Case report

ACUTE PANCREATITIS- A LESSER-KNOWN COMPLICATION OF CELPHOS POISONING- A CASE REPORT

ABSTRACT

Aluminium phosphide is used to control rodents and pests in grain storage facilities. It is readily available as a fumigant for stored cereal grains and sold under various brand names such as QuickPhos, Salphos and Celphos. It acts by causing cellular hypoxia by its effect on mitochondria, causing inhibition of cytochrome C and by free radicals induced injury with no available antidote. Aluminium phosphide affects various systems, more commonly causing gastrointestinal tract irritation causing marked abdominal pain, shock with refractory hypotension by direct toxicity to heart and various arrythmias, acute respiratory distress syndrome and respiratory failure. Less common features include hepatotoxicity, intravascular haemolysis with methemoglobinemia and/or renal failure. Here we present a case of 24-year-old non-alcoholic male, with history of ingestion of approximately 6 grams of Celphos. At presentation he was drowsy with 2 episodes of vomiting immediately after ingestion of toxin. On subsequent investigations patient was found to have acute pancreatitis which responded to standard fluid resuscitation and acute kidney injury for which 3 episodes of haemodialysis were required. His condition subsequently improved and did not require further dialysis.

There have been only few case reports on aluminium phosphide poisoning primarily causing acute pancreatitis and acute kidney injury. In this case report, we present the case of a young man presented with consumption of aluminium phosphide and then developed acute

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pancreatitis and acute kidney injury without involvement of other organ systems including heart.

Keywords- Aluminium phosphide, poisoning, acute pancreatitis, acute kidney injury.

INTRODUCTION:

Aluminium Phosphide (AlP) is a commonly used agricultural pesticide. It is cheap, effective, and easily available. Poisoning on self with aluminium phosphide is a common method of suicide in the agricultural community in northern India. The trade name of the fumigant is Celphos, and it comes in the form of dark grey tablets of 3 g each, consisting of aluminium phosphide (56%) and aluminium carbamate (44%). Aluminium phosphide is highly toxic, of low cost and easily accessible. AlP acts by causing cellular hypoxia by its effect on mitochondria, causing inhibition of cytochrome C and by free radicals induced injury with no available antidote. The mortality rates from acute AlP poisoning (AAlPP) vary from 40 to 80 percent. The actual numbers of cases may be much larger, as less than five percent of those with AAlPP eventually reach a tertiary care centre. Death results from profound shock, myocarditis, and multi-organ failure.

We report here a case of Aluminium phosphide poisoning presented with features of acute pancreatitis. The incidence of acute pancreatitis without any other organ involvement especially heart and lungs are quite less in fact not known.

CASE SUMMARY:

Twenty-four-year-old, non-alcoholic, married male, presented with alleged history of intake of 6 gram of Celphos (aluminum phosphide) mixed with water around 5pm in the evening. Immediately after ingestion, he developed 2 episodes of non-projectile, non-bilious vomiting containing water and food particles. After this, patient became drowsy and presented to emergency. At presentation his vitals were PR- 110/min, BP- 98/68mm hg, RR- 22/min,

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Spo2- 93% on room air and GCS of E1V3M3. Patient was intubated in view of type 1 respiratory failure. On examination, patient was drowsy, afebrile, pupils were sluggishly reactive to light, abdomen was soft, non-tender with no apparent organomegaly. Initial ECG showed sinus tachycardia [fig1] Initial arterial blood gas analysis showed normal anion gap metabolic acidosis with respiratory alkalosis [table1]. On laboratory evaluation, patient had raised urea of 280.5 mg/dl and creatinine of 7.73 with 24-hour urine output of 20 ml only. He underwent 2 episodes of dialysis subsequently. His initial amylase and lipase levels were 282.8 and 900.8 units/liter which increased rapidly over 24 hours to 969 and 4100 units/liter respectively. [Table2]

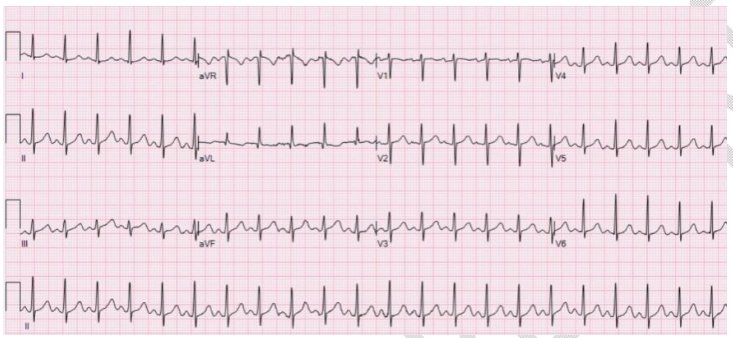

Fig 1- ECG showing sinus tachycardia

pH	7.15
Pco2	31
Po2	79
HCO3	11.9
Na+	141.6
K+	4.26
Cl-	113

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Lactate	3.5

Table 1- ABG showing high anion gap metabolic acidosis with respiratory alkalosis

Hb	**9.87**	**10.5**	**12.3**
TLC	**7808**	**6700**	**12,300**
PLATELETS	**80,000**	**100,000**	**135,000**
SODIUM	**152**	**150**	**145**
POTASSIUM	**4.55**	**3.32**	**3.61**
CALCIUM	**7.96**	**8.68**	**7.5**
UREA	**280.5**	**171**	**126**
CREATININE	**7.23**	**3.61**	**2.5**
SERUM AMYLASE	**282.8**	**969**	**686**
SERUM LIPASE	**900.8**	**4100**	**2061**
TOTAL BILIRUBIN	**0.67**		
DIRECT BILIRUBIN	**0.43**		
AST	**53.5**		
ALT	**47.2**		

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ALP	**119.4**		
GGT	**89.9**		
SERUM PROTEIN	**5.87**		
SERUM ALBUMIN	**3.44**		
SERUM GLOBULIN	**2.5**		

Table 2- baseline investigations revealing elevated amylase and lipase levels along with deranged Kidney function tests.

USG whole abdomen showed heterogeneously hypoechoic body and tail of pancreas with its enlargement (1.9 x 2.8 cm) [fig2]. Contrast enhanced CT scan was done after 72 hours which showed focal pancreatic body and tail enlargement with surrounding retroperitoneal fat stranding (2.7 x 3.9 x 1.5cm) [fig3].

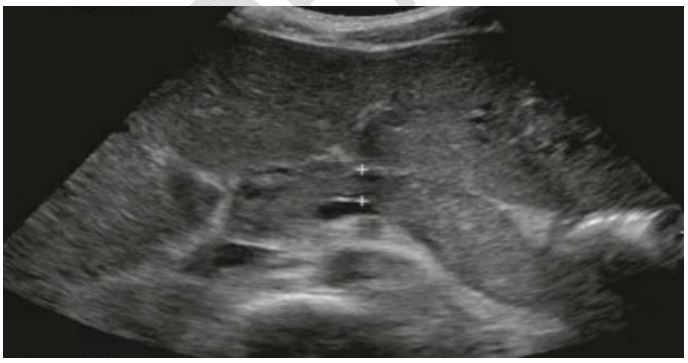

Fig 2-USG whole abdomen showed heterogeneously hypoechoic body and tail of pancreas with its enlargement (1.9 x 2.8 cm)

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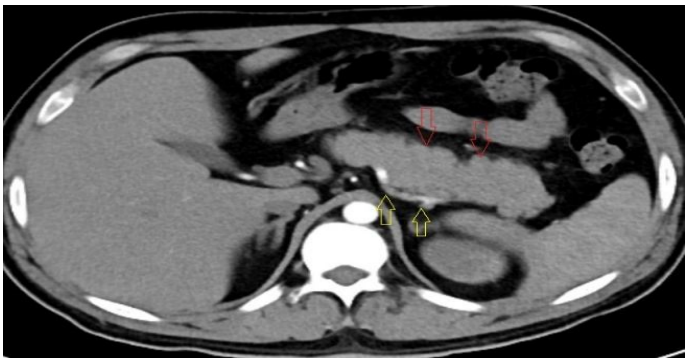

fig 3- Contrast enhanced CT scan was done after 72 hours which showed focal pancreatic body and tail enlargement with surrounding retroperitoneal fat stranding (2.7 x 3.9 x 1.5cm)

Fluid resuscitation was started immediately according to protocol and he responded well. Subsequent serum amylase and lipase levels were in decreasing trend.

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DISCUSSION:

Aluminium phosphide poisoning is a common mode of suicide in the agricultural community in northern India. The fatal dose has been reported as 0.5 g for a 70-kg adult with a mean time-interval between poisoning and death being 3 h with a range of 1–48 h. [1] The signs and symptoms of Acute AlP Poisoning are non-specific, dose dependent and evolve with time. After ingestion, toxic features usually develop within a few minutes. The major lethal consequence of AlP ingestion is profound circulatory collapse, secondary to direct effects of toxins on cardiomyocytes, fluid loss, and adrenal gland damage. The dominant clinical feature is severe hypotension refractory to dopamine therapy. Other features may include dizziness, fatigue, tightness in the chest, headache, nausea, vomiting, diarrhoea, ataxia, numbness, paraesthesia, tremor, muscle weakness, diplopia, and jaundice. If severe inhalation

occurs, the patient may develop ARDS, heart failure, arrhythmias, convulsions, and coma.[2] AlP can rarely induce complications including hepatitis, acute tubular necrosis, gastroduodenitis, bleeding diathesis, corrosive like oesophageal stricture and intravascular.

Toxins causing acute pancreatitis:
• Ethyl alcohol • Methyl alcohol • Organophosphorus poisoning • Azathioprine • Mercaptopurine • Valproic acid • Didanosine • Corticosteroids • Sulfa drugs • Scorpion venom • Zinc phosphide

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The above-mentioned patient weighing 60 kg consumed two tablets of aluminium phosphide of 3 g each amounting to a highly toxic dose. The United Kingdom guidelines for diagnosis of acute pancreatitis [3] include a desirable (not mandatory) rise of amylase (or lipase where available) within 48 h of characteristic abdominal pain. A high level of blood sugar, low level of serum calcium, evidence of metabolic acidosis at the time of admission, and raised amylase and lipase levels subsequently with imaging showing oedematous body and tail of pancreas confirmed acute pancreatitis.

On the basis of history all the common cause of toxin induced pancreatitis were ruled out. Radiologically, ultrasound and CECT abdomen helped us to rule out gall stone induced pancreatitis, hypertriglyceridemia, pancreatic duct injury and any other obstructive causes.

This case characterizes a causative association between acute pancreatitis and aluminium phosphide ingestion, a relationship that has never been observed in the literature available. Given the temporal relationship between ingestion and onset and the absence of any risk factors precluding pancreatitis in this patient, we believe it is reasonable to suggest a probable cause and effect relationship. The speculative mechanism of aluminium phosphide–induced pancreatitis is that, release of phosphine gas results in interaction and inhibition of intracellular enzymes involved in metabolic processes, the most important such enzyme being the cytochrome C oxidase resulting in the release of hydrogen peroxide, superoxide, and other free radicals. Such redox-active compounds are toxic to pancreatic β-cells by lipid peroxidation and other oxidant mechanisms, and oxygen-centred free radicals have been implicated in the induction of pancreatitis. [4,5] Alternatively, pancreatitis could have resulted from widespread cytokine release, acidosis, and probably ischemia as suggested by Bogle, et al. [6]

Learning points:
• Acute pancreatitis is rare complication of aluminium phosphide poisoning. • Because of inhibition of cytochrome-C, it causes respiratory chain inhibition and hence hypoxia which is the most probable cause organ toxicity in ALP poisoning including acute pancreatitis. • With the help of clinical features, various radiological and laboratory investigations, after excluding all causes of acute pancreatitis, cause is attributed to Aluminium phosphide.

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CONCLUSION:

In summary, we document a proven case of acute pancreatitis. The patient had no previous medical history or risk factors for the development of acute pancreatitis. Preceding the onset

of the attack, he took pellets of aluminium phosphide. Other causes of acute pancreatitis were excluded by clinical history, blood examination, and abdominal imaging. In the absence of re-challenge, we believe it is probable that aluminium phosphide has a causative link with acute pancreatitis.

REFERENCES

[1] Ranga, GS, Dwivedi, S, Agarwal, M, Kumar, D. Aluminium phosphide poisoning in a young adult: a suicidal cardiotoxin simulating myocardial ischemia. *J Indian Acad Clin Med* 2004; 5: 369.

[2] Bogle RG, Theron P, Brooks P, Dargan PI, Redhead J. Aluminium phosphide poisoning. *Emerg Med J*. 2006;23(1): e03.

[3] National Institute of Occupational Safety and Health. *NIOSH alert*: preventing phosphine gas poisoning and explosions during fumigation. 1–16, 99–126; 2003. Columbus, Ohio.

[4] Bolter, CJ, Chefurka, W. Extramitochondrial release of hydrogen peroxide from insect and mouse liver mitochondria using the respiratory inhibitors phosphine, myxothiazol, and actimycin and spectral analysis of inhibited cytochromes. *Arch Biochem Biophy* 1990; 278: 65–72.

[5] Chugh, SN, Arora, V, Sharma, A, Chugh, K. Free radical scavengers & lipid peroxidation in acute aluminium phosphide poisoning. *Indian J Med Res* 1996; 104: 190–193.

[6] Bogle, RG, et al. Aluminium phosphide poisoning. *Emerg Med J* 2006; 23: e3.