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# Venoarterial Carbon Dioxide Gradient and Central Venous Oxygen Saturation as a Prognostic Value in Severe Intraabdominal Sepsis

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## ABSTRACT

**Background:** For physicians, the prognostic value of central venous oxygen saturation ($ScvO_2$) and venoarterial carbon dioxide difference ($PvaCO_2$) among severe sepsis patients remains unclear.

**Objective:** The aim of this study was for assessment of the prognostic value of $PvaCO_2$ and $ScvO_2$ among severe intraabdominal sepsis.

**Patients and methods:** This prospective study included 62 patients with intraabdominal sepsis who were presented at the Emergency Hospital Mansoura University for one year duration. $PvaCO_2$ and $ScvO_2$ values were assessed at admission and every 6 hours till the end of 48 hours.

**Results:** The $PvaCO_2$ showed significant high levels among the survivor group compared to with the mortality group at 6-48 Hours. Among the included patients, the non survivors tended to have either above or below normal levels of $ScvO_2$ at 6-48 hours. Multivariate analysis showed that decreased $PvaCO_2$, increased APACHE-II score, increased SOFA score and increased qSOFA score at admission were shown as independent risk predictors for mortality.

**Conclusion:** Elevated $PvaCO_2$, above and below normal $ScvO_2$ levels are associated with poor outcomes and raised risk of mortality. Thus, they hold promise as prognostic values in severe sepsis patients.

**Keywords:** Intraabdominal Sepsis, $PvaCO_2$, $ScvO_2$.

## INTRODUCTION

In critically ill patients, inadequate tissue perfusion is a major factor in the onset and course of MOF. The majority of tissue perfusion monitoring techniques now in use have mostly focused on general blood circulation and the equilibrium between supply and demand for oxygen. It has been demonstrated that early hemodynamic optimization, with an emphasis on systemic hemodynamic parameters and central venous oxygen saturation, improves outcomes in severe sepsis and septic shock, highlighting the relationship between tissue perfusion abnormalities and blood flow in the early stages of illness [1]. However, reaching normal systemic hemodynamic parameters does not ensure sufficient tissue perfusion; in fact, many patients who meet $ScvO_2$ objectives still have multi-organ dysfunction and mortality. Furthermore, some research has shown that even in the presence of regional and tissue perfusion abnormalities, oxygen-related measures like central venous oxygen saturation ($ScvO_2$) often return to normal upon admission to the ICU, and that interventions like urgent intubation can quickly restore $ScvO_2$ levels [2].

In the past, scientists have seen that after cardiac arrest, venous acidosis and elevated venous ($CO_2$) levels coexist in both critically ill humans and animals. Consequently, in situations like hypovolemic, cardiogenic, obstructive, and septic shock, an increase in ($PvaCO_2$) has been seen. It's interesting to note that a nonlinear relationship has been found between $PvaCO_2$ and COP, highlighting the role that blood flow plays in the buildup of venous $CO_2$. $PvaCO_2$ has attracted therapeutic interest as a global perfusion indicator during shock states as a result of this [3].

Further evidence indicates that raised $PvaCO_2$ levels may detect septic patients who meet oxygen-related goals but do not receive adequate resuscitation, highlighting the significance of $PvaCO_2$ as an overall perfusion indicator because it can monitor blood flow variations and even identify the production of anaerobic $CO_2$ [4]. Moreover, the dynamics of $PvaCO_2$ in the early phases of septic shock resuscitation have not been thoroughly studied, and recent research aiming to confirm the effectiveness of $PvaCO_2$ as a tool in septic patient resuscitation may be biased due to selection bias because not all qualifying patients received goal-directed therapy and catheter insertion [5-8].

The aim of this study was to assess the prognostic value of venoarterial $CO_2$ gradient and central venous oxygen saturation among severe intraabdominal sepsis.

## PATIENTS AND METHODS

### Subjects

This was a prospective observational study that was conducted at the Emergency Department, Mansoura University Hospitals within the period from January 2022 to January 2023.

**Inclusion Criteria:** According to the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-III) [9], septic patients with severe intraabdominal sepsis who were older than 18 years old and met at least two of the following criteria (disturbed consciousness, respiratory rate of 22/min or higher, or SBP ≥100 mmHg were included in the current study.

**Exclusion Criteria:** pregnancy and patients with advanced tumors or irreversible organ failure,



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contraindication to central venous catheter and brain death.

**Methods**

**Study procedures:** All participants underwent the following procedures:

**Full history:** stressing on socio-demographic data, history suggestive of acute or chronic medical conditions, history suggestive of infections or surgeries within one-month period, history of recent administration of drugs, nutritional history and family HX.

**Full examination:** including general and systemic examination.

**Laboratory investigations:** CBC, (ABGs) daily, serum electrolytes, liver function tests, serum creatinine and serum lactate.

**Imaging:**

Pelviabdominal ultrasound was done to assess the intraabdominal organs and microbiologic culture and sensitivity was done (when indicated).

**Patients' assessment:**

Neurological state assessment was conducted by (qSOFA) score, (SOFA) score and APACHE II score.

**General Management:** all patients were subjected to the following:

Each patient received an arterial cannula and a CV catheter. Our early goal-directed therapy encompassed a set of interventions with the following objectives: maintaining a mean arterial pressure of at least 65 mmHg, ensuring a urine output of at least 0.5 ml/kg/minute. The administration of vasopressors followed a standardized protocol to maintain MAP at or more-than sixty-five mmHg. To minimize the required dosage of vasopressors and optimise stroke volume, we repeated fluid challenges using either crystalloids or colloids. Dobutamine was administered when $ScvO_2$ stayed consistently below seventy percent following fluid resuscitation, as long as the hematocrit level was below thirty percent. If the use of the vasopressor persisted after sufficient fluid replacement, a low dose of fifty milligrams of hydrocortisone was given within 6 hours of the resuscitation. When necessary, mechanical ventilation was started along with fentanyl for analgesia and midazolam for mild sedation. The maximum tidal volume allowed was 6–8 ml/kg. Furthermore, venous thrombosis prophylaxis and stress ulcer treatment were provided in compliance with international guidelines.

**Study protocol:**

Regarding the study protocol, blood samples were collected from both the CV line and the arterial catheter at specified time points: T0, 6 hr (T6), 12 hr (T12), 18 hr (T18), 24 hr (T24), 30 hr (T30), 36 hr (T36), 42 hr (T42), and 48 hr (T48) after the initiation of treatment. The measured parameters included ($ScvO_2$), and the calculation of Pcv-aCO2, which represents the difference between the partial pressure of carbon dioxide in the central venous and arterial blood. Furthermore, we regularly assessed SOFA, qSOFA, and APACHE scores.

**Ethical Considerations:**

**The study protocol was approved by El Mansoura University's Faculty of Medicine Research Ethics Committee prior to the start of fieldwork. Every patient gave their written informed consent. The Helsinki Declaration was followed throughout the study's conduct.**

***Statistical Analysis***

SPSS version 26 for Windows® was used to code and analyze the collected data (IBM, Chicago, IL, USA). Numbers (frequency) and percentages representing qualitative data were displayed. To compare across groups, the Chi-Square test was applied. The Kolmogorov-Smirnov test assessed the normalcy of quantitative data. The mean±SD was used to represent the data. When comparing two groups of students with regularly distributed quantitative variables, the independent samples (students') t-test was utilised; if the data collection revealed an anomalous distribution, the Mann-Whitney U-test was employed. Using the Youden index J, the furthest point on the ROC curve, the ideal cutoff value of the quantitative variable to distinguish between different groups was found and stated in terms of sensitivity and specificity. The independent risk predictors of the categorical outcomes were tested using multivariate logistic regression analysis. P values less than 0.05 are considered significant.

**RESULTS**

The current study comprised 62 cases with severe intraabdominal sepsis. The mortality rate in this study was 64.5%. We divided the included patients into 2 groups: Group A: the survivors (N=22) and Group B: mortality group (N=40).

**Table (1)** shows that there were no significant differences between the 2 groups regarding, age, sex, BMI, and the cause of sepsis. DM was statistically significantly more common among non-survivors. GCS was higher among the survivors with a statistically significant difference. Patients who died tended to be more tachypneic than the surviving group, with a statistically significant discrepancy in the vital signs of the included groups.

**Table (1):** Clinical and demographic data of the included patients.

| Variables | Survived (N=22) | Died (N=40) | P value |
|---|---|---|---|
| **Age/years** | 48.27±8.12 | 50.58±8.84 | 0.317 |
| **Sex** | | | |
| Male | 10(45.5) | 16(40) | 0.677 |
| Female | 12(54.5) | 24(60.0) | |
| **BMI(Kg/m$^2$)** | 27.32±3.71 | 28.83±3.90 | 0.144 |
| **Medical History** | | | |
| Smoking | 3 (13.6%) | 12 (30%) | 0.150 |
| DM | 3 (13.6%) | 17 (42.5) | 0.02* |
| HTN | 3 (13.6%) | 14 (35%) | 0.071 |
| CHF | 6 (27.3%) | 15 (37.5) | 0.576 |
| **Source of sepsis** | | | |
| Perforated appendix | 5 (22.7%) | 7 (17.5%) | 0.618 |
| Ruptured viscous | 4 (18.2%) | 11 (27.5%) | 0.641 |
| Mesenteric vascular occlusion | 3 (13.6%) | 4 (10%) | 0.618 |
| Intestinal obstruction | 5 (22.7%) | 7 (17.5%) | 0.666 |
| Strangulated hernia | 4 (18.2%) | 8 (20%) | 0.862 |
| Other causes | 1 (4.5%) | 3 (7.5%) | 0.650 |
| **GCS** | 13.2±0.6 | 8.76±1.23 | <0.001* |
| **Pulse (b/min)** | 107.05± 19.77 | 111.60± 10.53 | 0.240 |
| **MAP (mm/hg)** | 96.15± 11.6 | 95.39± 7.48 | 0.752 |
| **RR (Br/min)** | 25.77± 11.04 | 32.68± 4.54 | 0.001* |
| **Temperature (°C)** | 37.51± 2.5 | 38.15± 1.25 | 0.584 |

Quantitative data are presented as mean ± SD, Qualitative data are presented as number and percentage

*: Statistically significant at $p \leq 0.05$

**Table (2):** Patients who did not survive showed considerably higher WBC levels. Additionally, they exhibited greater serum lactate levels, with a difference that was statistically significant. Regarding the blood culture results, there were no discernible changes between the two groups. The CRP level was significantly lower in the survivors than in the patients who passed away. Additionally, their bilirubin and creatinine levels were significantly lower. Patients who did not survive had considerably higher levels of BUN and urea and regarding survivors PF ratio increased significantly.

**Table (2):** Laboratory data of the included patients.

| Variables | Survived (N=22) | Died (N=40) | P-Value |
|---|---|---|---|
| **WBC (mcL)** | 12.27±2.58 | 23.23±10.91 | #p<0.001* |
| **S. lactate (mmol/L)** | 4.85±0.55 | 6.59±1.04 | #p=0.005* |
| **Blood culture** | | | |
| E. Coli | 9 (40.9%) | 11 (27.5) | 0.279 |
| Klebsiella | 5 (22.7) | 12 (30%) | 0.539 |
| Pseudomonas | 3 (13.6) | 10 (25%) | 0.293 |
| Staph Aures | 3 (13.6) | 7 (17.5%) | 0.692 |
| Gram positive streptococcal | 2 (9.1) | 0 | 0.052 |
| **CRP (mg/L)** | 48.0±3.5 | 120.0±6.5 | <0.001* |
| **Creatinine (mg/dL)** | 1.01±0.23 | 3.5±0.86 | 0.001* |
| **AST (IU/L)** | 90.90±21.43 | 99.27±23.81 | 0.176 |
| **ALT (IU/L)** | 113.91±27.62 | 75.93±17.52 | <0.001* |
| **Urea (mg/dL)** | 46.86±11.21 | 129.68±17.36 | 0.001* |
| **Bilirubin (µmol/L)** | 1.6±0.39 | 9.69±2.3 | <0.001* |
| **Platelet (mcL)** | 189.48±46.51 | 183.78±43.23 | 0.821 |
| **BUN (UREA/2.14)** | 21.89±5.32 | 60.59±8.26 | <0.001* |
| **PF ratio** | 131.89±31.12 | 78.69±12.11 | <0.001* |
| **Respiratory support** | | | |
| Nasal prongs | 7 (31.8%) | 10 (25%) | 0.565 |
| CPAP | 5 (12.5%) | 10 (25%) | 0.841 |
| Mechanical ventilation | 2 (9.1%) | 18 (45%) | 0.004* |
| None | 8 (36.4%) | 2 (5%) | 0.001* |

CPAP: Continues positive airway pressure, Quantitative data are presented as mean ± SD, Qualitative data are presented as number and percentage

*: Statistically significant at $p \leq 0.05$

**Table (3)** shows that mechanically ventilated patients were more prone to death but no statistically significant discrepancy was found between the two groups. Additionally, patients receiving room air had a statistically significant result regarding their likelihood of survival. The APACHE, SOFA, and qSOFA scores of the survivors were considerably lower than those of the deceased patients. A statistically significant difference in risk of death between patients with $ScvO_2$ levels above and below normal. A significantly greater survival rate was found by patients with normal $ScvO_2$ levels. Initially, there was non-statistically significant difference between the 2 groups in terms of $PvaCO_2$. Nevertheless, there was a statistically significant discrepancy in the $PvaCO_2$ between the patients who died and the survivors after 6 hours and up to 48 hours.

**Table (3):** APACHE, SOFA and qSOFA, $PvaCO_2$ and $ScvO_2$ among the included patients:

| Variables | Survived (N=22) | Died (N=40) | P-Value |
|---|---|---|---|
| **APACHE** | 22.64±3.20 | 27.05±3.53 | <0.001* |
| **SOFA** | 8.45±2.36 | 10.08±2.04 | 0.006* |
| **qSOFA** | 2.09±0.61 | 2.80±0.41 | <0.001* |
| **$ScvO_2$** | | | |
| Below normal | 2 (10%) | 10 (25%) | 0.129 |
| Normal | 16 (80%) | 2 (5%) | <0.001* |
| Above normal | 2 (10%) | 28 (70%) | <0.001* |
| **$PvaCO_2$** At admission | | | |
| 6 Hours | 8.5±1.1 | 8. 89±0.9 | 0.139 |
| 12 Hours | 7. 68±1.2 | 8. 50±0.6 | <0.001* |
| 18 Hours | 6. 68±0.9 | 8. 90±2.0 | <0.001* |
| 24 Hours | 6. 14±1.0 | 8. 95±1.6 | <0.001* |
| 30 Hours | 5. 82±0.7 | 9. 05±1.6 | <0.001* |
| 36 Hours | 5. 50±1.1 | 9. 55±1.3 | <0.001* |
| 42 Hours | 5.36±1.0 | 9. 59±1.3 | <0.001* |
| 48 Hours | 5. 45±1.1 | 10. 53±1.2 | <0.001* |
| | 5. 23±0.9 | 10. 88±0.9 | <0.001* |

Quantitative data are presented as mean ± SD
Qualitative data are presented as number and percentage.
*: Statistically significant at $p \leq 0.05$

**Table (4):** When investigating the performance of serum lactate in predicting death, we found that it has a good diagnostic performance with an AUC of 0.736 at cutoff of 5.7. $ScvO_2$ also had a great performance in predicting death with a cutoff of 81 as it had an AUC of 0.845 among the hyperoxic patients; and an AUC of 0.789 among the hypoxic patients with a cutoff of 69.5. However, the $PvaCO_2$ cutoff of 6.95 had only a moderate performance in death prediction with an AUC of 0.557 at T0; and AUC of 0.752 at T6 at a cutoff of 6.55 (fig 3-6).

**Table (4):** Validity of serum lactate, $PvaCO_2$ and $ScvO_2$ in detecting prognostic value in mortality

| Predictors | AUC | P-value | Cutoff | Sensitivity | Specificity |
|---|---|---|---|---|---|
| **Serum lactate (mmol/L)** | 0.736 (0.60-0.872) | 0.002* | 5.7 | 80.0 | 59.1 |
| **$ScvO_2$ in hyperoxic cases** | 0.845 (0.693-0.997) | 0.052 | 81.0 | 75.9 | 66.7 |
| **$ScvO_2$ in hypoxic cases** | 0.789 (0.573-1.01) | 0.340 | 69.5 | 75 | 49.0 |
| **Pa $CO_2$ immediate** | 0.557 (0.368-0.745) | 0.462 | 6.95 | 62.5 | 54.5 |
| **Pa $CO_2$ 6h** | 0.752 (0.624-0.881) | 0.001* | 6.55 | 57.5 | 77.3 |

AUC: area under the curve
*: Statistically significant at $p \leq 0.05$

**Table (5)** shows that $PvaCO_2$, APACHE-II, SOFA score, and qSOFA score were independent predictors of mortality (multivariate logistic regression analysis).

**Table (5):** Multivariate analysis of predictors of mortality.

| Predictors | β | p value | odds ratio (95%CI) |
|---|---|---|---|
| **Age/years** | 0.031 | 0.313 | 1.03 (0.971-1.09) |
| **Sex**<br>Male<br>Female | 0.223 | 0.677 | 1.25 (0.437-3.58) |
| **DM** | -0.143 | 0.80 | 0.867 (0.286-2.62) |
| **Hypertension** | 0.201 | 0.706 | 1.22(0.431-3.47) |
| **Smoking** | 0.969 | 0.08 | 2.64(0.890-7.81) |
| **$PvaCO_2$ at admission** | -0.690 | 0.013* | 0.502(0.290-0.867) |
| **$ScvO_2$ at admission** | -2.33 | 0.996 | 0.097 (undefined) |
| **APACHE-II** | 1.54 | 0.016* | 4.65(1.34-16.19) |
| **SOFA score** | 1.84 | 0.02* | 6.27(1.33-29.49) |
| **Q SOFA score** | 0.538 | 0.001* | 1.71(1.25-2.33) |
| **Overall % Predicted=80.5%** | | | |

*: Statistically significant at $p \leq 0.05$

## DISCUSSION

A potentially fatal illness with a high morbidity and death rate is severe intraabdominal sepsis. Precise prognostic indicators are essential for managing risks, forecasting results, and classifying risks. The VA carbon dioxide gradient ($PvaCO_2$), central venous oxygen saturation ($ScvO_2$), and serum lactate are three measures that have drawn interest recently [6].

Sixty-two patients with intraabdominal sepsis were included in this study. The following are our study's primary findings: (1) In patients experiencing septic shock, a high $PvaCO_2$ was linked to an increased risk of death in the ICU and served as a separate predictor of death. (2) Low or high $ScvO_2$ was more common in non-survivors, but normal $ScvO_2$ was linked to better results. (3) The survivors had noticeably decreased serum lactate levels.

In this investigation, the plasma lactate and creatinine levels in the survivors were lower than in the non-survivors (1.01±0.36 μmol/L vs 3.5±0.88 μmol/L, P = 0.001 and 4.85±2.55 vs 6.59 ± 2.04 mmol/L, P = 0.005, respectively). This is consistent with research by **Kumar *et al.*** [7] that found that non-survivors had a mean blood lactate level that was considerably higher at baseline.

In addition, serum lactate levels were greater in non-survivors in **Mohamed *et al.*** [8] study than in the survival group. Contrary to our findings, they stated that there was no correlation between serum creatinine levels and mortality. Additionally, they stated that there was no statistically significant difference in the mean serum bilirubin value between the survivor and fatality groups. This also contradicts our findings, since there was a statistically significant difference in bilirubin levels between the both groups (p-value<0.001).

The equilibrium between $CO_2$ generation and removal is shown in the $PvaCO_2$. Tissue hypoperfusion and compromised oxygen delivery in the context of severe sepsis lead to anaerobic metabolism and elevated $CO_2$ generation. $PvaCO_2$, which gives a measure of tissue oxygenation, rises in these individuals as a result, making it a useful marker for proper microcirculation and a favorable prognostic sign in septic shock [9].

Research has indicated that in cases of severe sepsis, a high $PvaCO_2$ is linked to unfavorable outcomes. Elevated $PvaCO_2$ levels signify insufficient tissue oxygenation, potentially resulting in organ failure and death [10].

In the current investigation, we discovered that, with regard to $PvaCO_2$ gradient in the first 6 to 48 hours following admission, there was a highly statistically significant difference between patients who survived and patients who died. $PvaCO_2$ gradient values were significantly higher in patients who passed away. Remarkably, $PvaCO_2$ did not demonstrate predictive power at admission, but it was a significant predictor of mortality in the examined individuals only at 6 hours. The $PvaCO_2$ gradient produced a sensitivity of 62.5 percent and a specificity of 54.5 percent at an admission cutoff value of ≥ 6.95. It produced results with a sensitivity of 57.5 percent and specificity of 77.3 percent at a cutoff value of ≥ 6.55 at hour 6.

According to our research, **EL-Said *et al.*** [1] conducted a prospective study to examine the changes in the Pcv-$aCO_2$ gap and the $PcvaCO_2$/Ca-$cvO_2$ ratio (Pcv-$aCO_2$/Ca-$cvO_2$ ratio) during early resuscitation in sepsis and septic shock as a predictor for the development of multi-organ dysfunction and mortality. They discovered that patients who had a consistently high Pcv-$aCO_2$ gradient at T6 [8.64 ± 1.66] experienced a greater death rate (61.1 percent) and more organ failure.

**Helmy *et al.*** [11] conducted a prospective study to examine the predictive significance of $PvaCO_2$ difference in patients undergoing early resuscitation from septic shock. Enrolled were forty patients who had been hospitalized to one intensive care unit. The findings demonstrated that a high $PCO_2$ gap >7.8 mmHg after six hours of septic shock patients' resuscitation is linked to a significant death rate.

Furthermore, **Yuan *et al.*** [3] investigation discovered that in septic shock patients with a high $ScvO_2$ (>eight percent), a lower $PvaCO_2$ (3.5 mmHg but not 6 mmHg) had a good ability to predict ICU death. In our study, the non-survivor group had a high lactate level and a low $PvaCO_2$ <six mmHg. Therefore, additional research is necessary to validate the normal cutoff value of $PvaCO_2$ in patients experiencing septic shock who have a high $ScvO_2$ (>80 percent) and have tissue hypoxia. Additionally, **Fernández *et al.*** [12] made a systematic review, comprising studies that assessed $PvaCO_2$ in adult patients with severe sepsis or septic shock. They showed that $PvaCO_2$ was correlated with mortality and other clinical outcomes in septic shock patients.

After six hours of completely goal-directed therapy (EGTD), adult patients with septic shock were the subjects of a prospective observational research by **Araujo *et al.*** [13] in an academic intensive care unit. In order to ascertain if $PvaCO_2$ determined from a superior central venous sample is a prognostic index (ICU length of stay, SOFA score, 28$^{th}$ mortality rate) immediately following EGDT, the study compared the intensive care unit admission values of patients with normal and abnormal (>6 mm Hg) $PvaCO_2$. $PvaCO_2$ is said to have no bearing on the dire outcome of sepsis patients. This disparity may arise from the inclusion, in the evaluation of a sizable percentage, of high to normal blood flow patients after early goal-directed therapy.

The equilibrium between oxygen delivery and consumption is reflected in $ScvO_2$. Reduced $ScvO_2$ levels can result from tissue hypoperfusion and poor oxygen extraction in cases of severe sepsis. Low $ScvO_2$ readings point to an imbalance in the supply and demand of oxygen, which may be a sign of insufficient resuscitation, tissue hypoxia, and a higher risk of death [14].

Numerous research works have examined the predictive significance of $ScvO_2$ in cases of severe sepsis. Results imply that poorer outcomes are linked to low $ScvO_2$ levels at admission or levels that remain low even after resuscitation. $ScvO_2$ has been suggested as a useful tool for directing resuscitation efforts and evaluating how well therapies work to maximize tissue oxygenation [15].

There was a highly statistically significant difference in $ScvO_2$ between patients who died and patients who survived in the current study. The $ScvO_2$ readings of patients who died were either significantly higher or lower. Notably, among the patients under study, $ScvO_2$ at admission did not significantly predict mortality. In order to assess the predictive significance of the static and dynamic factors of $ScvO_2$ in patients suffering from septic shock or severe sepsis, **Lee *et al.*** [16] conducted a prospective study. According to the area under the ROC curve, $ScvO_2$ was a significant discriminator in the twenty-eight-day mortality prediction. $ScvO_2$ ≥70% in multivariable analysis only marginally associated with 28-day mortality.

In this study, we found that at a cut off value of ≤ 69.5 percent at admission, the $ScvO_2$ yielded a sensitivity of 75% and Specificity of 49%.

A group of patients with hyperoxia is an interesting group. Elevated $ScvO_2$ may simply result from oxygen delivery exceeding tissue demand. More concerningly, though, is that it might be a sign of reduced oxygen consumption by cells as a result of mitochondrial or microcirculatory malfunction. If the higher oxygen delivery was the only factor contributing to hospital mortality [7, 17].

According to our research, non-survivors with above-normal $ScvO_2$ levels had a statistically significant higher prevalence of mortality than the survivors group. Similar to our results, **Kumar *et al.*** [7] discovered that survivors had a corresponding value of 93.89 ± 3.19% (p-value<0.001), while the non-survivor group had a considerably higher mean baseline $ScvO_2$ value of 95.15 ± 3.02.

## CONCLUSION

A higher risk of death and unfavourable results are linked to elevated $PvaCO_2$ and both above and below normal $ScvO_2$ levels. They therefore have potential as prognostic factors for patients with severe sepsis.

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