ORIGINAL ARTICLE

A SIMPLIFIED ACUTE PHYSIOLOGY SCORE IN THE PREDICTION OF ACUTE ALUMINUM PHOSPHIDE POISONING OUTCOME

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ABSTRACT

BACKGROUND: *Aluminum phosphide (AlP) is used as a fumigant. It produces phosphine gas, which is a mitochondrial poison. Unfortunately, there is no known antidote for AlP intoxication, and also, there are few data about its prognostic factors.* ***AIMS:*** *The aim of this study was to determine the impact of the Simplified Acute Physiology Score II ($SAPS_{II}$) in the prediction of outcome in patients with acute AlP poisoning requiring admission to the Intensive Care Unit (ICU).* ***MATERIALS AND METHODS:*** *This was a prospective study in patients with acute AlP poisoning, admitted to the ICU over a period of 12 months. The demographic data were collected and SAPSII was recorded. The patients were divided into survival and non-survival groups due to outcome.* ***STATISTICAL ANALYSIS:*** *The data were expressed as mean ± SD for continuous or discrete variables and as frequency and percentage for categorical variables. The results were compared between the two groups using SPSS software.* ***RESULTS:*** *During the study period, 39 subjects were admitted to the ICU with acute AlP poisoning. All 39 patients required endotracheal intubation and mechanical ventilation in addition to gastric decontamination with sodium bicarbonate, permanganate potassium, and activated charcoal, therapy with $MgSO_4$ and calcium gluconate and adequate hydration. Among these patients, 26 (66.7%) died. SAPSII was significantly higher in the non-survival group than in the survival group (11.88 ± 4.22 vs. 4.31 ± 2.06, respectively) ($P < 0.001$).* ***CONCLUSION:*** *SAPSII calculated within the first 24 hours was recognized as a good prognostic indicator among patients with acute AlP poisoning requiring ICU admission.*

Key words: *Aluminum phosphide poisoning, intensive care unit, mortality, phosphine, prognosis, simplified acute physiology score II*

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INTRODUCTION

Phosphides are used throughout the world as pesticides to protect stored grains from rodents and other pests.[1,2] Solid phosphides, including aluminum phosphide (AlP), form toxic phosphine gas following contact with water, moisture in the air, or hydrochloric acid in the stomach.[1-4]

During the past 35 years, high mortality rates have been reported following significant exposures to aluminum, zinc and calcium phosphides. AlP is known as "rice tablet" in Iran and marketed in 3 g tablets under brand name "Phostoxin". Incidence of AlP poisoning in Iran is also comparatively high.[5-7] Exposure is rarely accidental with the majority of cases involving intentional suicide acts.[8-12] In Loghman Hakim Hospital Poison Centre (LHHPC) from 1997 to 1998 and in 2003, we encountered 349 and 318 fatalities among 35,580 and 24,179 poisoned patients, respectively, over 12 years of age. Of these fatalities, 2.6 and 2.83%, respectively, cases were due to acute AlP poisoning.[9,13] As the incidence and also the mortality rate of AlP poisoning is high in Iran, it should raise the attention of the physician to the problem of acute AlP poisoning and it also necessitates the awareness of the public to the hazards of this poison.[14]

To the best of our knowledge, there are scant data on evaluating markers such as Glasgow Coma Scale (GCS), electrocardiogram (ECG), blood glucose level and scoring systems like Acute Physiology and Chronic Health Evaluation (APACHE) to predict mortality in acute AlP poisoning. Also, in some instances, the role of a single clinical and/or paraclinical finding is inconsistent.[15,16] So, we aimed to access the role of Simplified Acute Physiology Score II ($SAPS_{II}$) in estimating the outcome in these kinds of patients.

MATERIALS AND METHODS

This was a prospective study on acute AlP-intoxicated patients who were treated in the intensive care unit (ICU) of LHHPC as a teaching hospital , over a 12-month period from 1 April 2007 to 1 April 2008.

Acute AlP-intoxicated patients with no history of diabetes, cardiovascular, respiratory, renal and hepatic failure, and no advanced medical management for AlP poisoning in any medical center before admission were included in the study. Establishment of the diagnosis in all cases was based on the history of exposure and clinical manifestations, and other circumstantial evidence such as availability of a poison bottle or a label found by the relatives who brought the case to hospital.

All the patients received gastric decontamination with sodium bicarbonate (44 mEq, orally), permanganate potassium (1:10,000), and activated charcoal (1 g/kg, orally) in the first 6 hours after onset of poisoning in the Emergency Department (ED). All the patients required endotracheal intubation and mechanical ventilation and were admitted to ICU. They were treated with the same protocol (magnesium sulfate 4–6 g by IV infusion daily, calcium gluconate 4 g by IV infusion daily and adequate hydration) under the supervision of the same physicians and nurses. According to the situation of the patients, some of them were treated with

standard doses of vasopressor drugs like norepinephrine, and dopamine.

The qualifying case records were extracted from the ICU admission office. We collected and abstracted patients' information regarding gender, age, amount of AlP consumed, time between exposure and onset of treatment, signs and symptoms of intoxication on admission time, therapeutic intervention and laboratory tests including arterial blood gas (ABG), ECG, and outcome from the medical records onto a data sheet developed specifically for this study. Data were kept confidential in all stages of the study.

A detailed multiple variable database was created. All data were collected either as dichotomous variables (e.g., gender, outcome) or as numeric variables including continuous (e.g., potassium, sodium, etc.) or discrete (e.g., heart rate, GCS, etc.). GCS, as described by Teasdale and Jennet,[17] was calculated at admission on ED. The $SAPS_{II}$ was calculated in accordance with the original methodology,[18] using the worst physiologic values on the first ICU day. The score chart is shown in Table 1. All the patients were followed up until discharge from the hospital or death. According to the outcome, the patients were divided into survival and non-survival groups.

All data were analyzed with SPSS software version 12. The data were expressed as mean ± SD for continuous or discrete variables and as frequency and percentage for categorical variables. Chi-square test was used for statistical comparison of qualitative variables. The normal distribution of quantitative variables was tested by Kolmogorov–Smirnov test. The statistical comparison was done with Mann–Whitney *U*-test for nonparametric variables and independent student *t* test for parametric variables. *P* values of 0.05 or less were considered to be statistically significant.

The protocol of the study was approved by ethical committee of Shahid Beheshti University of Medical Sciences.

RESULTS

During the study period, according to inclusion criteria, 39 patients with acute AlP poisoning (21 men, 18 women), of age ranging from 14 to 62 years and with a mean age of 27.56 ± 11.95 years, were studied. In most of the patients (46.15%) the ingested amount was one tablet of AlP; average ingested amount was 1.38 ± 0.89 tablets with a range of 0.25–4 tablets. Average time elapsed between poisoning and admission at the hospital was 3.35 ± 3.5 hours (range 0.3–18 hours). Most of the patients (59%) had vomiting; and systolic blood pressure (SBP) less than 100 mmHg was observed in 37 (94.87%) cases at the time of admission (mean 80.64 ± 17.81 mmHg with the range of 50–130 mmHg). Also, ECG abnormality was found in 17 (43.59%) cases at the time of admission. The evaluation of ABG showed that the pH ranged between 6.7 and 7.55, with a mean value of 7.23 ± 0.19. Also, the results showed that most of the patients (43.59%) had pH between 7.15 and 7.35 at the time of admission. The mean of $SAPS_{II}$ was 9.36 ± 5.11 (range 1–19) [Tables 2 and 3].

Thirteen patients survived and 26 patients died. The mortality rate was about 66.7% (26/39). The route of exposure was deliberate ingestion

Table 1: Simplified acute physiology score chart

Parameter	*Findings*	*Points*
Age (years)	≤45	0
	46–55	1
	56–65	2
	66–75	3
	>75	4
SBP (mmHg)	≥190	4
	150–189	2
	80–149	0
	55–79	2
	<55	4
Heart rate (beat/min)	≥180	4
	140–179	3
	110–139	2
	70–109	0
	55–69	2
	40–54	3
	<40	4
Glasgow coma scale	13–15	0
	10–12	1
	7–9	2
	4–6	3
	3	4
Respiratory rate (breaths/min)	≥50	4
	35–49	3
	25–34	1
	12–24	0
	10–11	1
	6–9	2
	3–5	3
	<3	4
Body temperature (°C)	≥41	4
	39–40.9	3
	38.5–38.9	1
	36–38.4	0
	34–35.9	1
	32–33.9	2
	30–31.9	3
	<30	4
Urinary output (L/24 hours)	≥5	2
	3.5–4.99	1
	0.7–3.49	0
	0.5–0.69	2
	0.2–0.49	3
	<0.2	4
Hematocrit (%)	≥60	4
	50–59.9	2
	46–49.9	1
	30–45.9	0
	20–29.9	2
	<20	4
White blood cells count (1000/μL)	≥40	4
	20–39.9	2
	15–19.9	1
	3–14.9	0
	1–2.9	2
	< 1	4
Serum glucose (mg/dL)	≥800	4
	500–799	3
	250–499	1
	70–249	0
	50–69	2
	29–49	3
	<29	4
Serum potassium (mEq/L)	≥7	4
	6–6.9	3
	5.5–5.9	1
	3.5–5.4	0
	3–3.4	1
	2.5–2.9	2
	<2.5	4
Serum sodium (mEq/L)	≥180	4
	161–179	3
	156–160	2
	151–155	1
	130–150	0
	120–129	2
	110–119	3
	<110	4
Blood urea nitrogen (mg/dL)	≥154	3
	101–153	2
	81–100	1
	21–80	0
	10–20	1
	<10	1
Serum HCO_3 (mEq/L)	≥40	3
	30–39.9	1
	20–29.9	0
	10–19.9	1
	5–9.9	3
	<5	4

Table (contd...)

Table 1: (contd...)

Scores are obtained in the first 24 hours of admission; score = summation of the points for each item

Table 2: Distribution of patients due to age, sex, number of ingested AIP tablets, clinical and paraclinical manifestations

		Number		*Percent*	
Age (years)					
12–19		12		30.77	
20–39		20		51.28	
40–59		6		15.38	
≥60		1		2.57	
Sex					
Male		21		53.85	
Female		18		46.15	
Number of AIP tablets					
<1		8		20.51	
1		18		46.15	
2		8		20.51	
3		4		10.26	
4		1		2.57	
Clinical signs and symptoms					
Hypotension	SBP ≤ 70 mmHg	37	14	94.87	3784
	70 < SBP ≤ 90 mmHg		23		62.16
Vomiting			23		58.97
Tachycardia			16		41.03
Abdominal pain			12		30.77
Thirst			11		28.21
Bradycardia			8		20.51
Agitation			1		2.57
Paraclinical findings					
pH	pH < 7.15		13		33.33
	7.15 ≤ pH < 7.35		17		43.59
	7.35 ≤ pH < 7.45		5		12.82
	7.45 ≤ pH		4		10.26
ECG abnormality	ST-T change	17	8	43.59	47.6
	Ischemic change		3		17.65
	Dysrhythmia		6		35.29

in all patients. There was no significant difference between survival and non-survival groups in age, gender, ingested amount of AIP tablets, time interval between the onset of poisoning and admission at hospital, respiratory rate, serum sodium, potassium, HCO_3, and $PaCO_2$ at the time of admission to the hospital [Table 3].

As illustrated in Table 3, a significant difference was observed between survival and non-survival groups with respect to immediate vomiting after the onset of poisoning, GCS, SBP, pulse rate, serum pH, ECG abnormality, hematocrit, white blood cell count, blood glucose, serum blood urea nitrogen and $SAPS_{II}$ at the time of admission in the hospital.

DISCUSSION

AIP poisoning is a major health problem with a high mortality rate in Iran and other countries,

Table 3: Comparison of demographic, clinical, paraclinical parameters and $SAPS_{II}$ in survival and non-survival groups

Parameter (normal range)	*All patients (n=39)*	*Survival group (n = 13)*	*Non-survival group (n = 26)*	*P*
	Mean ± SD (range)			
Sex				
Male	21	7	14	1
Female	18	6	12	
Age (years)	27.56 ± 11.95 (14 – 62)	24.62 ± 7.56	29.65 ± 14.48	0.31
Systolic blood pressure (<140 mmHg)	80.64 ± 17.81 (50 – 130)	90.77 ± 11.88	75.58 ± 18.29*	0.01
Pulse rate (60 – 100 beats/min)	99.85 ± 29.78 (35 – 160)	115.69 ± 24.98	91.92 ± 29.20*	0.02
GCS	12.08 ± 0.62 (3 – 15)	13.92 ± 0.80	11.15 ± 0.79	0.01
Respiratory rate (12 – 20 breaths/min)	22.05 ± 9.77 (0 – 55)	19.62 ± 6.49	23.27 ± 10.96	0.27
Hematocrit (35 – 45%)	40.64 ± 5.95 (29.90 – 58.30)	36.38 ± 4.74	42.77 ± 5.37**	0.001
White blood cells count (7000 – 10,000/μL)	11,800 ± 3437.95 (4800 – 18,300)	10,046.15 ± 2923.93	12,676.92 ± 3385.65*	0.02
Blood glucose level (70 – 110 mg/dL)	198.69 ± 93.46 (68 – 432)	150.23 ± 55.58	222.92 ± 99.81*	0.04
Serum potassium (3.5 – 5.5 mEq/L)	3.92 ± 0.63 (2.60 – 6)	3.86 ± 0.43	3.95 ± 0.72	0.67
Serum sodium (135 – 155 mEq/L)	140.15 ± 5.14 (130 – 152)	136.69 ± 4.09	140.38 ± 5.65	0.70
Blood urea nitrogen (7 – 18 mg/dL)	20.51 ± 11.04 (11 – 60)	26.92 ± 10.64	17.31 ± 9.94*	0.002
Serum HCO_3 (22 – 26 mEq/L)	11.61 ± 4.38 (3.59 – 19)	12.30 ± 3.90	11.26 ± 4.64	0.49
SAPSII	9.36 ± 5.11 (1 – 19)	4.31 ± 2.06	11.88 ± 4.22**	0.001
$PaCO_2$ (35 – 45 mmHg)	28.15 ± 12.44 (16 – 65)	24.57 ± 8.23	29.93 ± 13.88	0.32
pH on admission time (7.35 – 7.45)	7.23 ± 0.19 (6.70 – 7.55)	7.31 ± 0.12	7.19 ± 0.20*	0.04
ECG				
Normal	32	11	11*	0.01
Abnormal	17	2	15	
Time interval between onset of poisoning and admission (hours)	3.35 ± 3.51 (0.30 – 18)	4.26 ± 4.87	2.90 ± 2.58	0.53
Number of ALP tablets	1.38 ± 0.89 (0.25 – 4)	1.04 ± 0.76	1.55 ± 0.91	0.05
The use of AlP tablets with water				
Positive	4	4	0*	0.003
Negative	35	9	26	
Immediately vomiting after ingestion				
Positive	23	11	12*	0.02
Negative	16	2	14	

Data are mean ± SD. The difference between survival and non-survival groups is significant at *$P < 0.05$ and **$P < 0.001$

especially in developing countries.[7,15,19] In this regard, one of the exigent issues in acute AlP poisoning is predicting its outcome. Unfortunately, to date, there have been inadequate studies on the clinical and paraclinical findings to determine the severity and outcome of acute AlP poisoning.[15,16]

In this study, we aimed to evaluate $SAPS_{II}$ which consists of clinical and paraclinical parameters that can be obtained rapidly in cases with AlP poisoning, and could help in determining the outcome of AlP poisoning.

In this study, the prognostic factors were $SAPS_{II}$, low GCS, hypotension, hyperglycemia, acidosis, hemoconcentration, leukocytosis, hyperuremia and ECG abnormalities. Our data were consistent with the results achieved in the previous studies.[15,16,19,20]

In some instances, there are inconsistent studies regarding the role of a single clinical and/or paraclinical finding to predict the outcome of acute AlP poisoning.[16,19,21,22] Scoring systems like $SAPS_{II}$, which is among the most used of scoring systems in ill patients, and evaluation of the general condition of patients during the first 24 hours[18] can be used to predict the outcome of the patients with acute AlP poisoning.

Hajouji Idrissi *et al*, (2006) evaluated the efficacy of $APACHE_{II}$ and $SAPS_{II}$ to determine the severity of AlP poisoning and found that they were positively correlated with poor outcome.[20] In another study, the predictive power of $APACHE_{II}$ was evaluated in AlP poisoning.[16] Both the $APACHE_{II}$ and the $SAPS_{II}$ scoring systems demonstrated an ability to predict the mortality rates in this type of poisoning. As $SAPS_{II}$ scoring system is a simplified version of the APACHEII scoring system, we evaluated this scoring system in our study, and the results showed a significant difference in $SAPS_{II}$ between the survival and non-survival groups.

Limitation

The limitation of our study was the small sample size with regard to the long duration of the study. So, further multicenter studies with larger samples need to be done in the future to conclusively support our results.

CONCLUSION

According to our findings and previous studies, it can be concluded that $SAPS_{II}$ scoring system is a reliable index to predict the outcome of the acute AlP poisoning.

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