Scholars Journal of Applied Medical Sciences (SJAMS) **ISSN 2320-6691 (Online)**
Sch. J. App. Med. Sci., 2017; 5(11F):4720-4723 **ISSN 2347-954X (Print)**

(An International Publisher for Academic and Scientific Resources)
www.saspublishers.com DOI:10.36347/sjams.2017.v05i11.081

Acute Necrotising Pancreatitis: A Rarely Encountered Complication in Aluminium Phosphide (CELPHOS) Poisoning

Mayank Mishra[1], Puspendra Singh[2], Abhishek Pratap Singh[3], Jitendra Singh[4]
[1-4]Department of General surgery, Heritage IMS Varanasi India

Case Report

***Corresponding author**
Mayank Mishra

Article History
Received: 24.11.2017
Accepted: 27.11.2017
Published: 30.11.2017

Abstract: Aluminium phosphide is widely used as a pesticide and it is one of the preferred chemical utilized for suicidal poisoning with a high incidence reported from a predominantly agricultural northern India. The case of a young man is described who suffered from acute Necrotising pancreatitis related to the ingestion of aluminium phosphide (CELPHOS). This unusual complication was successfully managed with conservative treatment.
Keywords: aluminium, phosphide, pancreatitis, celphos, necrotising.

INTRODUCTION

Aluminium phosphide is widely used in India as a pesticide, in the various forms. But in recent past it use has been decline due to people awareness and sole avalibility in powder form .nevertheless after hair dye, it is one of the preferred chemical utilized for suicidal poisoning with a high incidence reported from a predominantly agricultural northern India[11]. Upon contact with moisture in the environment, it undergoes a chemical reaction yielding phosphine gas, the active ingredient. Gastrointestinal manifestations [1,2] including nausea, vomiting, and epigastric pain occur rapidly upon the release of phosphine gas in the stomach followed by cardio-vascular, pulmonary, and neurological symptoms and signs including bradycardia, hypotension, myocarditis, nonspecific electrocardiographic changes, pulmonary edema, headache, and dizziness in the presence of stable mental state3. A high mortality rate of 40-90% has been reported after ingestion of this poison[4].

We present a case of aluminium phosphide poisoning who developed acute necrotising pancreatitis, a complication reported once with the use of this poison [5,6].

CASE HISTORY

A 30 year old, male patient was admitted to the Emergency Medicine department of Heritage Institute of Medical Sciences, Varanasi after allegedly ingesting 6 tablets(each of 2 grams), 12 grams of aluminium phosphide(celphos) with a suicidal intent; 4 hour earlier to admission after which he had three episodes of vomiting. The patient complained of burning abdominal pain and he was delirious. He had tachycardia, blood pressure of 86/56 mm Hg ,respiratory rate 28/min Spo2 88% without O2, systemic examination was unremarkable except bilateral crepitation on chest auscultation and muffled heart rate and systolic cepitus .

A the time of admission, his total leukocyte count was 6100 cells/mm3 with 73% polymorphs, random blood sugar was 466mg/dL; the arterial blood gas analysis showed combined metabolic and respiratory acidosis with a ph 7.03; serum sodium 135.5meq/L, potassium 3.31meq/L, calcium 1.98 mmol/LPhosphate,1.2g/dL, magnesium3.9mg/dL[9], blood urea nitrogen(BUN) 11,6mg/Dl, creatinine 2.1mg/dL total bilirubin0.8mg/dL with normal levels of liver transaminases and serum proteins. The electrocardiogram8 showed non- specific S-T segment changes and the echocardiographic evaluation showed left ventricular ejection fraction of 45% probably indicating acute myocarditis. The serial investigation profile of the patient is shown in Table 1[7].

The patient was resuscitated with fluids, vasopressors (noradalineand dobutamine), and magnesium sulphate besides other supportive management. His condition stabilized after 72 h of aggressive management but the complaint of a excruciating epigastric and periumblical pain, radiating to back associated with nausea, high level of blood sugar, a low level of serum calcium, and evidence of

metabolic acidosis on day 4, a possibility of pancreatitis was considered. The patient was then referred to surgery department for further management. Serum amylase, lipase CRP and LDH levels were ordered which were 366 IU/L (normal 25-125), 1851U/L(normal<190U/L),109.9mg/dl (Normal 140-280U/L)and1238.4 U/L(Normal 1-3 mg/dl)respectively . Abdominal ultrasonography scan was within normal limits; however, CT scan of the abdomen demonstrated edematous pancreatic head with features of necrosis confirming *acute* necrotising pancreatitis.

The patient was treated conservatively on the lines of acute pancreatitis with fluid resuscitation input output monitoring, broad spectrum antibiotics, blood transfusion, albumin infusion ; the serum amylase and lipase levels settled down to normal values by the 16th day of admission; the left ventricular systolic functions improved, and the patient was, subsequently, discharged on the 16th day. Patient has some family dispute, history of previous suicidal attempt ,so a psychiatric consultation was taken and he on behavioural therapy.

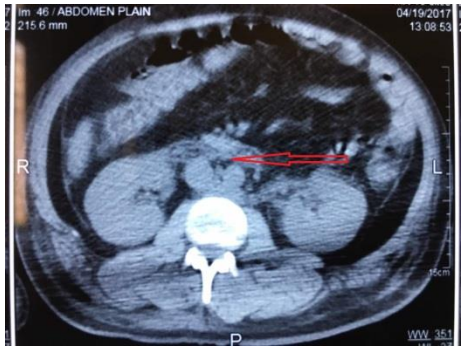

Fig-1& 2: Arrow showing Necrotising Pancreatitis with surrounding edema

Hematological and biochemical day to day parameters of the patient during hospital stay and follow up-

Table-1: showing Day to day assessment of haematological investigations

PARAMETRES	DAY 1 12/04/17	DAY 3	DAY 4	DAY 5	DAY 6	DAY 7	DAY 8	DAY 9	DAY 10	DAY 12	DAY 21	DAY 30
Hb(gm/dl)	12.0	10.9		9.8	6.9		9.7		8.3		10.9	12
TLC (/mm3)	6100	2400		5500	1600		15300		12300		6700	5700
DLC (mm3)	73/18/08/01	72/22/05/01		79/14/05/02	72/21/06/01		88/6/01/02		82/07/03/01		54/38/07/01	54/36/08/02
Platelet (lacs/mm3)	0.9	1.2		0.77	0.26		0.22	0.20	0.42		1.48	1..67
Hematocrit					19.2		30		26.6		30	28
RBS (mg/dl)			476	345	248	68	122	154	87	96	110	123
B.Urea(mg/dl)	25	114	54.4	61	69.7	125.7	167.5			86	72	67
S.Creatinine(mg/dl)	1.2	2.0	0.99	1.0	1.29	2.17	3.43			1.1	1.0	1.0
URIC ACID(mg/dl)	7.8	12.7		6.1		7.05				8.8	7,4	6.9
S .PHOSPHROUS(mg/dl)	2.1	4.6	1.01	1.7		5.95				2.5	2.8	
S,MAGNISIUM(mg/dl)	3.9	4.1	3.64	4.3	3.63	3.58	4.39				-	
S .SODIUM(mmol/l)	135.5	132	137	133	140.3	141.1	140.2	135.3		135.8	134	
S. POTASSIUM(mmol/l)	3.31	3.0	2.63	2.71	2.98	3.77	3.72	3.36		3.79	3.98	
S. CALCIUM(mmol/l)	1.48		1.61	1.75	2.13	1.77	1.69				2.1	
Ionised Calcium(mmol/l)	1.04		0.89	0.99	1.0	0.93	0.87					
nCalcium(mmol/L)	0.44		0.80	0.88	1.06	1.03	0.84					
S.Bilirubin(D) (mg/dl)	0.2			1.2			8.09			1.9	0.8	
S.Bilirubin(I)(mg/dl)	0.6			0.6			1.0			2.1	0.2	
SGOT(IU/l)	20			90			118.3			36	40	
SGPT(IU/L	39			94			69.4			47	56	
ALP(U/L)	75		205	285		378.3	398			254	212	
S.					241.1	252.3						

Trigycerides(mg/dl)												
S, Total protien(gm/dl)	8.1			5.9			6.15			5.9	6.1	6.3
S.Albumin(gm/dl)	4.5			3.1			3.29			2.6	3.2	4.1
S, globulin (gm/dl)	3.6			2.8			2.86			3.3		
A:G ratio	1.25			1.10			1.15			0.78		
ESR ((mm)					98		104		64	42	18	20
S.lipase(U/L)					1851		1901		1734	1266	637	330
S.amylase(IU/L)					366							
CRP (mg/L)					109.9		194.0			64.8	12	7
LDH(U/l)					1238.6		1408.4			988.6	342	401

Table-2: Showing Day to Day ABG assessment of the patient

PARAMETRES	DAY 1	DAY 2	DAY 3	DAY 4	DAY 5	DAY 6	DAY 7	DAY 8	DAY 9	DAY 10	DAY 11	DAY 12
ABG values												
Ph	7.03	7.13	7.3	-	-	6.98	7.10	7.10	7.24	7.31	-	-
PaO2	76	88	96	-	-	70	75	82	88	94	-	-
PCO2	24	19	28	-	-	16	26	28	32	33	-	-
HCO3	19	24	24	-	-	18	20	22	22	23	-	-
S. Sodium	128	130	130			126	121	127	129	131	-	-
S. Potassium	3.4	3.1	3.1			2.8	2.9	3.0	2.9	3.2	-	-
S. Calcium	1.54	1.61	1.58			1.44	1.52	1.58	1.60	1.60	-	-
Haemoglobin	13.1	10.7	10.2			7.3	6.9	8.0	8.4	8.7	-	-

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 hours, with a range of 1-48 hours[10]. The above mentioned patient weighing 60 kg consumed 6 tablets of aluminium phosphide of 12 gm amounting to a highly toxic dose (lethal dose 15mg-50mg).

The United Kingdom guidelines for diagnosis of acute pancreatitis include a desirable (not mandatory) rise of amylase (or lipase where available) within 48h of characteristic abdominal pain. A high level of blood sugar, low level of serum calcium, evidence of metabolic acidosis[13] at the time of admission , and raised amylase and lipase levels subsequently with imaging showing edematous head of pancreas confirmed acute pancreatitis. We eliminated the most common etiological factors in developing pancreatitis such as alcoholism, gall stones, hypertriglyceridemia, and drugs by history and relevant investigations as discussed earlier, and are, therefore, left with aluminium phosphide-induced pancreatitis.

This case characterizes a causative association between acute pancreatitis and aluminium phosphide ingestion[12], a relationship that has very rarely been observed in the literature available(approximately 6 cases reported till date). Given the temporal relationship between ingestion and onset and absence of any risk factors precluding pancreatitis in the 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 beta cells by lipid peroxidation and other oxidant mechanism, and oxygen-centred free radicals have been implicated in the induction of pancreatitis. Alternatively, pancreatitis could have resulted from widespread cytokine release, acidosis and probably ischemia as suggested Bogle,*et al*.

In summary, we documented a proven case of acute necrotising pancreatitis following Celphos Ingestion. 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. Hence we can conclude to define Celphos as a probable causative agent of Acute Pancreatitis [14].

REFERENCES

1. Bajaj R, Wasir HS, Agarwal R, Malhotra A, Chopra P, Bhatia ML. Aluminum phosphide poisoning: Clinical toxicity and outcome in eleven intensively monitored patients. Natl Med J India. 1988;1(6):270-4.

2. Gupta S, Ahlawat SK. Aluminum phosphide poisoning—a review. Journal of Toxicology: Clinical Toxicology. 1995 Jan 1;33(1):19-24.
3. Singh S, Singh D, Wig N, Jit I, Sharma BK. Aluminum phosphide ingestion—a clinico-pathologic study. Journal of Toxicology: Clinical Toxicology. 1996 Jan 1;34(6):703-6.
4. Katira R Elhence GP Mehrotra ML Srivastava SS Mitra A Agamala R Ram A. A study of aluminum phosphide poisoning with special reference to electrocardiographic changes. J Assoc Phys India 1990; 38: 471 – 473. Google Scholar Medline
5. Gupta MS, Mehta L, Chugh SN, Malhotra KC. Aluminium phosphide poisoning. Two cases with rare presentation. J Assoc Physicians India 1990; 38:509–510.
6. Gupta SK, Peshin SS, Srivastava A, Kaleekal T.A study of childhood poisoning at National Poisons Information Centre, All India Institute of Medical Sciences, New Delhi. J Occup Health 2003; 45:191–196.
7. Kalra GS, Anand IS, Jit I, Bushnurmath B, Wahi PL. Aluminium phosphide poisoning: haemodynamic observations. Indian heart journal. 1991;43(3):175-8.
8. Katira R, Elhence GP, Mehrotra ML, Srivastava SS, Mitra A, Agarwala R, Ram A. A study of aluminum phosphide (AlP) poisoning with special reference to electrocardiographic changes. The Journal of the Association of Physicians of India. 1990 Jul;38(7):471-3.
9. Khayam-Bashi H, Liu TZ, Walter V. Measurement of serum magnesium with a centrifugal analyzer. Clin Chem 1977; 23:289–291.
10. Lohani SP, Casavant MJ, Ekins BR, Walson PD, Wolowich WR. Zinc Phosphide Poisoning: A Retrospective Study Of 21 Cases. Journal of Toxicology-Clinical Toxicology. 2000 Aug 1;38(5):515.
11. Misra UK, Bhargava SK, Nag D, Kidwai MM, Lal MM. Occupational phosphine exposure in Indian workers. Toxicology letters. 1988 Sep 1;42(3):257-63.
12. Sinha US, Kapoor AK, Singh AK, Gupta A, Mehrotra R. Histopathological changes in cases of aluminium phosphide poisoning. Indian journal of pathology & microbiology. 2005 Apr;48(2):177-80.
13. Siwach SB, Jagdish, Katyal VK, Dhall A, Bhardwaj G. Prognostic indices in aluminium phosphide poisoning observations on acidosis & central venous pressure. J Assoc Physicians India 1997;45:693-5.
14. SK Verma1, S Ahmad1, N Shirazi2, SP Barthwal1, D Khurana1, M Chugh1 and HS Gambhir'Acute pancreatitis:a lesser-known complication of aluminum phosphide poisoning." Human & Experimental Toxicology (2007) 26: 979–981