Aluminium Phosphide Poisoning in a Warehouse Workers in Bangladesh

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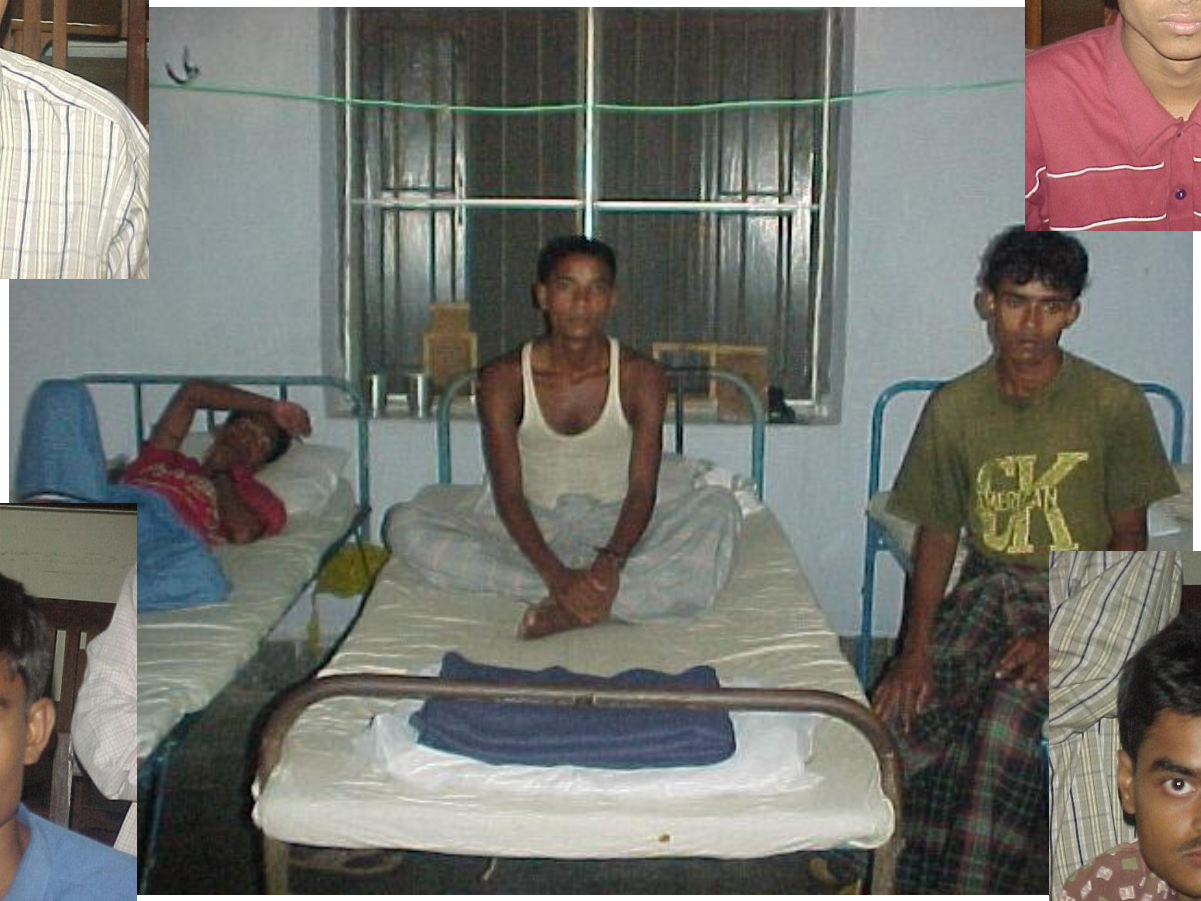
Aluminium phosphide

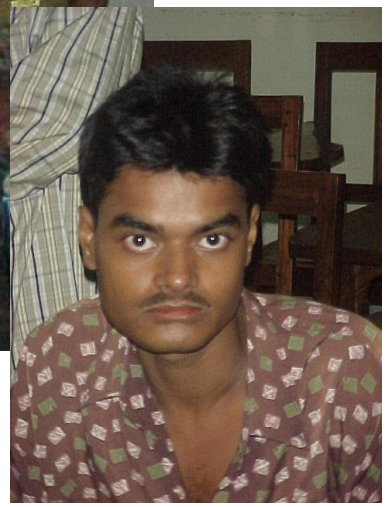
- A solid fumigant, liberates Phosphine (PH_3) on exposure to moisture
- Used as grain preservative in warehouse.
- Pesticide and rodenticide.
- Phosphine: Colourless, flammable, garlic or rotten fish odour

- Preparation: 3 gm pellets contain 57% Al ph, 43% Amm carb. Each pellete release ~1 gm phosphine gas.
- “Quickfume” “Celphos” “Alphos” “Quickphos” “Phosfume”
- Mode of poisoning: Sucidal, accidental, homicidal. Adults> child

Description of the event

- *At around 11A.M. on 2nd January 2003 six young men were rushed to an adult medicine unit of Chittagong Medical College Hospital (CMCH) with complaints of dizziness, fatigue, vertigo, ataxia following severe nausea and vomiting. They all work on contractual basis in a food warehouse in Sitakundu, Chittagong. A seventh person who was not admitted had similar mild form of symptoms. Their job is to implant fumigating pesticide pellets in between the stacked grain bags.*





Description of the event

- *Three of these men first experienced nausea and few vomiting episodes previous evening which they neglected and took no specific measures. They slept in the warehouse the previous night as they used to do. But this time they wake up in the middle of the night with feeling of uneasiness, headache and a strong pungent odour around them causing breathing difficulty. They came out of the warehouse. Fresh air eased some of their uneasiness.*

Description of the event

- *But with in a short time some of them developed severe nausea followed by repeated vomiting (>10 times in two). They were brought to a nearby clinic where they were given I.V. infusion and later on referred to CMCH.*

Name Age(yr)	Jo 18	Mi 22	Mo 30	ASA 24	Sa 28	Sh 15	Bi 28
Dizziness	+	+	+	+	+	+	+
Fatigue	+	+	+	+	+	+	+
Chest tightness	+	-	-	-	+	-	+
Headache	-	+	-	-	+	-	+
Nausea	+	+	+	+	+	+	+
Vomiting	3	5	3	> 10	> 20	8-9	4
Diarrhoea	-	-	-	-	-	-	-
Vertigo	+	+	+	+	+	-	+
Blurring of vision	-	-	-	+	+	+	-

Name Age(yr)	Jo 18	Mi 22	Mo 30	ASA 24	Sa 28	Sh 15	Bi 28
Signs							
Anaemia	-	Mild	-	Mild	-	-	-
Cyanosis	-	-	-	-	-	-	-
Resp. rate	28	20	22	34	20	22	20
Pulse/min	120	80	80	124 AF	108	88	60
BP mm Hg	90/ 50	120/ 90	95/ 60	70/ 40	100/ 70	110/ 60	130 /80
Temp (^{0}F)	101	N	N	N	101	N	N
GCS	15	15	15	15	15	15	15
Ataxia	+	+	+	+	+	-	-
Tremor	+	+	-	+	+	-	-

Name Age(yr)	Jo 18	Mi 22	Mo 30	ASA 24	Sa 28	Sh 15
Lab invest						
Electrolytes: K HCO_3	N 16	N N	3.22 N	3.4 N	N 21	3.4 N
(On 4/01/03) K	5.01	5.42	5.68	5.85	4.59	6.47
TC	12500	9500	6500	8500	8500	10000
Urea Creatinine	30.5 0.81	47.6 1.10	27.6 1.02	39.7 0.92	25.4 0.83	23.4 0.73
CPK-MB	16.0	32.0	14.0	25.0	26.0	16.0
SGOT	21	33.0	15.0	26.0	24.4	29.0
ECG	ST↓ T↓	N	N	AF	ST↓ T↓	N
Treatment	Nitromint s/l Disprin	Chol saline	5% DNS	Inj. Mg Sulph Dopamin	Chol saline Disprin	Chol saline

Name Age(yr)	Jo 18	Mi 22	Mo 30	ASA 24	Sa 28	Sh 15
Follow up 17/01/03						
Complaints and findings	Vertigo	Vertigo, Nausea	Phlebitis	Head-ache	Vertigo	Abdo minal pain
Pulse	84	88	56	72	72	56
BP	90/50	100/70	80/50	100/60	85/60	80/50
ECG	N	N	Tall T in chest leads	N	N	N

Mechanism of toxicity

- Phosphine is liberated in stomach and absorbed. Some absorbed as Al Phos metabolized in liver slow release of phosphine. Prolongation of symptoms.
- Binds to cytochrome oxidase: cause cellular hypoxia.
- Cardiotoxicity: subcellular transmembrane exchange of ions (Na, K, Mg, Ca) due to focal myocardial necrosis.
- Excretion: through lung and kidney.

Toxic dose

- Lethal at <500 mg ingestion
- Inhalation:
  - even at 7 ppm for several hrs
  - Severe toxicity: 300 ppm
  - Lethal: 400-600 ppm in 30 mints.

Clinical feature- Inhalation

- Mild exposure: Irritation of mucous memb, acute respiratory distress, dizziness, fatigue, chest tightness, nausea, vomiting, diarrhoea, headache.
- Moderate exposure: Diplopia, ataxia, tremor, paraesthesias.
- Severe exposure: ARDS, arrhythmias, convulsions, coma, death.
- Complications: Liver and renal failure

Clinical feature- Ingestion

- Mild case: Epigastric distress, vomiting, diarrhea.
- Moderate to severe case:
- Cardiovascular: Hypotension, shock, tachy or brady-cardia, CCF, Arrhythmias (AF, A/VPC, VT, VF, AV junc Tachycardia, Cond defects) ST depression, T inversion, Oedema.

Clinical feature- Ingestion

- Respiratory: Cough, dyspnoea, cyanosis, creps, ARDS, Pl effusion,
- Liver injury, ARF, Adrenocortical involvement
- CNS: Headache, dizziness, usually no change in consciousness level until late.
- Others: Metabolic acidosis, Hypo or hypermagnesaemia, hypokalaemia.

Diagnosis

- Clinical
- Circumstantial evidences
- Gastric fluid test, Breath test (Silver nitrate impregnated filter paper)
- Air analysis

Management

- No specific antidote.
- Supportive to sustain life till phosphine is liberated.
- Reduction of absorption: Gastric lavage with Pot permanganate with in 30-45 mints. Oxidises to phosphate.
- Reduction of toxicity: Magnessium sulphate infusion.
  Uncontrolled trials. Recent controversy.
  Dose: 3gm bolus, 6 gm infusion over 12 hrs for 5-7 days.

Management

- Arrhythmias: Does not respond to usual drugs. Mg Sulph, ineffective in shock.
- Enhancement of excretion: IV fluid, maintenance of normal renal function.
- Supportive: Very important, Oxygen, intubation and assisted ventilation. Management of shock.

Prognosis

- Mortality: usually due to ingestion
- 37-100% in different series
- usually in 24 hrs may occur as late as 7 days
- Inhalation cases: Only one reported death*

**Wilson R, Lovejoy FH, Jaeger RJ, Landrigan PL. Acute phosphine poisoning aboard a grain freighter. Epidemiologic, clinical, and pathological findings. JAMA. 1980 Jul 11;244(2):148-50*

Literature review

- Reported from India, Australia, France, Denmark, Poland, Italy, Jordan
- Most are from India and due to ingestion.
- Inhalation cases from Germany, USA.
- around 15,000 cases per year of AP poisoning are reported from India, two third of whom are fatal.
- Only known fatal case of AP poisoning case in Bangladesh: Cardiac, renal, hepatic involvement, hypernatremia, acidosis, haemolysis, thrombocytopenia; Died 8 days after ingestion of single pellete. *DMCH (29/08/2004; unpublished data)*

Conclusion

- Ignorance of the basic principles of personal protection of workers by the employer and the workers themselves.
- Adherence to regulations of occupational health is to be reinforced to prevent recurrence.
- Immediate recognition was difficult due to unfamiliarity of the agent to the physicians.
- ?!? change of pattern in pesticide poisoning.

Conclusion

- Necessitates the awareness of the public to the hazards of this poison.
- Education, proper handling, strict observation and abiding by the regulations controlling this material are good protective measures against AP poisoning

Acknowledgement

- Doctors and nursing staff of medicine unit 3 Chittagong Medical College Hospital, Chittagong who managed the cases.
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Thank You