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The Role of Central Venous Oxygen Saturation as a Prognostic Factor for Intensive Care Unit-Admitted Aluminum Phosphide-Poisoned Patients

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ABSTRACT

Background: Aluminum phosphide (ALP) is one of the most significant reasons of fatal poisoning globally. Many ALP-poisoned patients deteriorate even in high-level specialized hospitals with advanced life-support equipment & a known antidote do not exist.

Objectives: This study aimed to focus on the role of central venous oxygen saturation as a prognostic factor in aluminum phosphide-poisoned patients.

Patients and methods: A prospective study had been carried out on 84 AlP-poisoned cases who met the inclusion criteria. Clinical sheets had been created and contained data collection such as sociodemographic data, history and physical examination. Hemodynamic assessment, laboratory tests, left ventricular ejection fraction (LVEF%) by transthoracic echocardiography, and central venous oxygen saturation ($ScvO_2$) were measured on admission, following six hours of therapy, following 24 hours, and on discharge or mortality and were compared between non-survivors and survivors.

Results: A statistically significant variance had been detected among the groups under investigation in terms of LVEF% after 6 hrs, LVEF% after 24 hrs, LVEF% on discharge or death, $ScvO_2$ after 6 hrs, $ScvO_2$ after 24 hrs, and $ScvO_2$ on discharge or death. Cutoff value of 50.5 LVEF% on admission had a sensitivity of 73% & specificity of 77% for predicting AlP-poisoned patients' survival with a statistically significant difference. Cutoff value of 41.5 LVEF% after 6 hrs had a sensitivity of 80.5% and a specificity of 75% for expecting AlP-poisoned patients' survival with a statistically significant difference. Cutoff value of 47.5 LVEF% after 24 hrs had a sensitivity of 80.5% & a specificity of 74.4% for expecting AlP-poisoned patients' survival with a statistically significant difference. Cutoff value of 39 LVEF% on discharge or death had a sensitivity of 90.2% and specificity of 99% for expecting AlP-poisoned patients' survival with a statistically significant difference.

Conclusion: $ScvO_2$ had a prognostic role for expecting of mortality in cases that had ALP poisoning.

Keywords: ALP, ICU, LVEF, Poisoning, $ScvO_2$.

INTRODUCTION

Aluminum phosphide (AlP) is a fumigant that is utilized for protecting grains that are stored [1]. Aluminum phosphide is available in three-gram tablets, each of which contains 56% (total 1680 milligrams) aluminum phosphide and 44% ammonium carbonate [2, 3].

A highly toxic phosphine gas is produced when AlP is subjected to an acidic environment and moisture [4]. It is an essential cause of suicidal poisoning, which leads to a high rate of death [5].

There is a wide range of symptoms and signs related to acute AlP poisoning, as the toxin affects nearly all organs. Nausea, agitation, vomiting, dyspnea, anxiety, abdominal pain, and a garlic-like odor on the breath are among the initial symptoms. Tachycardia, tachypnea, acidosis, marked hypotension, and shock, which are typically unresponsive to conventional management, are additionally found. Drowsiness, delirium, and coma are the result of cerebral anoxia caused by shock, which persists until the cases' mental clarity becomes impaired. Arrhythmias, acute kidney failure, and disseminated intravascular coagulation are among the most prevalent complications of acute AlP toxicity. Furthermore, acute gastrointestinal hemorrhage, congestive heart failure, hepatitis, pericarditis, and pulmonary edema are occasionally observed [6].

Shock is described as worldwide tissue hypoxia that results from a disturbance among systemic oxygen demand (VO_2) and systemic oxygen delivery (DO_2). Death and morbidity are exacerbated by global tissue hypoxia that remains undiagnosed and unmanaged. Consequently, it is crucial to ensure that global tissue hypoxia is accurately detected. Physical results, vital signs, urinary output, and central venous pressure measurement are essential, but they are insufficient for the precise identification of global tissue hypoxia [7-9].

The indirect index of tissue oxygenation has been promoted through the determination of mixed venous oxygen saturation (SvO_2) from pulmonary artery [10]. Nevertheless, the utilization of pulmonary artery catheter has become somewhat disfavor as a consequence of a comprehensive literature review [11, 12].

In contrast, the insertion of a central venous catheter into the superior vena cava through jugular of the subclavian vein is regarded as standard care for cases who are critically diseased. The evaluation of central venous oxygen saturation ($ScvO_2$) has been recommended for the detection of global tissue hypoxia, similar to central venous oxygen saturation. The normal range for SvO_2 is between 60 and 75% [13].

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A negative outcome is predicted by low SvO_2 [14], while normal or supranormal SvO_2 (or $ScvO_2$) values don't ensure adequate tissue oxygenation [15].

In this investigation, we assessed the efficacy of $ScvO_2$ as a prognostic factor to inform the management of AlP-poisoned cases who were admitted to ICU at Menoufia University.

PATIENTS AND METHODS

A prospective study had been carried out at the Menoufia University Intensive Care Unit between April 1, 2023, and March 31, 2024, on 84 AlP-poisoned cases who met the inclusion criteria.

Inclusion criteria: Cases who had a history of ingesting an AlP tablet, cases who were over the age of eighteen clinical results, & a confirmed positive silver nitrate test (SNT) that has been conducted in the Emergency Department at the time of admission. Despite a negative SNT, case was additionally classified as Aluminum phosphide-poisoned if they had a history of prior Aluminum phosphide ingestion (as indicated by the case or their closest relative) & presented with relevant clinical manifestations (SBP less than eighty millimeters of of mercury/; serum HCO_3 less than fifteen meq/L; pH less than 7.2), as the test is relatively low-sensitivity & might produce false-negative outcomes for specific cases.

Exclusion criteria: Individuals under the age of eighteen have been excluded from the investigation. Ingestion of AlP tablets that have been exposed to air, The investigation excluded cases with any pre-existing medical condition, as diabetes mellitus, heart failure, & renal failure, as well as those with a doubtful history, absence of all clinical features of poisoning, with aluminum phosphide & the presence of concomitant poisoning with any other agent.

On admission, all patients received treatment (Initial resuscitation with fluids then inotropes, and vasopressors if needed) and according to their Glasgow coma scale (GCS) patients with GCS less than eight were intubated and mechanically ventilated. Clinical sheets were created and contained data collection such as sociodemographic information (age, sex, and residence), and route of administration. The initial evaluation of the individuals consisted of taking a history, a physical examination, a conscious level assessment and a hemodynamic assessment (diastolic blood pressure, arterial oxygen saturation, heart rate, systolic blood pressure and respiratory rate). Transthoracic echocardiography was done and LVEF% was measured on admission, following six hours, 12 hours, after 24 hours and on discharge or death. A central venous line was inserted and $ScvO_2$ was measured on admission, after 6 hours, following twenty-four hours, and on discharge or death. Laboratory tests (Hemoglobin level, total leucocyte count, liver enzymes, urea, creatine, sodium level, potassium level, INR, CO_2, pH & serum bicarbonate level) were recorded. According to the mortality, cases had been categorized into two groups: Non-survivors and survivors. All data were compared between the 2 groups.

Ethical approval: The authorization granted by the Institutional Review Board of Menoufia University (trial registration 3/2023FORE8) and Declaration of Helsinki & National Guidelines have been followed. After outlining the goal of the study, written informed consents were taken from the case's legal surrogates. To protect privacy, patient's data were kept anonymous.

Statistical analysis

The statistical package of the social science software (SPSS), version 21, was utilized to collect, tabulate, and analyze all data. Numbers and percentages have been utilized to characterize qualitative data. The mean and standard deviation were utilized to describe quantitative data. The significance level for all statistical comparisons was two-tailed, with a P-value $\leq$ 0.05 indicating significance, p -value $\leq$ 0.001 revealing a highly significant distinction, and P-value > 0.05 indicating a non-significant variation. The cutoff values of various research variables that could predict the survival of AlP-poisoned cases were investigated using ROC curve analysis. A binary logistic regression analysis was conducted to predict the survival of cases who were exposed to AlP. The Chi-square (X^2) test of significance was utilized to compare the proportions of qualitative variables, while the independent T-test was utilized to compare the results of two groups with parametric quantitative data.

RESULTS

As shown in table (1) young adults were involved in both the non-survivors group (mean = 29.21 years) and survivors (mean = 25.17 years). Nevertheless, the detected age differences didn't reach statistical significance (p -value equal 0.06). Regarding gender, 58.5% were males, while 41.5% were females in group 1, and 27.9% were males while 72.1 % were females in group 2.

There was no significant sex difference among cases under investigation. Regarding residence, 56.1% were from rural areas whereas 43.9% were from urban areas in group 1, while 58.1% were from rural areas and 41.9% were from urban areas in group 2. All patients of ALP poisoning were intentional, and there was insignificant variance among both groups with regard to mode of poisoning.

Table (1): Distribution of demographic data between the studied groups

	Group 1 Survivors (N=41)	**Group 2 Non-Survivors (N=43)**	**P value**
	Group 1 Survivors (N=41)	Group 2 Non-Survivors (N=43)	P value
Age (Years) Mean ±SD	25.17±7.4	29.21±11.61	0.06
Sex			
Male	24 (58.5%)	18 (27.9%)	0.126
Female	17 (41.5%)	35 (72.1%)	
Marital status			
Single	41 (100%)	43 (100%)	1
Residence			
Rural	23 (56.1%)	25 (58.1%)	0.85
Urban	18 (43.9%)	18 (41.9%)	
Manner of poisoning			
Suicidal	41 (100%)	43 (100%)	1

As shown in table (2), there was statistically insignificant variance among the groups under investigation with regard to temperature, while a statistically significant variance was observed among the groups under investigation with regard to SBP, DBP, pulse rate, and respiratory rate.

Table (2): Distribution of vital signs between the studied groups

	Group 1 Survivors (N=41)	**Group 2 Non-Survivors (N=43)**	**P value**
SBP (mmHg) Mean± SD	99.6±15.5	75.2±15.3	**<0.001**
DBP (mmHg) Mean± SD	65.3±9.2	44.3±9.1	**<0.001**
Pulse Rate (Beat/min) Mean± SD	106.8±7.9	116.4±6.3	**<0.001**
Temperature (^{o}C) Mean± SD	36.7±0.4	36.8±0.4	0.244
Respiratory rate (Number/min) Mean± SD	18.5±0.7	19.2±0.4	**<0.001**

As shown in table (3**),** there was statistically insignificant alteration among the group under investigation with regard to WBCs, and Hb, while a statistically significant variance was detected among the groups under investigation with regard to SGOT, SGPT, urea, sodium, potassium, magnesium, O_2, HCO_3, INR, creatinine, CO_2, and PH.

Table (3): Distribution of laboratory investigations between the studied groups

	Group 1 Survivors (N=41)	**Group 2 Non-Survivors (N=43)**	**P value**
WBCs (10^3/mm^3) Mean ±SD	6.57±4.8	6.48±3.6	0.92
Hb (gm/dl) Mean ±SD	13.31±1.2	13.31±1.2	0.99
SGOT (IU/l) Mean ±SD	31.15±20.9	66.16±21.66	**<0.001**
SGPT (IU/l) Mean ±SD	30.07±6.01	34.44±3.66	**<0.001**
Urea (mg/dl) Mean ±SD	23.39±8.57	26.65±6.73	**0.05**
Creatinine (mg/dl) Mean ±SD	0.78 ±0.19	1.32 ±0.24	**<0.001**
Sodium (mEq/L) Mean ±SD	138.29±3.68	129.26±8.23	**<0.001**
Potassium (mEq/L) Mean ±SD	4.59±0.47	3.86±0.87	**<0.001**
Magnesium (mg/dL) Mean ±SD	2.49 ±0.41	2.32 ±0.4	**0.05**
CO_2 (mmHg) Mean ±SD	31.35 ±7.5	36.08 ±6.64	**0.003**
O_2 (mmHg) Mean ±SD	73.43 ±23.3	47.78 ±4.60	**<0.001**
HCO_3 (mmol/L) Mean ±SD	19.94 ±4.25	10.66 ±4.14	**<0.001**
PH Mean ±SD	7.35 ±0.09	7.21±0.16	**<0.001**
INR Mean ±SD	1.39±0.28	1.6±0.27	**0.001**

As shown in table (4), there was statistically insignificant variance among the groups under investigation with regard to LVEF% on admission, and $ScvO_2$ on admission. While, a statistically significant variance was detected among the groups under investigation with regard to LVEF% after 6 hrs, LVEF% after 24 hrs, LVEF% on discharge or death, $ScvO_2$ after 6 hrs, $ScvO_2$ after 24 hrs, and $ScvO_2$ on discharge or death.

Table (4): Distribution of central venous oxygen saturation (ScvO2) and LVEF% between the studied groups

	Group 1 Survivors (N=41)	**Group 2 Non-Survivors (N=43)**	**P value**
LVEF% on admission Mean± SD	35.9±10.0	33.0±9.6	0.178
LVEF% after 6 hrs Mean± SD	46.0±5.6	33.5±6.4	**<0.001**
LVEF% after 24 hrs Mean± SD	50.4±3.4	35.6±8.1	**<0.001**
LVEF% at discharge or death Mean± SD	51.9±8.9	33.1±5.1	**<0.001**
$ScvO_2$ on admission Mean± SD	58.96 ±9.65	55.63±12.4	0.174
$ScvO_2$ after 6 hrs Mean± SD	71.61 ±10.63	55.62±12.8	**<0.001**
$ScvO_2$ after 24 hrs Mean± SD	80.7±10.5	53.26±8.1	**<0.001**
$ScvO_2$ on discharge or death Mean± SD	88.8±12.0	50.62±9.21	**0.001**

As shown in table (5), at a cutoff value of 50.5, EF% at admission had a sensitivity of 73% & a specificity of 77% for expecting AlP-poisoned patients' survival with a statistically significant difference. Cutoff value of 41.5 EF% after 6 hrs had a sensitivity of 80.5% and a specificity of 75% for expecting AlP-poisoned patients' survival with statistically significant difference.

Cutoff value of 47.5 EF% after 24 hrs had a sensitivity of 80.5% and a specificity of 74.4% for expecting of AlP-poisoned patients' survival with statistically significant difference. Cutoff value of 39 EF% at discharge or death had a sensitivity of 90.2% and a specificity of 99% for expecting of AlP-poisoned patients' survival with statistically significant difference. At cutoff value of 85, $ScvO_2$ at admission had a sensitivity of 51% & a specificity of 30% for expecting AlP-poisoned patients' survival with a statistically significant difference. Cutoff value of 80 $ScvO_2$% after 6 hrs had a sensitivity of 51% and a specificity of 35% for expecting of AlP-poisoned patients' survival with no statistically significant difference.

Cutoff value of 71 $ScvO_2$ after 24 hrs had a sensitivity of 63.4% and a specificity of 67.4% for expecting of AlP-poisoned patients' survival with no statistically significant difference. Cutoff value of 52 $ScvO_2$ at discharge or death had a sensitivity of 95.1% and a specificity of 88.4% for expecting of AlP-poisoned patients' survival with a statistically significant variance.

Table (5): ROC curve of EF%, and $ScvO_2$for predicting of AlP-poisoned patients' survival

						95% CI	
	Cutoff	**AUC**	**Sensitivity**	**Specificity**	**Sig.**	**Lower**	**Upper**
LVEF% on admission	50.5	0.697	73%	77%	0.002	0.571	0.822
LVEF% after 6 hrs	41.5	0.746	80.5%	75%	<0.001	0.635	0.856
LVEF% after 24 hrs	47.5	0.802	80.5%	74.4%	<0.001	0.700	0.905
LVEF% on discharge or death	39	0.943	90.2%	99%	<0.001	0.884	1
SCVO2 on admission	85	0.259	51%	30%	<0.001	0.150	0.367
SCVO2 after 6 hrs	80	0.544	51%	35%	0.491	0.413	0.675
SCVO2 after 24 hrs	71	0.569	63.4%	67.4%	0.279	0.441	0.696
SCVO2 on discharge or death	52	0.960	95.1%	88.4%	<0.001	0.906	1

According to table (6), the median GCS was 13 in group 1 and 7 in group 2. In terms of the necessity for mechanical ventilation, 41% of patients in group 1 required MV, while 72% of patients in group 2 required MV. Upon admission to the intensive care unit, 50% of cases needed mechanical ventilatory support, while noninvasive ventilation has been utilized in only one cases.

Table (6): Distribution of GCS, and mechanical ventilation between the studied groups

	Group 1 Survivors (N=41)	**Group 2 Non-Survivors (N=43)**	**P value**
GCS Mean ±SD	13.44±1.93	7.84±4.38	**<0.001**
Mechanical ventilation			
Yes	17 (41.5%)	31 (72.1%)	**0.004**
No	24 (58.5%)	12 (27.9%)	

As shown in table (7), the distribution of medical management between the studied groups. There was a statistically insignificant variance among the groups under investigation with regard to inotropes (dobutamine). While, a statistically significant variance has been detected among the groups under investigation with regard to vasopressors (norepinephrine).

Table (7): Distribution of medical management between the studied groups

	Group 1 Survivors (N=41)	**Group 2 Non-Survivors (N=43)**	**P value**
Inotropes and vasopressors			
Dobutamine	41(100%)	43(100%)	1
Norepinephrine	3(7.3%)	32(74.4%)	**<0.001**

As shown in figure (1), LVEF% on admission had a sensitivity of 73% and a specificity of 77% for expecting of AlP-poisoned patients' survival with statistically significant difference. LVEF% after 6 hrs had a sensitivity of 80.5% and a specificity of 75% for expecting of AlP-poisoned patients' survival with statistically significant difference.

LVEF% after 24 hrs had a sensitivity of 80.5% and a specificity of 74.4% for expecting of AlP-poisoned patients' survival with statistically significant difference. LVEF% on discharge or death had a sensitivity of 90.2% and a specificity of 99% for expecting of AlP-poisoned patient survival with statistically significant difference.

$ScvO_2$ on admission had sensitivity of 51% and specificity of 30% for expecting of AlP-poisoned patients' survival with statistically significant difference. $ScvO_2$% after 6 hrs had a sensitivity of 51% and a specificity of 35% for predicting of AlP-poisoned patients' survival with no statistically significant difference.

$ScvO_2$ after 24 hrs had a sensitivity of 63.4% and a specificity of 67.4% for expecting of AlP-poisoned patients' survival with no statistically significant difference, and $ScvO_2$ on discharge or death had a sensitivity of 95.1% and a specificity of 88.4% for expecting of AlP-poisoned patients' survival with statistically significant difference.

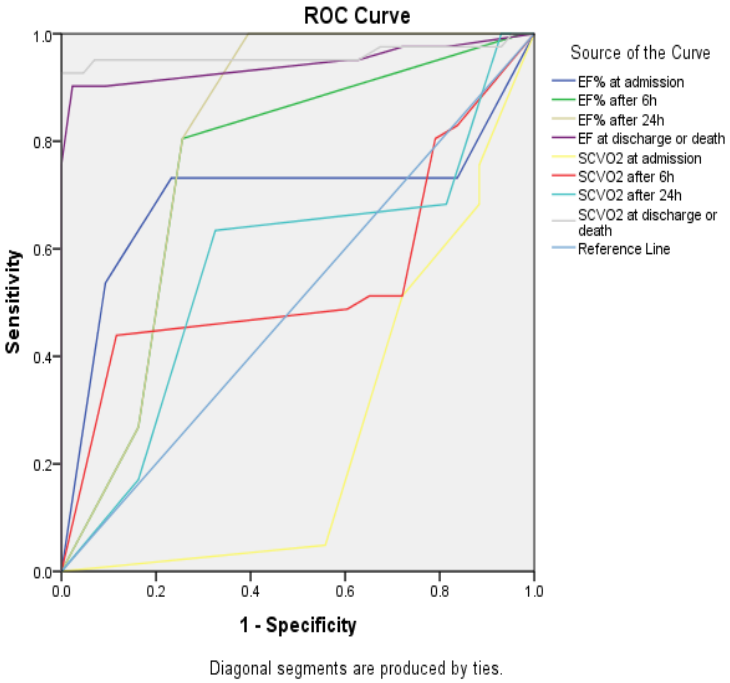

Figure (1): ROC curve of LVEF%, and $ScvO_2$ for predicting of AlP-poisoned patients' survival

As shown in figure (2)**,** GCS had a sensitivity of 73.2% and a specificity of 89% for expecting AlP-poisoned patient survival with a statistically significant difference. SBP had a sensitivity of 85.4% & a specificity of 62.8% for expecting AlP-poisoned patients' survival with statistically significant difference. HCO_3 had a sensitivity of 65.9% and a specificity of 86% for predicting of AlP-poisoned patient survival with a statistically significant difference.

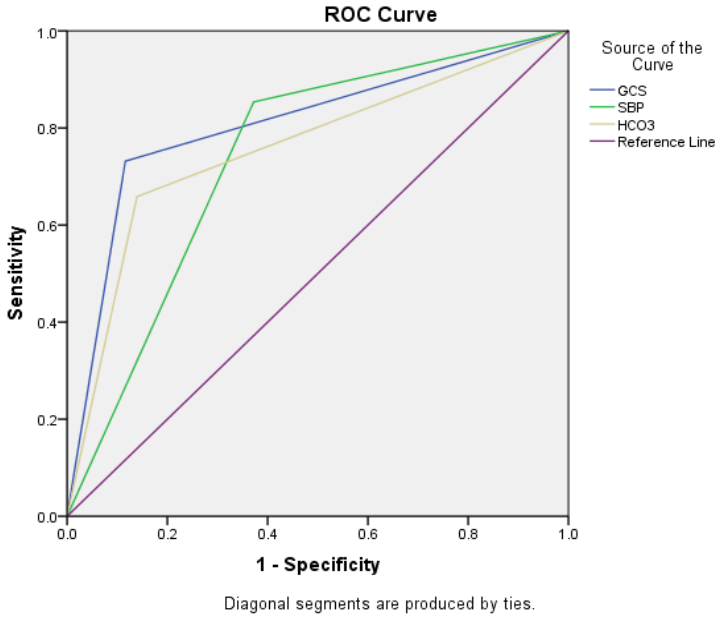

Figure (2): ROC curve of GCS, SBP, and HCO_3 for predicting of AlP-poisoned patients' survival.

DISCUSSION

The frequency of acute aluminum phosphide poisoning has been steadily rising in current years, particularly in developing countries, as a result of its widespread availability and low cost. This has resulted in elevated rates of death [16].

According to our outcomes, there was a statistically insignificant variance among the groups under investigation with regard to gender, age, marital status, or residence. These results are in line with **Sheta *et al.*** [17] who found insignificant variance has been observed among non-survivors and survivors with regard to sex, residence, circumstances of exposure, occupation, and age.

In opposite to **Sulaj *et al.*** [18] who stated that the greatest frequency of death (15.7%) has been observed in the sixteen to nineteen years age group with a women preponderance. **Khurana *et al.*** [19] discovered that the most of acute aluminum phosphide poisoned cases were between the ages of twenty and thirty, with a men dominance.

The majority of cases in both groups were from rural areas. This is in agreement with **Moghadamnia** [20]**.** This may be due to the easy availability of phosphides in rural areas, failure in education, and financial problems.

All patients of aluminum phosphide poisoning were intentional to commit suicide among the younger productive age group of society. Cases who were unintentionally subjected were not encountered. The same results were obtained by **Navabi *et al.*** [21]**.** In our region, the availability and affordable price of AlP tablets have made it a common method of suicide. Another contributing factor might be the absence of an antidote to assist cases who have been subjected to AlP and the lack of awareness regarding its severity [20]. The mode of poisoning did not exhibit a significant distinction among both groups.

The statistical analysis revealed significant differences between both groups as regards pulse rate, diastolic and systolic blood pressure, and respiratory rate. Similar results are obtained by **Erfantalab *et al.*** [22] who found that the blood pressure & pulse rate of AlP survivors & non-survivors were significantly distinct.

Additionally, **Wahab *et al.*** [23] concluded that the severity of hypotension that the cases develop is the most significant determinant of the prognosis from AlP. It is possible that the cause of hypovolemic shock is the massive loss of intravascular fluid that occurs as a consequence of insufficiency of vascular wall following the absorption of phosphine gas. Additionally, the direct cardiotoxic impacts of phosphine gas result in a significant collapse of the circulatory system [16].

The 1^{ry} lethal consequence of aluminum phosphide ingestion, which is profound circulatory collapse, is believed to be 2^{ry} to the toxins that are produced. These toxins have direct impacts on fluid loss, adrenal gland and cardiac myocytes injury. Furthermore, phosphine has corrosive impacts on tissues [24].

Concerning Arterial Blood Gases (ABG), serum HCO_3 & pH levels were very low in the non-surviving group compared to the surviving group. Such result is in agreement with those of **Jamshidi *et al.*** [25]**.** All manifested cases developed metabolic acidosis, but this was more severe in non-survivors, and it was directly proportional to severity of clinical manifestations. This is in accordance with the outcomes of **Abdel Wahab *et al.*** [26] who explained that lactic acidosis was secondary to hypoperfusion and poor tissue perfusion and inhibition of oxidative phosphorylation that results from the irreversible shock.

In agreements with **Hosseinian *et al.*** [27]**,** the current research illustrated a significant correlation among O_2 saturation and death, as non-survivors had lower O_2 saturation compared to survivors.

The present investigation has shown that nonsurvivors had significantly lower concentration of serum potassium compared to survivors, which is consistent with the outcomes of **Boukatta *et al.*** [28].

The present investigation demonstrated that death was significantly correlated with increased ALT, serum creatinine, blood urea, and AST. These findings are consistent with those of **Louriz *et al.*** [29]**.** Furthermore, **Mathai & Bhanu** [24] showed that death was correlated with increased serum creatinine.

In accordance with **Ghonem *et al.*** [30]**,** this investigation discovered that the respiratory rate was significantly elevated and oxygen saturation has been significantly diminished in non-survivors.

The current investigation illustrated that death was significantly correlated with the requirement for mechanical ventilation and administration of vasoactive medications, including dobutamine and noradrenaline. These outcomes are consistent with the findings of **Louriz *et al.*** [29] and **Mathai & Bhanu** [25]**.**

According to our investigation, there was statistically insignificant variance among the groups under investigation with refed to LVEF% on admission, and $ScvO_2$ on admission. While, a statistically significant variance has been detected among the groups under investigation with regard to LVEF% after 6 hrs, LVEF% after 24 hrs, LVEF% on discharge or death, $ScvO_2$ after 6 hrs, $ScvO_2$ after 24 hrs, and $ScvO_2$ on discharge or death. In the form of hypokinesia or akinesia of left ventricle and septum or a diminished ejection fraction, many investigations have illustrated severe cardiac dysfunction as a result of being exposed to AlP poisoning [31, 32]**.**

Gallet *et al.* [33] conducted an investigation on the prognostic value of central venous oxygen saturation in acute decompensated heart failure. They discovered that in cases admitted for acute decompensated heart failure (ADHF) who require inotrope support, central venous oxygen saturation not more than sixty percent is a warning sign of a poor

results and may suggest the need for more aggressive treatment.

A study by **De Saint-Aurin *et al.* [34]** found that change in central venous oxygen saturation under diuretic & intra-venous inotropic management is a potent predictor of findings in cases that have cardiogenic shock. **Ho *et al.* [35]** discovered that central venous oxygen saturation has significant capacity to predict the global cardiac output state & might be beneficial in the regulation of hemodynamic resuscitation protocols. The low cardiac output state that is related to a low central venous oxygen saturation might, at least in part, elucidate why a low central venous oxygen saturation is related to a worse prognosis in many different of clinical scenarios [35].

As shown these studies demonstrated the role of $ScvO_2$ in the prediction of the outcome of patients with impaired cardiac function, and as shown by **Mohan *et al.* [31]** and **Mehrpour *et al.* [32]** who illustrated severe cardiac dysfunction after exposure to AlP.

Also, as shown in our results the group of non-survivors had lower LVEF% and lower $ScvO_2$ than that of survivors. $ScvO_2$ can be used as a predictor of results in cases that had Aluminum phosphide poisoning.

CONCLUSION

$ScvO_2$ had a prognostic role with other factors for expecting of death in cases that had ALP poisoning.

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