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An outbreak of aluminium phosphide poisoning in Mashhad, Iran

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On 6 June 2015, accidental aluminium phosphide (AlP) poisoning took four lives in the north-eastern Iranian city of Mashhad. The victims were Saudi Arabian tourists, 32 of whom survived. This event attracted massive media attention (1), as recent tension in the Middle East led to implications of a terrorist attack, which was later dismissed. According to Iran's Ministry of Interior, this accident was the result of "negligence and ignorance" (2).

In Asia, records of AlP suicide poisoning are not uncommon; AlP is cheap and available as a pesticide. It has been banned in Iran, except for specific use as rodenticide protecting stored rice and grain. Phosphine gas, as the main toxic agent, is released when phosphide comes in contact with moisture or acid (3). To the best of our knowledge, this is the first report of accidental outbreak of AlP poisoning through the air.

The aim of our investigation was to describe this poisoning outbreak from the public health emergency point of view. This report focuses on the pattern of referral and clinical findings that first responders should be aware of if they encounter an airborne AlP poisoning outbreak. The investigation followed the recommendations of the Mashhad University of Medical Sciences Ethics Committee for descriptive studies.

Case series

Thirty six Saudi tourists (mean age 15 years; range: 1-45) staying in a hotel in Mashhad, Iran were admitted to the Imam Reza University Hospital for symptoms of acute AlP poisoning. One of them died en route to the hospital. They inhaled phosphine gas liberated from AlP powder which was placed in their rooms for pest control. Twelve patients were admitted between four and five a.m., 12 between seven a.m. and one-thirty p.m., six between one-thirty and seven-thirty p.m., and the remaining six patients after that time. The mean hospitalisation time was 1.5±0.8 days (range: 0-2), including the self-discharged patients.

Four patients died before or soon (4-6 h) after admission despite all attempts at supportive treatment and were significantly younger than the surviving group (two less than one-year olds, one three-year old, and one 14-year old). Gas chromatography with a nitrogen phosphorous detector of the vomit and gastric secretions of the deceased patients confirmed phosphine exposure. The main clinical manifestation of three deceased patients on admission was shock and severe metabolic acidosis, which was refractory to treatment by volume expander sera and sodium bicarbonate with close monitoring of arterial blood gases and serum electrolyte balance. In addition to shock, the fourth deceased patient had a massive pulmonary haemorrhage due to disseminated intravascular coagulation (DIC). All deceased patients were unresponsive or barely responsive to painful stimuli on admission, whereas all surviving patients were alert on admission. We find this information potentially useful for triage and rapid intervention.

Of the 32 survivors, 21 were men and nine women (two of them pregnant). All survivors had increased heart rate (94 to 120 beats per min) on admission. Two had systolic blood pressure <90 mmHg and mild metabolic acidosis that turned back to normal on the first day in hospital. They received serum therapy with normal saline, infusion of N acetyl cysteine, and Mg SO_4 in addition to other antioxidant drugs such as vitamins E and C. Echocardiography was performed for all 32 survivors and all had normal left ventricular ejection fraction. All survivors were alert throughout hospitalisation. On admission their mean arterial pH, HCO_3, and CO_2 were 7.36±0.04 (7.29-7.42), 19.8±3.7 (13.3-28.4), and 33.3±6.6 (20.0-46.3), respectively. Blood sugar, serum electrolytes, urea, and creatinine were within normal ranges. Late admission to the hospital inversely correlated with days of hospitalisation (r=-0.393) and arterial pH (r=-0.291), HCO_3 (r=-0.227), and CO_2 (r=-0.166). Most survivors were anxious, which was the main

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reason for tachycardia without any other aetiology, such as hypotension or infection.

DISCUSSION

Our motive to report on this incident was to take a public health emergency point of view that could be useful for first responders who may find themselves in a similar situation, as this may not be the last airborne outbreak of AlP poisoning, at least in countries where AlP is still used. In addition, we wanted to show that the pattern of referrals differed for severe and mild cases. In the early stage of the outbreak, severe cases were admitted first, and as the time passed milder cases were admitted, many of whom had minimal symptoms, but also symptoms related to panic.

Suicidal or accidental poisoning with aluminium phosphide (AlP) has become rather notorious in Iran and other Asian countries over the past few decades. Phosphide in contact with moisture or acid releases a colourless, highly toxic and flammable phosphine gas, which has an odour of decaying fish or garlic (4-6).

Even though the exact mechanism of phosphine gas toxicity is still poorly explained, we believe that it inhibits cytochrome c oxidase and causes oxidative stress followed by severe mitochondrial dysfunction, impaired cellular respiration, tissue hypoxia, and eventually multiple organ damage (5, 7).

Patients mostly present with gastrointestinal symptoms, refractory hypotension, severe metabolic acidosis, and atrial and ventricular dysrhythmia. Less common are acute respiratory distress syndrome, acute tubular necrosis, disseminated intravascular coagulation, methaemoglobinaemia, and metabolic disorders (7).

Even if AlP is not ingested, phosphine gas inhalation can lead to severe toxicity, even in large spaces (7, 8) as the Mashhad hotel incident confirms.

Unfortunately, there is still no definitive management or specific antidote for this rapidly progressing poisoning, and treatment remains mainly supportive. Immediate diagnosis and continued intensive supportive measures are critical in the management of AlP poisoning.

Field inspections revealed that the hotel employees used AlP powder as a rodenticide. They were later arrested and accused of manslaughter. The main issues this tragedy has raised is that AlP is readily available and that users are poorly informed about safe use and the associated risks, both of which can be completely addressed and further tragedies prevented.

Although airborne AlP poisoning is highly fatal, we noticed that the patients who were admitted while they were rather alert and who received extensive care survived.

Health promotion policies should focus on both general public and AlP users if the total ban is not possible. First responders should receive training to recognise clinical signs and symptoms of airborne AlP poisoning and respond quickly and appropriately.

Considering the highly fatal nature of this agent and potential use in terrorist attacks, regulatory strategies and action plans for controlled AlP distribution should be enforced all over the world.

Conflict of interest

None to be declared.

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