiesto understand theactual mechanism of action and toassess and formulate an appropriate treatment strategy forthese cases based on such studies.

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