PMID: 10758265 [PubMed - as supplied by publisher]

25: Indian Pediatr. 1999 Nov;36(11):1161-3. Related Articles, Links

Pleural effusion-a rare complication of aluminium phosphide poisoning.

Suman RL, Savani M.

Department of Pediatrics, Bal Chikitsalya, R.N.T. Medical College, Udaipur, Rajasthan, India.

Publication Types:
- Case Reports

PMID: 10745342 [PubMed - indexed for MEDLINE]

26: J Anal Toxicol. 2000 Mar;24(2):90-2 Related Articles, Links

Fatal aluminum phosphide poisoning.

Anger F, Paysant F, Brousse F, Le Normand I, Develay P, Gaillard Y, Baert A, Le Gueut MA, Pepin G, Anger JP.

Laboratoire de Toxicologie Pharmaceutique, U.F.R. des Sciences Medicales et Pharmaceutiques, Rennes, France.

A 39-year-old man committed suicide by ingestion of aluminum phosphide, a potent mole pesticide, which was available at the victim's workplace. The judicial authority ordered an autopsy, which ruled out any other cause of death. The victim was discovered 10 days after the ingestion of the pesticide. When aluminum phosphide comes into contact with humidity, it releases large quantities of hydrogen phosphine (PH3), a very toxic gas. Macroscopic examination during the autopsy revealed a very important asphyxia syndrome with major visceral congestion. Blood, urine, liver, kidney, adrenal, and heart samples were analyzed. Phosphine gas was absent in the blood and urine but present in the brain (94 mL/g), the liver (24 mL/g), and the kidneys (41 mL/g). High levels of phosphorus were found in the blood (76.3 mg/L) and liver (8.22 mg/g). Aluminum concentrations were very high in the blood (1.54 mg/L), brain (36 microg/g), and liver (75 microg/g) compared to the usual published values. Microscopic examination revealed congestion of all the organs studied and obvious asphyxia lesions in the pulmonary parenchyma. All these results confirmed a diagnosis of poisoning by aluminum phosphide. This report points out that this type of poisoning is rare and that hydrogen phosphine is very toxic. The phosphorus and aluminum concentrations observed and their distribution in the different viscera are discussed in relation to data in the literature.

Publication Types:
- Case Reports

PMID: 10732945 [PubMed - indexed for MEDLINE]

ELSEVIER
FULL-TEXT ARTICLE

Phosphine-induced oxidative damage in rats: attenuation by melatonin.

Hsu C, Han B, Liu M, Yeh C, Casida JE.

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Phosphine (PH(3)), from hydrolysis of aluminum, magnesium and zinc phosphide, is an insecticide and rodenticide. Earlier observations on PH(3)-poisoned insects, mammals and a mammalian cell line led to the proposed involvement of oxidative damage in the toxic mechanism. This investigation focused on PH(3)-induced oxidative damage in rats and antioxidants as candidate protective agents. Male Wistar rats were treated ip with PH(3) at 2 mg/kg. Thirty min later the brain, liver, and lung were analyzed for glutathione (GSH) levels and lipid peroxidation (as malondialdehyde and 4-hydroxyalkenals) and brain and lung for 8-hydroxydeoxyguanosine (8-OH-dGuo) in DNA. PH(3) caused a significant decrease in GSH concentration and elevation in lipid peroxidation in brain (36-42%), lung (32-38%) and liver (19-25%) and significant increase of 8-OH-dGuo in DNA of brain (70%) and liver (39%). Antioxidants administered ip 30 min before PH(3) were melatonin, vitamin C, and beta-carotene at 10, 30, and 6 mg/kg, respectively. The PH(3)-induced changes were significantly or completely blocked by melatonin while vitamin C and beta-carotene were less effective or inactive. These findings establish that PH(3) induces and melatonin protects against oxidative damage in the brain, lung and liver of rats and suggest the involvement of reactive oxygen species in the genotoxicity of PH(3).

PMID: 10719245 [PubMed - indexed for MEDLINE]

Fumigant-related illnesses: Washington State's five-year experience.

Burgess JL, Morrissey B, Keifer MC, Robertson WO.

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OBJECTIVE: Exposure to fumigants may have severe or persistent health effects. Washington State's fumigant-related illnesses were reviewed to better understand the circumstances surrounding exposure and resultant health effects. METHODS: Fumigant-related illnesses reported to and investigated by the Washington State Department of Health were reviewed. Illnesses considered by Department of Health to be definitely, probably, or possibly related to pesticide exposure were then analyzed. RESULTS: From 1992-1996, 39 (3.3%) of 1192 definite, probable, or possible cases of pesticide-related illnesses involved exposures to fumigants. Fumigant exposures during this period were to aluminum phosphide (15), methyl bromide (12), metam-sodium (9), and zinc phosphide (3). Symptoms included respiratory problems and eye and/or skin irritation for the majority of exposures, and no deaths were reported. The nature of exposure for these cases included exposure to applicators (17), reentry into a fumigated structure (9), improper storage or disposal (6), reentry into treated agricultural fields (4), drift from treated fields (2), and other (1). CONCLUSIONS: Review of fumigant exposures should be used to prevent future events through continued enforcement of established regulations and training of applicators.

Publication Types:
- Case Reports

PMID: 10696918 [PubMed - indexed for MEDLINE]

29: Am J Emerg Med. 1999 Sep;17(5):488-9. Related Articles, Links

Intravascular hemolysis in aluminium phosphide poisoning.

Aggarwal P, Handa R, Wig N, Biswas A, Saxena R, Wali JP.

Department of Emergency Medicine, All India Institute of Medical Sciences, New Delhi.

Intravascular hemolysis is most often secondary to exposure to a variety of drugs or infections, and usually occurs in patients who are deficient in glucose-6-phosphate dehydrogenase (G-6-PD) enzyme. Aluminium phosphide, a fumigant widely used in India, has been reported to produce intravascular hemolysis in only one patient who also had concomitant G-6-PD deficiency. This report describes the occurrence of intravascular hemolysis with aluminium phosphide poisoning in a patient with normal G-6-PD levels. This is of significance as jaundice in patients with this poisoning is often attributed to hepatic damage alone.

Publication Types:
- Case Reports

PMID: 10496516 [PubMed - indexed for MEDLINE]

30: Am J Forensic Med Pathol. 1999 Jun;20(2):203-10. Related Articles, Links

Changing trends in acute poisoning in Chandigarh zone: a 25-year autopsy experience from a tertiary care hospital in northern India.

Singh D, Jit I, Tyagi S.

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A 25-year autopsy study (1972-1997) of acute poisoning deaths from a tertiary care hospital in northern India (Postgraduate Institute of Medical Education and Research, Chandigarh) revealed a steep increase in the incidence of acute poisoning since 1987. The majority (68%) of subjects were between the ages of 14 and 30 years, and there was a male preponderance (69%). The main victims were students and unemployed youths, followed by agricultural workers and domestic workers. The proportion of urban victims increased from 45% in the period from 1972 to 1977 to 72% in the period from 1992 to 1997. The proportion of suicidal deaths increased from 34% in the period from 1972 to 1977 to 77% in the period from 1992 to 1997, whereas accidental deaths decreased from 63% to 17% in the same period. Barbiturates (37%) and copper sulfate (22%) were the most common poisons causing mortality between 1972 and 1977; organophosphates (46%) became the most common between 1977 and 1982. Since 1982, aluminum phosphide (65%) has been the most common poison.

PMID: 10414665 [PubMed - indexed for MEDLINE]

31: Am J Emerg Med. 1999 Mar;17(2):219-20. Related Articles, Links

Treatment of the cardiovascular manifestations of phosphine poisoning with trimetazidine, a new antiischemic drug.

Duenas A, Perez-Castrillon JL, Cobos MA, Herreros V.

Publication Types:
- Case Reports
- Letter

PMID: 10102341 [PubMed - indexed for MEDLINE]

32: Vet Hum Toxicol. 1999 Feb;41(1):31-2 Related Articles, Links

Effect of aluminum phosphide on blood glucose level.

Abder-Rahman H.

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Aluminum phosphide (AlP), a poison extensively used as a grain fumigant and rodenticide, can cause an increase or decrease in blood glucose levels Both hypo- and hyper-glycemic effects of AlP can be attributed to the wide variety of changes in magnesium, calcium, phosphate, citrate and cortisol levels. These biochemical changes can act as active stimulatory or inhibitory modulators to enzymes and hormones that catalyze and regulate glucose metabolism. According to the type of biochemical changes, AlP can cause either elevation, decrease or no change in blood glucose levels. A case of AlP-caused death is reported.

Publication Types:
- Case Reports

PMID: 9949483 [PubMed - indexed for MEDLINE]

33: Toxicol Sci. 1998 Nov;46(1):204-10. Related Articles, Links

Phosphine-induced oxidative stress in Hepa 1c1c7 cells.

Hsu CH, Quistad GB, Casida JE.

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Phosphine (PH3), from hydrolysis of metal phosphides, is an important insecticide (aluminum phosphide) and rodenticide (zinc phosphide) and is considered genotoxic and cytotoxic in mammals. This study tests the hypothesis that PH3-induced

genotoxicity and cytotoxicity are associated with oxidative stress by examining liver (Hepa 1c1c7) cells for possible relationships among cell death, increases in reactive oxygen species (ROS) and lipid peroxidation, and elevated 8-hydroxyguanine (8-OH-Gua) in DNA. PH3 was generated from 0.5 mM magnesium phosphide (Mg3P2) to give 1 mM PH3 as the nominal and maximal concentration. This level causes 31% cell death at 6 h, measured by lactate dehydrogenase leakage, with appropriate dependence on concentration and time. The intracellular ROS level is elevated within 0.5 h following exposure to PH3, peaking at 235% of the control by about 1 h. Lipid peroxidation (measured as malondialdehyde plus 4-hydroxyalkenals) is increased up to 504% by PH3 at 6 h in a time-dependent manner. The level of 8-OH-Gua in DNA, a biomarker of mutagenic oxidative DNA damage analyzed by GC/MS, increases to 259% at 6 h after PH3 treatment. Antioxidants significantly attenuate the PH3-induced ROS formation, lipid peroxidation, 8-OH-Gua formation in DNA, and cell death, with the general order for effectiveness of GSH (5 mM) and D-mannitol (10 mM) (hydroxyl radical scavengers), then Tempol (2.5 mM) and sodium azide (3 mM) (superoxide anion and singlet oxygen scavengers, respectively). These studies support the hypothesis that PH3-induced mutagenic and cytotoxic effects are due to increased ROS levels, probably hydroxyl radicals, initiating oxidative damage.

PMID: 9928684 [PubMed - indexed for MEDLINE]

34: J AOAC Int. 1998 Nov-Dec;81(6):1190-201. Related Articles, Link

Determination of phosphine residues in whole grains and soybeans by ion chromatography via conversion to phosphate.

Carlson M, Thompson RD.

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An ion chromatographic (IC) method was developed for determining phosphine (PH3) in whole grains (barley, corn, oats, rice, rye, and wheat) and soybeans. The method converts phosphine to phosphate (i.e., orthophosphate) and isolates the phosphate by IC with eluent-suppressed conductivity detection. Recoveries of unbound phosphine by the method were similar to those obtained by an established colorimetric method for 7 different products fortified at 3 levels. Mean recoveries were low (i.e., 30-60%) and varied with product type and level of fortification. Recoveries of PH3 from previously fumigated products fortified with aluminum phosphide ranged from 19.0% for barley fortified at 0.734 ppm to 88.3% for corn fortified at 1.691 ppm. Precision data from 3 products based on replicate analyses (n = 4 or 5) gave relative standard deviations of 1.78-4.66% for mean laboratory-fumigated PH3 levels of 0.679-1.309 ppm. Estimated limits of detection (LOD) and quantitation (LOQ) for PH3 were 0.010 microgram/g (10 ppb) and 0.0275 microgram/g (27.5 ppb) at signal-to-noise ratios (S/N) of 4:1 and 10:1, respectively. These values were also determined for a nonchemically suppressed IC system with LOD of 0.02 microgram/g (20 ppb) and LOQ of 0.055 microgram/g (55 ppb) at S/N of 4:1 and 10:1, respectively. Phosphate response was linear over the concentration range equivalent to 0.30-10.0 micrograms P/mL, with a mean correlation coefficient of 0.9988 based on replicate standard curves. The relationship of product composition to recovery from various products was also examined.

PMID: 9850582 [PubMed - indexed for MEDLINE]

A critical evaluation of anti-peroxidant effect of intravenous magnesium in acute aluminium phosphide poisoning.

Chugh SN, Kolley T, Kakkar R, Chugh K, Sharma A.

Department of Medicine and Biochemistry, Postgraduate Institute of Medical Sciences, Rohtak, Haryana, India.

The anti-peroxidant effect of intravenous magnesium was evaluated in 50 patients with acute aluminium phosphide poisoning. The patients were divided into two groups, one who received magnesium sulphate therapy (Group I) and the other who did not (Group II). The clinical and biochemical parameters in both groups were comparable. Finding of increased mean malonyl-di-aldehyde (MDA) levels in group I (3.18 +/- 0.93 micromol/L) and group II (3.15 +/- 0.78 mmol/L) combined with low blood levels of reduced glutathione (18.5 +/- 1.6 mg/dl in group I) and (17.8 +/- 1.4 mg/dl in group II) indicated oxidative stress leading to accelerated lipid peroxidation in the early phase (0-6 h) of AlP poisoning. A significant fall in MDA levels was observed after 2 h in the magnesium treated group (group I) compared to the non-treated group (group II) and levels became normal between 48-72 h. Similarly reduced glutathione started recovering between 12-24 h which became significant after 24 h and full recovery took place between 48-72 h in the magnesium treated group (group I). Both these parameters suggested an anti-peroxidant effect of magnesium. There was also a slight fall in MDA levels and a rise in reduced glutathione in the non-treated group II patients. This could be due to elimination of phosphine (PH3). We hypothesize that oxidative stress in AlP poisoning buffered the magnesium leading to a transient fall in magnesium and magnesium dependent GSH, resulting in increased susceptibility of oxygen free radical injury and accelerated lipid peroxidation. The fall in MDA and slower rise in GSH in group I than in group II suggested magnesium combated free radical stress slowly and independent of elimination of phosphine. This hypothesis was further strengthened by similar observations when both these parameters were compared in survivors in both groups. Mortality was higher in group II than in group I (44 per cent vs 20 per cent) and was probably related directly to oxidative stress.

Publication Types:
- Clinical Trial
- Controlled Clinical Trial

PMID: 9483483 [PubMed - indexed for MEDLINE]

An experimental study on cardiotoxicity of aluminium phosphide.

Lall SB, Sinha K, Mittra S, Seth SD.

Department of Pharmacology, All India Institute of Medical Sciences, Ansari Nagar, New Delhi, India.

Aluminium phosphide(AlP), a grain fumigant pesticide, was studied for its cardiotoxicity in anaesthetised rats. The hemodynamic and cardiac biochemical changes were investigated following intragastric administration of different doses of AlP (10, 20 and 40 mg). With 10 and 20 mg dose of AlP an immediate fall in BP was observed which recovered partially and stabilized for 10 minutes followed by a gradual fall till

the animal died. However, with a higher dose (40 mg) there was no recovery in BP, instead the initial fall continued till the death of the animal. An increase in the heart rate was observed with 10 and 20 mg dose of AlP for 15 minutes which was followed by a marked fall till cardiac arrest ensued. On the other hand, 40 mg dose produced only a transient tachycardia followed by a prolonged bradycardia. ECG changes at all dose levels included initial tachycardia and ST segment elevation progressing to QRS broadening. However, marked conduction defects as evidenced by the ventricular ectopics were noticed only with 40 mg. The mean survival time dose dependently decreased with 10 mg(55 +/- 3 min), 20 mg(35 +/- 2 min) and 40 mg(18 +/- 2 min) of AlP. The cardiac glycogen, ATP and CP levels were significantly lowered in animals treated with 10, 20 and 40 mg of AlP. Higher levels of MDA in the cardiac tissue were observed with 10, 20 and 40 mg of AlP. Thus it is suggested that the deleterious effect of AlP on heart is mediated by both declined cellular metabolism of the myocardium as well as by necrosis of the cardiac tissue resulting in the release of reactive oxygen intermediates.

PMID: 9475040 [PubMed - indexed for MEDLINE]

37: Indian Pediatr. 1997 Jul;34(7):650-1 Related Articles, Links

Aluminium phosphide poisoning: a growing concern in pediatric population.

Singh UK, Chakraborty B, Prasad R.

Publication Types:
- Letter

PMID: 9401264 [PubMed - indexed for MEDLINE]

38: Indian J Physiol Pharmacol. 1997 Apr;41(2):189. Related Articles, Links

Need for antidote for aluminium phosphide poisoning.

Yadav J.

Publication Types:
- Letter

PMID: 9142571 [PubMed - indexed for MEDLINE]

39: J R Soc Med. 1997 Jan;90(1):47-8 R ated Articles iks

Intravascular haemolysis after aluminium phosphide ingestion.

Sood AK, Mahajan A, Dua A.

Postgraduate Department of Medicine, Pandit B D Sharma Postgraduate Institute of Medical Sciences, Rohtak, Haryana, India.

Publication Types:
- Case Reports

PMID: 9059385 [PubMed - indexed for MEDLINE]

40: J Assoc Physicians India. 1996 Nov;44(11):841-2 Related Articles, Links

Comment on:
- J Assoc Physicians India. 1996 Mar;44(3):184-5

Serial blood phosphine levels in aluminium phosphide [ALP] poisoning.

Singh S.

Publication Types:
- Comment
- Letter

PMID: 9251470 [PubMed - indexed for MEDLINE]