Original Article

Demographic and Clinical profile of Acute Zinc-phosphide poisoning in a south Indian tertiary care Hospital: a five-year retrospective study

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ABSTRACT

Aim: To predict the clinical & demographic profile of patients who presented to a tertiary centre in southern India with Zinc Phosphide poisoning.

Material and Methods: A retrospective cross-sectional analysis of all acute zinc phosphide poisoning cases who presented to our emergency medicine department within January 1st 2010 to June 30th 2015 was done.

Results: The average age of the cases was 23.6 years. 53.34% were female and 35.55% of total cases were married. Out of 45, 88.88% took the product in an attempt of suicide and the rest 11.11% ingested it accidentally. Of the 40 suicide attempts, 15.5% had previous history for suicide attempts. All patients had nausea & vomiting as initial symptoms, while 60% had breathlessness & 48.88% had abdominal pain too. All patients had tachycardia as a common sign while 75% cases also had tachypnea. 75% patients developed bleeding manifestations & 53.33% had encephalopathy during the course. 64.4% had hypotension needing inotropic support. Renal failure was seen in 48.8% cases. Within 72hours 60% of cases had high anion gap non-compensated metabolic acidosis. Of the 33.33% subjects who had an elevation of SGOT/SGPT within 24hrs of ingestion, 53.33% died while 46.67% required liver transplantation.

Conclusion: Although it is a rare form of suicide attempt, the mortality rate (46.66%) we got was high. In most patients alteration of SGOT/SGPT values was apparent only after 72hrs of ingestion & mortality is high in those who showed an elevation within 24hrs. Patients developing severe hepatic failure, increased INR, hypotension, encephalopathy and metabolic acidosis had high mortality rate.

Keywords: suicide attempt; zinc phosphide; poisoning; emergency department; fulminant hepatic failure

INTRODUCTION

It has been documented that some form of poisoning is responsible for more than one million illnesses worldwide annually in one way or the other. This is just an estimate as most cases of poisoning actually go unreported, particularly in Third World countries.[1] The issue of poisoning worsens as time progresses as newer drugs and chemicals become easily available in large quantity. Incidents are also influenced by social issues & behaviour. Self-poisoning has reached epidemic proportions and has become a major public health issue in parts of the developing world.[2,3] Occupational & accidental exposure to poisons is by hazardous occupational practices and unsafe storage of pesticides. Based on the limited data, it is estimated that three million cases of pesticide poisoning occur world-wide annually with 220,000 deaths, the majority intentional.[4]

Zinc Phosphide is a dark-grey, crystalline compound which is a highly effective rodenticide, commonly used in agricultural sector by mixing with food as bait.[5] Phosphides are normally found as powders or pellets, usually as zinc or aluminium phosphide (Zn_3P_2 and AlP); calcium and magnesium phosphide salts are also available.[6] Acute poisoning can be direct by ingestion of salt or indirect by accidental inhalation of phosphine gas (PH_3) generated during its use. Since zinc Phosphide is cheap & widely used in our region, the use of it as a suicidal agent is also increasing.[7] Orally taken Zinc phosphide reacts with water and acid in the stomach and produces phosphine gas, which account for the large part of observed toxicity. Being an extremely toxic gas, it irritates respiratory tract and also cause severe systemic toxicity.[8] It disrupts mitochondrial function through blocking cytochrome C oxidase enzyme and increases free radical generation resulting in lipid peroxidation and producing energy failure in cells.[9] Phosphides produce rapid toxicity within 30 minutes of ingestion and death may follow in less than 6 hours.[10] Phosphide ingestions over 500 mg are often fatal.[11] Zinc phosphide is a potent gastric irritant; profuse vomiting and abdominal pain

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are often the first symptoms.[12] Respiratory complaints could be tachypnea, dyspnea, cough and can even progress to acute lung injury over days.[13,14] Also non-cardiogenic pulmonary oedema may develop later and should be managed aggressively by positive end-expiratory pressure ventilation.[15] Tachycardia, hypotension and dysrhythmias like atrial fibrillation, flutter, heart block and ventricular tachycardia and fibrillation may develop.[16] Nervous system involvement can cause coma, tonic-clonic convulsions and delirium.[13] Raised transaminases, hepatic failure, severe hypoglycemia or severe metabolic acidosis with acute distal renal tubular acidosis, have been associated with ingestion.[17,18]

Absence of a specific antidote & rapid multi organ failure results in very high mortality. Even though rapid decontamination by the use of activated charcoal, gastric lavage with and aggressive resuscitative measures according to various protocols are practised from centre to centre; none of these have been proven to be effective by clinical studies. Even though its use as a suicidal agent is known, there are only a handful of studies from Indian sub-continent detailing the clinical features & prognosis of the patients.[1,15,20,21,] There are significant numbers of studies detailing the effects, treatment & prognosis of poisoning with aluminium phosphide and other rodenticides, but zinc phosphide is not that elaborately studied. Patients with acute zinc phosphide poisoning can have fulminant hepatic failure as a complication. High mortality is seen in patients having coagulopathy, encephalopathy, hypotension and severe metabolic acidosis. The purpose of this study was to predict the clinical and demographic profile of patients who presented with zinc phosphide poisoning & the prognosis of the same.

MATERIAL AND METHODS

Study design and patients

All cases of acute Zinc Phosphide poisoning, presented to our Emergency Department (ED) over past five-years from January 1st 2010 to June 30th 2015 were retrospectively reviewed.

The clinical and laboratory data for 73 patients who were affected by Zinc Phosphide & presented to our ED was analysed. Patient files and electronic records were used to gather data. The cases were diagnosed as zinc phosphide poisoning based on history and laboratory findings. All patients were transferred to the Critical Care department after initial resuscitation and gastric lavage in our ED. Blood samples for biochemical and haematologic determinations were sent from the ED within 1 hour of presentation. Information was collected on age, gender, nature of poisoning and any delay in presentation to the hospital. The severity of poisoning was assessed from laboratory and clinical parameters, which included hepatic enzyme levels (aspartate aminotransferase [AST, normal range: 5-45 U/dL] and alanine aminotransferase [ALT, normal range: 5-40 U/dL]), elevated billirubin levels (>2mg/dl), deranged INR (>1.5), presence of shock; hypotension (mean arterial pressure [MAP] < 60), elevated serum creatinine (>1.4mg/dl), hypoglycemia(<70mg/dl) and alterations in arterial pH level (normal range: 7.35-7.45). Statistical significance of these demographic & clinical variables to outcome was looked upon.

Exclusion criteria

Paediatric patients (age<18years), patients who consumed zinc phosphide by mixing with other substances or poisons (alcohol, sedatives etc) were excluded from the study. Also patients who were diagnosed to have any concomitant liver diseases were also excluded. All chronic poisoning cases & cases presenting after 48hrs of zinc phosphide consumption were excluded. Any patient who did not give consent or got transferred to other facility was also excluded from the study. After applying exclusion criteria we got a sample size of 45 from initial pool of 73 patients. The SPSS software version 20 was used for statistical analysis & a p-value of < 0.05 was considered to be significant.

RESULTS

The average age in the study was 23.06 years. 53.34% of total was female and 46.66% male. 35.55% cases were married while the rest single. Total 88.88% subjects ingested the poison in an attempt of suicide. Post-ingestion 55.55% cases presented directly (primary) to our hospital, whereas the rest 17 patients were delayed in presentation due to referral from outside hospitals. Minimum ICU admission days was 5 while maximum of 15, average ICU stay was of 8.9 days (**Table 1**). All subjects had nausea & vomiting as initial symptom, while 60% had breathlessness & 48.88% had abdominal pain too as initial symptoms. All patients had tachycardia as an early sign while 75% also had tachypnea. (**Figure 1**) Total of 75% patients developed bleeding manifestations as the disease progressed, while 53.33% developed encephalopathy too. 64.44% developed hypotension needing inotropic support and 42.22% of them developed refractory hypotension. 26.66% patients required mechanical ventilation mainly for severe acidosis and airway protection due to decreased sensorium. On presentation 17 (37.78%) had respiratory alkalosis in

ABG, but within 72 hours 27 (60%) of cases developed non-compensated high anion gap metabolic acidosis **[Table 2]**. Out of 45cases, 24.4% had Mild to Moderate hepatic injury while 13.3% had severe hepatic injury and 62.22% developed fatal liver injury in accordance with Drug Induced Liver Injury Network (DILIN) grading scale. The grade fatal consists of mortality due to liver injury and patients who required transplant. Essentially all fatal grade patients had developed severe hepatic failure but their outcome was different. In most patients alteration of LFT & RFT values was apparent only after 72hrs of ingestion. All patients showed raised liver function tests by 72hours. Of the 15 (33.33%) who showed elevation of SGOT/SGPT within 24hrs of ingestion, 8(53.33%) dies while the rest 7(46.67%) underwent liver transplantation. Renal failure, hypoglycemia & hyperbillirubinemia were seen by 5^{th} – 6^{th} day of ingestion of poison while 60% developed acidosis within 72 hours. Mortality in the study was 46.66%. An average of 6.9 days was calculated for patients to develop mortality from day of ingestion.

DISCUSSION

Almost around the globe phosphide is commonly used for suicide attempts by the younger productive age group of society.[19] In previous studies mean age of patients involved are described from 27years to 40years, generally idea is that patients in their 3rd decade of life are more prone for suicide.[20,21] In this study, mean age was 23.6years, even though lower it was still comparable to earlier literature. The incidence of attempted suicide in this study was only slightly greater in females than males, while increased incidence of poisoning in females had been described by various studies[20,22] the cause of increased male incidence can be attributed to adaptation issues to social life, financial difficulties both of which increase possibility of depression.[20,23] Like others we also found sex to be a non-significant factor for mortality

According to Curcić M et al.[23] 34% patients had depression, 32% were alcoholics, and 26% suffered neurosis. In our study, the number of patients using antidepressants (n=14, 31.1%) was lower than those not using them, this can be due to poor recognition of disease or reluctance in seeking medical help; 15.5% of the total patients had history of previous suicide attempts also.

After complete history and physical examination from Emergency Room, symptomatic and supportive care was immediately initiated. Some studies suggest induction of emesis within 30 min of ingestion,[24] gastric lavage with 3–5% sodium bicarbonate (to reduce gastric acid and production of phosphine) or 1:5000 potassium permanganate (to oxidize phosphine to less absorbable phosphide) has been advised[25]; however, other researchers stated that its effectiveness is unproven.[26] Each of these measures has its own risks and we must be aware of those before administration. Gastrointestinal decontamination with administration of sodium bicarbonate solution was performed in 62.22% (28) of our patients who presented within 2hours of poisoning; of that only 50% (14) survived in comparison to 35.3%(7) patients out of total 17cases who did not receive gastric lavage. 17cases which presented 2hours post-ingestion received oral activated charcoal and other symptomatic treatments, like fluid resuscitation, antispasmodics, anti-emetics, inotropic support, mechanical ventilation and antibiotics as per patient characteristics.

The onset of clinical signs & symptoms following ingestion is highly variable but averages within 4hours.[20] Proudfoot et al.[25] has reported that, usually systemic toxicity is noticed within a short interval after the ingestion. The average time taken for patients to be brought to ED from place of incidence was 6.30 hours. The number of referred patients that we got was 44.45%, since they were initially stabilized and managed in outside hospitals before shifting to our ED explains the longer access time.

Phosphine acts as a strong reducing agent capable of inhibiting cellular enzymes involved in several metabolic processes causing failure of organ systems.[27] Profound circulatory collapse which is a lethal consequence secondary to the toxins generated, which have direct effects on cardiac myocytes, fluid loss, and adrenal gland damage.[25] The amount of poison reported in poisoning cases differs among publications. Chugh et al.[24] reported that serum phosphine levels correlated positively with the severity of poisoning, and levels equal to or less than 1.067±0.16 mg % appeared to be at the limit of phosphine toxicity. Another study indicated lethal zinc phosphide dose as 4-5grams.[28] In our study, average amount of poison consumed by the patients was 3grams.

All patients in the study developed vomiting as an initial symptom, associated with acute breathlessness in 60% & abdominal pain 48.88% patients. Multiple system affection was evident in patients who did not get medical help immediately or ingested large amounts of poison. Metabolic acidosis indicates moderate to severe degree of poisoning as per Proudfoot.[25] Severe Metabolic acidosis alone or in association with acute respiratory alkalosis, is very common.[18] In one study by Mathai A et al.[19] the

mean pH on admission was found to be 7.20±0.14 and mean bicarbonate concentration was 12.32±5.45 mmol/L. In the present study, five patients (11.1%) had low pH levels at time of admission and all developed mortality; low pH being the causative factor for deaths in these patients. The mean bicarbonate level in this study was 16.5±4.22 mmol/L. 62.22% of cases were metabolically stable on admission while 26.66% patients had acute respiratory alkalosis explained by hyperventilation due to anxiety and 11.1% had compensated metabolic acidosis. Within 24 hours of admission metabolic acidosis increased to 66.7% from the initial 11.1%. Severe metabolic acidosis is a prominent factor for mortality seen in our study similarly with others.[20,24,25] Patients with severe respiratory compromise, 26.7% required intubation and artificial ventilation. Hemodynamic instability and refractory hypotension have been reported in certain studies.[6-8] In our study, 64.4% patients had hypotension and 65.5% of them developed mortality. Central nervous system depression may arise due to hypotension, toxin as such or due to hepatic or other organ system failure; this can present as unresponsiveness either at time of admission or after admission.[24,25,28] A study by Louriz et al.[30] reported that mortality in acute phosphine poisoning (APP) correlated with shock and altered consciousness. In our study, 53.3% developed depressed sensorium at some point of treatment; while three of them who had it from presentation itself succumbed to death. 70.8% of those with depressed sensorium died, refractory state of shock and encephalopathy due to hepatic failure being the cause of high mortality.

It has been proven that the systemic toxicity caused by phosphine, is mainly to the heart, lungs, liver, gastrointestinal tract, kidney and brain since PH3 molecules targets them particularly.[31,32] The effect of phosphine causing failure of heart, lungs and gastrointestinal tract is well studied with aluminium phosphide poisoning. But the effect of it in liver & renal parenchyma is not well studied.[7,16,25,29] Altered liver function tests (LFTs) in non-fatal cases of zinc phosphide poisoning suggest that there is some pathology targeting liver.[7,32,33] A study in Iran reported that the liver biopsies from 37 patients who had died of zinc phosphide poisoning showed injury to hepatic parenchyma which ranged from congestion to necrosis at different stages. Main changes found were fine cytoplasmic vacuolization of hepatocytes and sinusoidal congestion.[34-36]

Saleki et al.[34] stated that PH3 can cause liver dysfunction, especially after the first day of poisoning. All 45 patients of our study group developed altered liver function tests by the 3rd day(72hrs) of admission; 33.3%(15) patients showed altered LFTs within 24hours of admission, of these 53.3% developed mortality. The rate of mortality was twice as high in patients with elevated LFTs within 24hrs of admission when compared to those who developed derangement over 72hrs. Grading scale developed by Drug Induced Liver Injury Network (DILIN)[37] was used to grade severity of liver injury due to poisoning. Patients were categorised into 4groups of Mild, Moderate, Severe & Fatal liver injury according to presence of jaundice, hospitalization, signs of hepatic or other organ failure and ultimate outcome. According to the grading scale, out of the 45cases, 24.4% had mild to moderate liver injury, 13.33% had severe & 62.22% developed fatal liver injury. Clinically 75% (34) patients had features of fulminant hepatic failure. Coagulopathy in form of raised INR was seen in 75% patients, this is a new observation and haven't been described in other studies as not much have concentrated on the hepatic manifestations of phosphide. Renal failure was present in 48.8% patients in our study of which 59% succumbed to death. 81.8% of the patients who had renal failure also had signs of shock too. The cause of renal failure can be due to phosphine or due to severe hypotension. Sarma et al.[38] have reported that zinc phosphide ingestion leads to acute pancreatitis, but our study did not find any patients with elevated serum lipase levels. About 55.5% of our total subjects had episodes of hypoglycemia of which 60%(15) dies; hypoglycemia sets in by 5th day of admission for about 33.3% patients of which 53.3%(8) develops mortality. In our study hypoglycemia was seen in patients with severe/fulminant hepatic failure and out of the 10 survivors with hypoglycemia seven underwent liver transplantation after which normoglycemia was acheived. Thus the presence of hypoglycemia can be explained by fulminant hepatic failure alone than with involvement of pancreas. No antidote is available for poisoning with phosphides. The reported mortality for aluminium phosphine and zinc phosphide greatly varies across different studies, although they are generally high.[9,29,39] The mortality rate in our study was 46.66% (21).

CONCLUSION

Although it is a rare form of suicide attempt, the mortality rate 46.66%(21) for patients with Zinc Phosphide poisoning is very high. Early decontamination measures can be used but statistically did not show any significance. In most patients alteration of SGOT/SGPT values was apparent only after 72hrs of ingestion. Mortality was high in those who showed an elevation of SGOT/SGPT within 24hrs. Factors like severe hepatic failure, increased INR, encephalopathy, hypotension and metabolic acidosis was

seen in patients with higher mortality.

Limitations

Study group was limited to admissions to our center and thus represent only a small percentage of cases of zinc phosphide poisoning in Kerala; most of the cases would present to other poison control centres and general hospitals. There are also missed cases due to misdiagnosis. Unfortunately, it is not currently possible to link all the databases to provide a more comprehensive overview of poisoning with zinc phosphide in our country; so it is important to highlight our results on this type of poisoning and to publishing statistical records of all cases of poisoning in specialized and other general hospitals.

Recommendations

Due to their easy availability, pesticides have become the most commonly used agent for suicide in the developing world.[24,26] Since intentional poisoning is often impulsive and simplified by easy availability of poison, a proportion of it can be prevented by reducing access. So regulating availability of pesticides and improving medical management may reduce fatality from pesticide poisoning and also reduce the number of suicide attempts in youths.[28]

CONFLICTS OF INTEREST

Declared none.

Table 1 : Association of Demographic Variables to Outcome

Category		Frequency(n) (%)	P-Value
Gender	Male	21 (46.66)	0.556
	Female	24 (53.34)	
Maritial Status	Married	16 (35.55)	–
	Unmarried	19 (64.45)	
Type of cases	Primary	25 (55.55)	–
	Referred	20 (44.45)	
Time to presentation	0-2 hours	28 (62.22)	0.059
	2-12 hours	12 (26.66)	
	12-24 hours	5 (11.12)	
Type of poisoning	Self-Harm	40 (88.88)	–
	Accidental	5 (11.12)	

Gender & Time of presentation didn't show any statistical significance on association with outcome.

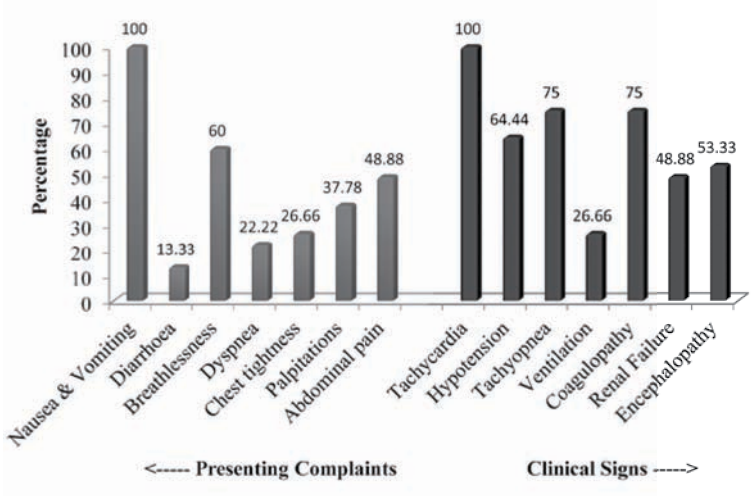
Table 2 : Association of clinical variables to outcome

Category	Frequency n (%)	Mortality (% of frequency)	P-Value
Tachycardia	45 (100)	21 (46.6)	-
Tachypnea	34 (75)	17 (50)	-
Mechanical Ventilation	12 (26.66)	8 (66.66)	-
Hyperbillirubinemia	40 (88.89)	20 (50)	-
Hypoglycemia	25 (55.55)	10 (45.5)	0.059
Hypotension*#	29 (64.44)	19 (65.5)	0.001
Coagulopathy*	34 (75)	21 (61.8)	<0.001
Encephalopathy*#	24 (53.33)	17 (70.8)	0.001
Metabolic Acidosis*	39 (86.66)	21 (53.8)	0.023
Renal Failure	22 (48.88)	13 (59.1)	0.139

* - Variables that showed significance in univariate analysis

- Variables that showed significance in multivariate analysis

Fig.1: Distribution of Presenting Symptoms & Clinical Signs in the study group



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