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# Successful treatment of acute aluminium phosphide poisoning: possible benefit of coconut oil

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Aluminium phosphide is used to control rodents and pests in grain storage facilities. It produces phosphine gas, which is a mitochondrial poison. Unfortunately, there is no known antidote for aluminium phosphide intoxication, but our recent experience with a case showed that rapid prevention of absorption by coconut oil might be helpful. In the present case, we used the same protocol in a 28-year-old man who had ingested a lethal amount (12 g) of aluminium phosphide with suicidal intent and was admitted to hospital approximately 6 hours postingestion. The patient had signs and symptoms of severe toxicity, and his clinical course included metabolic acidosis and liver dysfunction. Treatment consisted of gastric lavage with potassium permanganate

solution, oral administration of charcoal and sorbitol suspension, intravenous administration of sodium bicarbonate, magnesium sulphate and calcium gluconate, and oral administration of sodium bicarbonate and coconut oil. Conservative and supportive therapy in the Intensive Care Unit was also provided. The patient survived following rapid treatment and supportive care. It is concluded that coconut oil has a positive clinical significance and can be added to the treatment protocol of acute aluminium phosphide poisoning in humans. *Human & Experimental Toxicology* (2005) 24, 215–218

**Key words:** coconut oil; phosphide; poisoning

## Introduction

Phosphides are used throughout the world as pesticides to protect stored grains from rodents and other pests.<sup>1–3</sup> Solid phosphides, including aluminium phosphide, form toxic phosphine gas following contact with water, moisture in the air, or hydrochloric acid in the stomach.<sup>1–5</sup> Phosphine is a colourless, flammable gas with an odour of garlic or decaying fish.<sup>1,2,6</sup>

During the past 35 years, high mortality rates have been reported following significant exposures to aluminium, zinc and calcium phosphides. Exposure is rarely accidental with the majority of cases involving intentional suicide acts. In our poison centre from 1997 to 1998, we encountered 349 fatalities among 35 580 poisoned patients over 12 years of age. Of these fatalities, nine (2.6%) cases were due to aluminium phosphide poisoning.<sup>7</sup>

During a six-month period in 1994, 48 of the 7000 patient admissions to the Loghman–Hakim Hospital Poison Center were due to aluminium phosphide or zinc phosphide poisoning.<sup>8</sup> Both accidental and intentional poisonings are occasionally reported in countries where phosphides are less readily available.<sup>9–11</sup> This should raise the attention of the physician to the problem of aluminium phosphide poisoning and also necessitates the awareness of the public to the hazards of this poison.<sup>12</sup>

Phosphine is a mitochondrial poison that interferes with enzymes and protein synthesis.<sup>13</sup> Once absorbed systemically, either following inhalation or via the gastrointestinal route, it can damage cell membranes and enzymes of the mitochondrial respiratory system. Animal studies have shown that phosphine inhibits mitochondrial cytochrome oxidase and causes fluctuations in glucose levels secondary to changes in electrolyte and hormones.<sup>14</sup> The mechanism of toxicity by phosphine gas is not completely understood, but lesions suggest direct damage to blood vessels and erythrocyte

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membranes.<sup>1,15</sup> Phosphine causes noncompetitive inhibition of the cytochrome oxidase in myocardial mitochondria<sup>3,13</sup> and also inhibits the incorporation of amino acids into myocardial proteins.<sup>6</sup> These alterations in myocardial mitochondria and proteins produce permeability disturbances to sodium, potassium, magnesium, calcium and other ions, causing changes in transmembrane action potentials.<sup>16</sup> These changes are much more prevalent in the myocardium, small peripheral blood vessels and pulmonary cells. Pulmonary oedema is believed to result from direct cytotoxicity to the pulmonary cells.<sup>6</sup> Many cases of acute deliberate zinc or aluminium phosphide poisoning by ingestion have been reported in the literature as reviewed by the International Program on Chemical Safety (IPCS). In most of the lethal cases, the ingested dose was 4.5–180 g, while in minor cases the ingested dose was 20 g or more. In the 10 nonfatal cases, the doses ranged from 0.5 to 50 g and even less than 20 g in minor cases. The main clinical manifestations were metabolic acidosis, methaemoglobinaemia, hypocalcaemic tetani, reduced blood coagulation, pulmonary oedema and gastrointestinal, neuropsychiatric and cardiovascular disorders. Post-mortem findings included blood in all the serous cavities, pulmonary congestion and oedema, haemorrhagic changes in the intestinal epithelium, centrilobular congestion and necrosis and yellow discoloration of the liver, and patchy necrosis of the proximal convoluted tubules of the kidneys.

The main recommendation in most reported cases is that early recognition and treatment of phosphine/phosphide poisoning is of great importance and treatment of shock and metabolic acidosis together with the intensive care therapy of the cardiopulmonary effects are essential. Early vomiting is thought to improve the prognosis.<sup>17</sup> Our recent experience of a 47-year-old woman who had ingested 16.8 g of aluminium phosphide and expected to have a fatal outcome, showed that early gastric lavage with potassium permanganate solution, oral sodium bicarbonate solution, activated charcoal, oral coconut oil, intravenous magnesium and intravenous calcium contributed to survival.<sup>18</sup> Therefore we tried to manage the present case with that protocol.

### Case report

A 28-year-old man was admitted to our poison centre approximately 6 hours after ingestion of aluminium phosphide. According to interviews with the patient and his relatives, he had taken seven pellets of aluminium phosphide with suicidal

intent. Each pellet weighed 3 g and contained 56% aluminium phosphide to give a total dose of 11.76 g.

The patient had a Glasgow Coma Score of 14-15/15, his blood pressure was 90/50 mmHg and his peripheral pulses were about 115/min. He complained from severe headache, malaise and dizziness.

In the emergency room, the patient was given gastric lavage with potassium permanganate (1:10 000 solution), followed by oral administration of activated charcoal (1 g/kg of 30% suspension) and sorbitol (1 mL/kg of 70% suspension). The patient was subsequently admitted to the Intensive Care Unit for cardiovascular and respiratory support.

On the day of hospital admission the patient experienced abdominal pain, thirst, agitation and anxiety. He was treated with the following intravenous drugs: sodium bicarbonate (8.4%, 100 mEq every hour for metabolic acidosis, Table 2); magnesium sulphate (20%, 20 mL every six hours because of antioxidant effect of magnesium); calcium gluconate (10%, 10 mL every six hours because of its membrane stabilizing effects); and ranitidine (50 mg every 12 hours to suppress stomach acid secretion). Activated charcoal (0.5 g/kg of 30% suspension every three hours), and coconut oil (200 mL) plus sodium bicarbonate (8.4%, 50 mL) every two hours) were administered via nasogastric tube. Administration of the charcoal and sorbitol suspension was ceased after 24 hours, while the other treatments continued for approximately 72–144 hours. No arrhythmias were detected. Serum magnesium levels could not be measured on admission and before beginning of magnesium sulphate 20%, as our hospital laboratory could not provide this measurement as an emergency test.

On day 2 of hospital admission, the patient's liver enzymes and bilirubin started to elevate and reach peak levels on day 5 (Table 1). In response, lactulose syrup (30 mL every 6 hours) was administered orally.

On day 3 of hospital admission, nasogastric feeding was initiated and serum intake was decreased. The patient's liver function tests began to improve on day 6 of admission (Table 1). Conservative treatment and supportive therapy was continued during hospitalization. The patient was discharged from the hospital 8 days after poisoning and followed up.

### Discussion

Poisoning by suicidal or accidental ingestion of aluminium phosphide is a frequent medical emergency seen all over the world. On exposure to

**Table 1** Biochemistry and liver function tests

Parameter (normal range)	Days after hospital admission						
	0	1	2	3	5	6	7
Blood glucose (70–110 mg/dL)	143*	NM	131*	NM	226*	105	152*
Urea (8–25 mg/dL)	17	NM	38*	NM	38*	22	25
Creatinine (0.5–1.7 mg/dL)	1.1	NM	1.3	NM	1	0.6	0.9
Sodium (135–145 mEq/L)	140	NM	131*	NM	140	140	141
Potassium (3.5–4.5 mEq/L)	4.5	NM	3.5	NM	5.3*	4.6*	3.6
Calcium (8.5–10.5 mg/dL)	7.5*	NM	8.5	NM	NM	NM	8.6
Phosphate (2.5–4.5 mg/dL)	1.3*	NM	2.4*	NM	NM	NM	2.6
Total bilirubin (0.2–1.3 mg/dL)	NM	NM	1.7*	NM	2.3*	1.6*	0.5
Direct bilirubin (<0.2 mg/dL)	NM	NM	0.4*	NM	0.5*	0.2	0.1
Aspartate aminotransferase (AST) (11–47 IU/L)	45	NM	51*	NM	60*	60*	25
Alanine aminotransferase (ALT)(7–53 IU/L)	51	NM	54*	NM	320*	259*	21
Alkaline phosphatase (38–126 IU/L)	123	NM	NM	NM	230*	212*	124
Prothrombin time (11–13 s)	NM	16*	NM	20*	17*	NM	13
Partial thromboplastin time (25–36 s)	NM	70*	NM	78*	40*	NM	32

NM, not measured.

\*Value is out of normal range.

There was no significant change in parameters of day 4.

moisture, aluminium phosphide liberates the highly toxic gas, phosphine. Generally in phosphine poisoning, symptoms of toxicity usually develop rapidly, sometimes within 15 min.<sup>1</sup> The majority of deaths occur within the first 12–24 hours, usually due to cardiovascular arrest.<sup>19,20</sup> Deaths occurring after 24 hours are often due to liver failure.<sup>6</sup> The current recommended treatment protocol mentioned in textbooks includes lavage with potassium permanganate (1:10 000) or initial dilution with sodium bicarbonate (3–5% solution), and administration of activated charcoal. Calcium gluconate 10% and magnesium sulphate 25% have also been advocated.<sup>1,2,6,21</sup> The main difference between the treatment protocol done in the present case and those recommended in the literature is the use of coconut oil. Coconut oil is high in saturated fatty acids and can be combined with other oils. Coconut oil has been reported to inhibit the release of phosphine gas from aluminium phosphide due to physicochemical properties of aluminium phosphide and nonmiscibility with fat.<sup>22</sup>

The acidic gastric environment enhances conversion of aluminium phosphide to phosphine, thus initial dilution with sodium bicarbonate is helpful.<sup>1,21</sup> Potassium permanganate (1:10 000 solution) also oxidizes phosphine gas in the stomach to

phosphate, thereby reducing the available amount of toxic phosphine gas.<sup>18,23–25</sup> Activated charcoal can decrease the absorption of phosphide particles, which are thought to be responsible for delayed toxic effects.<sup>6,18,26</sup> Calcium gluconate and magnesium sulphate have been used with good results, presumably due to their membrane-stabilizing effects<sup>1,2,6,18</sup> and also probably due to the antioxidant effect of magnesium.<sup>27–30</sup> Acute aluminium phosphide poisoning produces hypomagnesaemia with or without ECG changes. However, the mortality rate is significantly higher in those patients with hypomagnesaemia who have ECG changes.<sup>31</sup> A positive correlation has been demonstrated between mortality and lower magnesium concentrations in serum and red blood cells.<sup>32</sup>

Despite the scepticism expressed in some medical texts regarding the efficacy of treatment for patients who have ingested large doses of phosphides, the present case shows that effective management can result in survival and supports our previous case report.<sup>18</sup> Measures of possible benefits include early gastric lavage with potassium permanganate solution, oral administration of sodium bicarbonate, activated charcoal, coconut oil, and intravenous administration of magnesium and calcium. All of these factors, especially coconut oil, were probably

**Table 2** Arterial blood gas

Parameter (normal range)	Days after hospital admission							
	0	1	2	3	4	5	6	7
pH (7.35–7.45)	7.36	7.23	7.23	7.47	7.50	7.49	7.47	7.40
HCO <sub>3</sub> (22–26 mEq/L)	12.1	11	14	28	37	37.7	39	25
PCO <sub>2</sub> (35–45 mmHg)	30	29.6	32	35	40	40	39.6	42
PaO <sub>2</sub> (>80 mmHg)	85	86	90	88	91	89	90.5	93

important in preventing what would otherwise have been a fatal ingestion of aluminium phosphide. Attention should be paid to the fact that survival can follow the ingestion of large doses of aluminium phosphide, providing there is no delay in diagnosis

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