RESEARCH LETTER

Aluminium Phosphide Poisoning: Late Presentation as Oesophageal Stricture

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Aluminium phosphide is emerging as a poison of suicidal deaths as this pesticide with no effective antidote is freely available in the market (1). Its toxicity results from the liberation of phosphine gas upon exposure of tablet to moisture, which leads to multisystem involvement. The release of cytotoxic phosphine gas primarily affects the heart, lungs, gastrointestinal tract and kidneys, although all organs can be involved (2). Poisoning with Aluminium phosphide clinically present as nausea, vomiting, restlessness, abdominal pain, palpitation, shock unresponsive to conventional treatment, cardiac arrhythmias, pulmonary edema, dyspnoea, cyanosis, & altered sensorium. Other rare effects include hepatitis, acute tubular necrosis, disseminated intravascular coagulation. The complications noticed are pericarditis, congestive cardiac failure, acute gastrointestinal haemorrhage and acute respiratory arrest (3-6). Severity of poisoning depends upon type of compound consumed. Fresh and active compound (tablets) commonly affect heart, lungs, GI tract and kidneys; cause severe metabolic acidosis and high mortality. Broken or granular form of tablets cause mild hypotension & ECG changes; mild metabolic acidosis and low mortality as activity of compound is less. Powder form of tablets are inactive; cause no systemic effects & no mortality (7). Mortality depends upon dose of poison, severity of poisoning, duration of shock, failure of response of shock to resuscitative measures & severity of hypomagnesaemia (8). Nonsurvivors have more severe hypotension and metabolic acidosis than the survivors who have more severe vomiting (9). Diagnosis is made by clinical suspicion, exhalation of Phosphine (PH_3) detected by positive silver nitrate paper test & biochemical exam. of gastric aspirate and viscera (2). Treatment consists of early gastric lavage, vasopressors and supportive care. Specific therapy with intravenous magnesium sulphate is recommended. No known antidote is avilable (2).

A 25 yr old girl presented with dysphasia. There was history of intake of Celphos (aluminium phosphide) 2weeks before for suicidal intention; patient had severe abdominal pain & vomiting which was immediately managed by local doctor with gastric lavage and IV fluid. Patient discharged satisfactorily after 3 days of uneventful hospital stay. Patient remained asymptomatic for 10 days then developed dysphagia which was rapidly progressing. Patient had no previous h/o dysphagia. Barium swallow showed stricture at lower end of oesophagus (Fig.1).

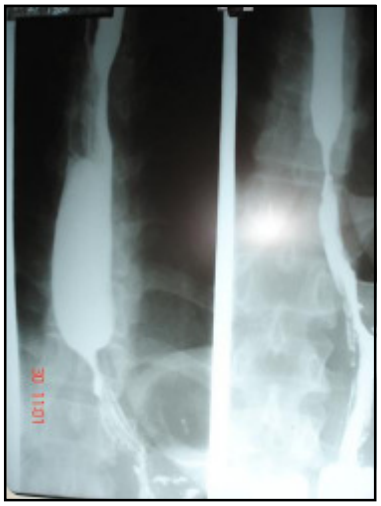

Fig. 1. Barium swallow showing stricture at lower end of oesophagus

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UGI endoscopy showed a circumferential inflammatory stricture at 35 cm which bled to touch; rest of oesophagus, stomach and duodenum were normal. Patient was managed with repeated Oesophageal dilatations and recovered.

Case is reported as stricture of oesophagus due to ingestion of aluminium phosphide is not reported before. Possible cause could be piling of tablets of aluminium phosphide at lower end of oesophagus because of large size of tablet or because celphos inhibit cholinesterase (10) thereby inhibiting peristalsis of oesophagus. This effect by increasing direct irritant effect of phosphine locally which later on resulted in stricture of oesophagus.

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