Sultan Qaboos University Med J, August 2018, Vol. 18, Iss. 3, pp. e299–303, Epub. 19 Dec 18
Submitted 5 Mar 18
Revisions Req. 10 Apr & 28 May 18; Revisions Recd. 10 May & 5 Jun 18
Accepted 7 Jun 18
doi: 10.18295/squmj.2018.18.03.006

CLINICAL & BASIC RESEARCH

Prognostic Significance of Blood Glucose Levels and Alterations Among Patients with Aluminium Phosphide Poisoning

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دلالة مستويات الجلوكوز في الدم وتغيراته كعوامل تنبؤ بنتائج المرضى المصابين بتسمم فوسفيد الألومنيوم

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ABSTRACT: ***Objectives:*** This study aimed to assess the prognostic significance of blood glucose levels and blood glucose alterations (i.e. hyper- or hypoglycaemia) among patients with aluminium phosphide (AlP) poisoning. ***Methods:*** This prospective observational study was conducted at the Postgraduate Institute of Medical Education & Research, Chandigarh, India, between January 2010 and June 2011. All patients presenting to the emergency department with a definitive history of AlP ingestion or symptoms compatible with AlP poisoning were included in the study. Blood glucose levels were recorded at presentation and every six hours thereafter. Alterations in blood glucose levels and other clinical and laboratory variables were subsequently compared between survivors and non-survivors. ***Results:*** A total of 116 patients with AlP poisoning were identified. Of these, 57 patients (49%) survived and 59 patients (51%) died. At presentation, the mean blood glucose levels of survivors and non-survivors were 119.9 ± 35.7 mg/dL and 159.7 ± 92.5 mg/dL, respectively (P <0.001). In comparison to the survivors, non-survivors had significantly higher heart rates, total leukocyte counts, blood glucose level alterations and serum creatinine levels (P <0.050). In addition, systolic blood pressure, Glasgow coma scale scores, arterial blood gas pH and bicarbonate values and duration of hospital stay was significantly lower compared to survivors (P <0.001). However, neither blood glucose levels at admission nor blood glucose alterations correlated independently with mortality in a multivariate analysis. ***Conclusion:*** The role of blood glucose level alterations in predicting patient outcomes in AlP poisoning cases remains inconclusive. Further studies with larger sample sizes are required.

Keywords: Aluminum Phosphide; Poisoning; Blood Glucose; Hyperglycemia; Hypoglycemia; Mortality; Prognostic Factors; India.

الملخص: الهدف: هدفت هذه الدراسة إلى تقييم دلالة مستويات الجلوكوز في الدم وتغيراته (زياده أو نقصان) كعوامل تنبؤ بنتائج المرضى المصابين بتسمم فوسفيد الألومنيوم. **الطريقة:** أجريت هذه الدراسة الرصدية في معهد الدراسات العليا للتعليم الطبي والبحوث في شانديغار، الهند، في الوقت بين يناير 2010 و يونيو 2011 على جميع المرضى القادمين إلى قسم الطوارئ بعد التأكد من ابتلاع فوسفيد الألومنيوم أو لديهم أعراض متوافقة مع هذا التسمم. تم تسجيل مستويات الجلوكوز في الدم عند قدومهم وكل ست ساعات بعد ذلك. تمت بعد ذلك مقارنة التغييرات في مستويات الجلوكوز في الدم وغيرها من المتغيرات السريرية والمخبرية بين الناجين وغير الناجين من التسمم. **النتائج:** تم تحديد مجموعه 116 مريضا أصيبو بتسمم فوسفيد الألومنيوم. من هؤلاء، نجا 57 مريضا (49%) وتوفي 59 مريضا (51%). عند القدوم إلى قسم الطوارىء، كان متوسط مستويات السكر في الدم للناجين وغير الناجين 35.7 ± 119.9 ملغ/ديسيلتر و 92.5 ± 159.7 ملغم/ديسيلتر، على التوالي (P <0.001). كان لدى غير الناجين بالمقارنة مع الناجين قيم أعلى بشكل ملحوظ لمعدل ضربات قلب، أعداد كريات الدم البيض، تغيرات مستوى الجلوكوز ومستويات الكرياتينين في الدم (P <0.050). بالإضافة إلى ذلك، كان ضغط الدم الانقباضي ومقياس غيبوبة غلاسكو ودرجة الحموضة ومستوى البيكربونات في الدم الشرياني ومدة الإقامة في المستشفى أقل بكثير مقارنة بالناجين (P <0.001). ومع ذلك، لا ترتبط مستويات الجلوكوز في الدم عند القدوم ولا تغيرات الجلوكوز في الدم بشكل مستقل مع معدل الوفيات في التحليل متعدد المتغيرات. **الخلاصة:** لا يزال دور تغيرات مستوى الجلوكوز في الدم في التنبؤ بنتائج المرضى في حالات تسمم فوسفيد الألومنيوم غير حاسم. مطلوب مزيد من الدراسات مع أحجام عينة أكبر.

الكلمات المفتاحية: تسمم فوسفيد الألومنيوم؛ جلوكوز الدم؛ ارتفاع السكر في الدم؛ نقص السكر في الدم؛ معدل الوفيات؛ عوامل التنبؤ؛ الهند.

Advances in Knowledge

- *Elevated blood glucose levels have been previously identified as an indicator of poor prognosis in cases of aluminium phosphide (AlP) poisoning. However, the current study found that blood glucose level alterations—namely, hyper- or hypoglycaemia—were not associated with mortality in AlP cases.*

Application to Patient Care

- *Based on the findings of the current study, blood glucose level alterations should not be considered predictors of poor patient prognosis in AlP poisoning cases. Patients should therefore be assessed based on a range of clinical and laboratory parameters.*

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In India, aluminium phosphide (ALP) is the most commonly encountered poison after anticholinesterase.[1] As a pesticide, AlP is used extensively to protect stored products and crops and is available in the form of pellets, tablets or compressed discs. In northern India, an epidemic of AlP poisoning was reported in 1988 in which 285 cases of AlP ingestion were documented, most of which were intentional/suicidal.[2] Although the toxicokinetic properties of AlP are poorly understood, its toxicodynamic properties are exerted by phosphine (PH_3), which is released in the stomach when it comes into contact with moisture or hydrochloric acid and results in the inhibition of cytochrome C oxidase and a release of free oxygen radicals, causing oxidative stress.[3,4] After oral ingestion, PH_3 is quickly absorbed into the body, resulting in systemic toxicity, with death usually resulting from cardiovascular dysfunction.[5] Phosphides may also be absorbed in their unhydrolysed salt form.[6]

Apart from a definitive history of ingestion, a diagnosis of AlP poisoning can be established by the odour of the patient's breath, which often smells of garlic or decaying fish, although the former symptom is also seen in calcium carbide poisoning cases.[7–9] Other early symptoms include nausea, vomiting, epigastric and retrosternal pain, anxiety, agitation and dyspnoea.[7] In addition, the diagnosis can also be made by silver nitrate testing of the gastric fluid or breath.[9] Peripheral circulatory failure and shock are early signs indicating fatal toxicity.[7] The mortality rate in AlP poisoning cases varies from 30–100% and depends upon the dose and freshness of the poison, the onset of clinical manifestations, time until presentation, duration and severity of shock, time until vomiting and treatment modality. The main complications observed in fatal AlP poisoning cases are cardiac dysrhythmias, severe metabolic acidosis, shock, respiratory distress syndrome and hypomagnesaemia.[7]

Prognostic factors that may predict poor outcomes in AlP poisoning cases include hyperglycaemia, hypotension, acidosis, leukocytosis, hyperuricaemia, electrocardiography abnormalities, high Acute Physiology and Chronic Health Evaluation scores, high Simplified Acute Physiology Scores, low Glasgow coma scale scores, acute renal failure, low prothrombin rates, methaemoglobinaemia and the use of inotropes and mechanical ventilation.[10–14] Serial blood glucose monitoring is a convenient and easy method of assessing hypo- or hyperglycaemia, particularly in primary healthcare settings with limited resources. The current study aimed to determine the incidence of blood glucose level alterations in patients with AlP poisoning and the correlations between blood glucose levels and alterations and mortality.

Methods

This prospective observational study was conducted at the Postgraduate Institute of Medical Education & Research, a tertiary referral centre in Chandigarh, northeast India, from January 2010 to June 2011. All patients presenting to the emergency department during this period with a definitive history of AlP ingestion or symptoms compatible with AlP poisoning—including *vomitus* smelling of decaying fish or garlic, severe hypotension or shock, metabolic acidosis and abnormalities in cardiac rate or rhythm—were included in the study. Cases in which the ingestion of AlP was doubtful or with concomitant ingestion of other drugs or alcohol were excluded, as were patients with a history of dextrose administration prior to presentation or those with diabetes mellitus.

The vital signs of the patients were assessed, including their blood pressure (BP), heart rate and respiratory rate. Temperature and oxygen saturation were recorded using a pulse oximeter. Blood glucose levels were recorded using the Hitachi Modular Analytics System P800 chemistry analyser (Roche Diagnostics K.K., Tokyo, Japan), after appropriate calibration and quality control measures. Alterations in blood glucose levels were defined as the occurrence of either hyperglycaemia (random blood glucose levels of >200 mg/dL) or hypoglycaemia (random blood glucose levels of <55 mg/dL) at admission or any point during hospital stay.[15,16] A complete blood count was performed along with biochemical tests to determine serum sodium, potassium, urea, creatinine and bilirubin levels. Arterial blood gas (ABG) analyses were performed to determine pH, partial pressure of oxygen, bicarbonate value, partial pressure of carbon dioxide and base excess for the given fraction of inspired oxygen. All clinical and laboratory measurements were recorded at admission, every six hours thereafter for the first 72 hours and then every 24 hours until discharge. If necessary, certain variables were assessed more frequently, depending on the clinical condition of the patient.

All patients received standard supportive treatment, including basic emergency medical care and gastrointestinal decontamination (i.e. gastric *lavage*). Hypoglycaemic patients were treated with rapid intravenous infusions of two units of 100 mL of 25% dextrose solution and then maintained with 5% dextrose or 5% dextrose and normal saline solution, titrated according to electrolyte levels and glycaemic status.[17,18] A maximum of two units of 5% dextrose and normal saline solution up to a total of 500 mL daily were administered as a maintenance regimen for euglycaemic patients, titratred according to the patient's input-output status. Hyperglycaemic patients were treated initially according

Table 1: Characteristics of patients with aluminium phosphide poisoning (N = 116)

Variable	Mean ± SD		*P* value
	Survivors (n = 57)	Non-survivors (n = 59)	
Sociodemographic characteristic			
Age in years	28 ± 9	30 ± 11	0.399
Male gender, n (%)	38 (67)	33 (56)	0.235
Form of AlP ingested, n (%)			
Powder	40 (70)	51 (86)	
Tablet	11 (19)	3 (5)	0.051
Unknown	6 (11)	5 (9)	
Vital signs at admission			
Heart rate in bpm	101 ± 14	109 ± 11	0.001
SBP in mmHg	106 ± 29	77 ± 20	<0.001
Glasgow coma score	12.9 ± 0.1	2.3 ± 0.8	<0.001
Blood glucose measurements			
Blood glucose level* in mg/dL	119.9 ± 35.7	159.7 ± 92.5	<0.001
Frequency of blood glucose alterations,† n (%)	3 (5)	25 (42)	<0.001‡
Biochemistry results			
Serum sodium in mmol/L	142.5 ± 7.0	144.6 ± 7.4	0.058
Serum potassium in mmol/L	4.0 ± 0.7	4.1 ± 1.1	0.216
Serum creatinine in mg/dL	0.8 ± 1.0	1.4 ± 1.2	0.003
Serum bilirubin in mg/dL	0.9 ± 0.6	0.8 ± 0.5	0.116
TLC × 10^3 per mm^3	8.6 ± 3.4	12.2 ± 5.1	0.005
ABG measurements			
pH	7.3 ± 0.7	7.1 ± 1.3	<0.001
HCO_3	16.3 ± 4.8	11.3 ± 5.6	<0.001
Presentation/hospital stay characteristics			
Median interval between ingestion and presentation in hours (IQR)	4 (3–6)	4 (2–7)	0.816§
Median dextrose administered in g (IQR)	62 (25–125)	75 (43–106)	0.856§
Median LOS in hours (IQR)	36 (24–66)	5 (2–12)	<0.001§

SD = standard deviation; AlP = aluminium phosphide; bpm = beats per minute; SBP = systolic blood pressure; TLC = total leukocyte count; ABG = arterial blood gas; HCO_3 = bicarbonate; IQR = interquartile range; LOS = length of stay.

**Assessed via random blood glucose tests. †Defined as either hyperglycaemia (random blood glucose levels of >200 mg/dL) or hypoglycaemia (random blood glucose levels of <55 mg/dL). ‡Using a Mann-Whitney U test. §Using a Chi-squared test.*

to a continuous insulin infusion protocol and, subsequently, via a subcutaneous insulin regimen.[19]

The statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS), Version 22.0 (IBM Corp., Armonk, New York, USA). Various clinical and laboratory parameters were compared between survivors and non-survivors, with the primary variables of interest being blood glucose level at presentation and the frequency of blood glucose alterations at admission or during hospital stay. The normality of quantitative variables was determined using the Kolmogorov-Smirnov test. For normally distributed data, means and standard deviations were calculated for continuous variables while frequencies, percentages and proportions were calculated for qualitative variables. A t-test was used to compare the mean differences between the study groups and a Chi-squared test was used to compare proportions. Univariate and multivariate logistic regression analyses were conducted with mortality as the dependent variable. Independent variables found to be significant on the univariate analysis were included in the logistic regression model and analysed using the Enter method. Random blood glucose levels were considered to be a continuous variable in the univariate and multivariate regression analyses, whereas alterations in blood glucose levels were considered categorical (i.e. nominal) variables. A *P* value of <0.050 was considered statistically significant.

This study was conducted with prior approval from the institutional ethics committee. Written informed consent was obtained from all patients or their relatives prior to participation in the study.

Results

A total of 116 cases of AlP poisoning were identified. Of these, 59 patients (51%) were 15–25 years old, 32 (28%) were 26–35 years old, 14 (12%) were 36–45 years old, nine (8%) were 46–55 years old and two (2%) were over 55 years old. The most frequent symptoms were vomiting (99%) and epigastric pain (61%), followed by dyspnoea (41%) and altered *sensorium* (28%). The median length of hospital stay was 18 hours (range: 5–42 hours). At presentation, 12 (10%) patients had hypoglycaemia, 16 (14%) had hyperglycaemia and 88 (76%) had normal blood glucose levels.

In total, 57 patients (49%) survived and 59 patients (51%) died. The cause of death was refractory cardiogenic shock in 20 patients (34%), multiorgan dysfunction syndrome in six patients (10%), cardiac arrhythmias in five patients (8%) and respiratory failure in four patients (7%). The cause of death in the remaining 24 cases (41%) could not be determined. In terms of blood glucose alterations, all of the patients with hyperglycaemia, nine patients (75%)

Table 2: Logistic regression model to predict mortality among patients with aluminium phosphide poisoning (N = 116)

Predictor	Regression coefficient	SE	OR (95% CI)	*P* value
Heart rate	−0.009	0.050	0.991 (0.899–1.093)	0.860
SBP	−0.080	0.037	0.923 (0.859–0.992)	0.029
Glasgow coma score	−0.586	0.454	0.557 (0.229–1.355)	0.197
Blood glucose level*	0.012	0.009	1.012 (0.994–1.031)	0.187
Blood glucose alterations†	2.036	1.544	7.662 (0.372–157.919)	0.187
Serum creatinine level	0.293	0.594	1.340 (0.418–4.294)	0.623
TLC	0.000	0.000	1.000 (1.000–1.000)	0.913
ABG pH	−1.760	3.620	0.172 (0.000–207.369)	0.627
ABG HCO_3	−0.045	0.135	0.956 (0.734–1.246)	0.741
Duration of LOS	−0.094	0.035	0.910 (0.850–0.976)	0.008

SE = standard error; OR = odds ratio; CI = confidence interval; SBP = systolic blood pressure; TLC = total leukocyte count; ABG = arterial blood gas; HCO_3 = bicarbonate; LOS = length of stay.

**Assessed via random blood glucose tests. †Defined as either hyperglycaemia (random blood glucose levels of >200 mg/dL) or hypoglycaemia (random blood glucose levels of <55 mg/dL).*

with hypoglycaemia and 34 patients (39%) with normal blood glucose levels died.

Both mean blood glucose levels (159.7 ± 92.5 mg/dL versus 119.9 ± 35.7 mg/dL; *P* <0.001) and the frequency of blood glucose alterations (5% versus 42%; *P* <0.001) were significantly higher among non-survivors compared to survivors. This was also the case for heart rate (109 ± 11 beats per minute [bpm] versus 101 ± 14 bpm; *P* = 0.001), total leucocyte count (12.2 ± 5.1 × 10^3 per mm^3 versus 8.6 ± 3.4 × 10^3 per mm^3; *P* = 0.005) and serum creatinine levels (1.4 ± 1.2 mg/dL versus 0.8 ± 1.0 mg/dL; *P* = 0.003). In addition, non-survivors had significantly lower systolic BP at admission (77 ± 20 mmHg versus 106 ± 29 mmHg), Glasgow coma scale scores (2.3 ± 0.8 versus 12.9 ± 0.1), arterial blood gas pH (7.1 ± 1.3 versus 7.3 ± 0.7) and bicarbonate values (11.3 ± 5.6 versus 16.3 ± 4.8) and median duration of hospital stay (5 versus 36 hours) compared to survivors (*P* <0.001 each) [Table 1].

Statistically significant variables from the univariate analysis were subsequently included in a logistic regression model to predict mortality at a 95% confidence interval. Although systolic BP at admission (*P* = 0.029) and duration of hospital stay (*P* = 0.008) remained significant, neither blood glucose levels nor blood glucose alterations were found to be independent predictors of mortality (*P* >0.050 each) [Table 2].

Discussion

According to international recommendations, blood glucose levels should be maintained at 140–180 mg/dL for normoglycaemic patients and 180–200 mg/dL for previously diabetic individuals.[20] Both the hypo- and hyperglycaemic effects of AlP are attributable to the wide variety of changes in magnesium, calcium, phosphate, citrate and cortisol levels that result from AlP poisoning.[11,21] These biochemical changes can act as either stimulatory or inhibitory modulators of the enzymes and hormones that catalyse and regulate the metabolism of glucose. Therefore, depending on the type of biochemical change, AlP poisoning can result in the elevation or decrease of blood glucose levels or neither.[11,21]

Nevertheless, the specific mechanism by which AlP poisoning causes hypoglycaemia is poorly understood. Possible explanations include as a result of liver damage due to the release of PH_3 gas and the toxic effects of PH_3 on the adrenal cortex, leading to decreased cortisol levels.[22] In addition, certain symptoms of AlP poisoning such as recurrent vomiting and lack of appetite may also play a role in the development of hypoglycaemia. In contrast, hyperglycaemia occurs due to either the stimulation of cortisol, glucagon and adrenaline secretion or the inhibition of insulin synthesis.[11,21] A glucose-insulin-potassium infusion has been suggested as a potential therapeutic modality in the treatment of hyperglycaemia in AlP poisoning.[23]

Among critically-ill patients, blood glucose alterations have been associated with prolonged hospital stays as well as increased mortality.[20,24] However, to the best of the authors' knowledge, only one previous study has examined the relationship between hyperglycaemia and AlP poisoning outcomes. Mehrpour *et al.* published a prospective study of 45 patients with acute AlP poisoning, of which 32 patients (71%) died.[11] Non-survivors had significantly higher mean blood glucose levels than survivors (222.6 ± 20 mg/dL versus 143.4 ± 13.7 mg/dL; *P* = 0.021). Furthermore, blood glucose levels were an independent predictor of mortality in a multivariate analysis.[11] This contradicts the findings of the current study in which alterations in blood glucose levels (i.e. either hyperglycaemia or hypoglycaemia) were not independent predictors of mortality. This may be because Mehrpour *et al.* utilised a different cut-off value to indicate hyperglycaemia than that of the present study (>140 mg/dL versus >200 mg/dL).[11]

Strengths of the current study include its sample size and the evaluation of both hyper- and hypoglycaemia as predictors of mortality. However, the results are limited by the absence of any long-term follow-up of the survivors and the lack of analysis of other factors which may play a role in glucose homeostasis, such as serum calcium, magnesium, phosphate, insulin and cortisol levels.

Conclusion

As a result of several complex mechanisms, AlP poisoning can cause both hyper- and hypoglycaemia. However, the role of blood glucose levels and blood glucose alterations in predicting patient outcomes in AlP poisoning cases remains inconclusive. The authors recommend that further studies with larger sample sizes be conducted to evaluate other factors involved in glucose homeostasis.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

FUNDING

No funding was received for this study.

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