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The Use of End-tidal Carbondioxide Monitoring in the Prediction of 30-day Mortality in Patients Admitted to Emergency Department With Shock

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Abstract

Objectives

The primary objective of this study was to evaluate the utility of end-tidal carbon dioxide ($ETCO_2$) monitoring in predicting 30-day mortality in patients presenting to the emergency department with hypotensive shock. The secondary objective was to compare $ETCO_2$ values, traditional vital signs, and laboratory parameters between survivors and non-survivors.

Methods

In this prospective observational study, 58 adult patients presenting to the emergency department with shock were enrolled. Vital signs and $ETCO_2$ values were measured at 0 and 120 minutes after arrival. Arterial blood gas analysis, including pH, bicarbonate, base excess, and lactate levels, was performed at presentation. All patients received standard treatment according to current shock management protocols. Survival status was recorded at 30 days after hospital admission.

Results

$ETCO_2$ values at both 0 and 120 minutes were significantly lower in non-survivors than in survivors ($p < 0.001$). Non-survivors also had significantly lower pH, bicarbonate, and base excess values and higher lactate levels compared with survivors (all $p < 0.05$). In multivariate analysis, pH, lactate, and $ETCO_2$ at 0 minutes were independently associated with 30-day mortality (all $p < 0.05$), whereas bicarbonate and base excess were not ($p > 0.05$). $ETCO_2$ at 0 minutes demonstrated good discriminative ability for predicting mortality (AUC 0.863, $p < 0.001$). Two $ETCO_2$ cut-off values were identified for mortality prediction: 23.5 mmHg (sensitivity 74.1%, specificity 80.6%) and 24.5 mmHg (sensitivity 88.9%, specificity 71.0%).

Conclusions

$ETCO_2$ monitoring is a useful and easily obtainable tool for predicting 30-day mortality in patients presenting to the emergency department with shock and may aid in early risk stratification in this high-risk population.

1. INTRODUCTION

Shock is a condition caused by cellular and tissue hypoxia due to reduced oxygen transport, increased oxygen consumption or insufficient oxygen use. Shock is usually associated with hypotension which is a sign of circulatory failure. Hypotensive shock is the main finding of many diseases in patients admitted to the emergency department. Patients may present with hypovolemic, cardiogenic, anaphylactic,

neurogenic or septic shock. Despite the emerging shock treatments and managements, mortality remains high. Emergency physicians and scientists are constantly trying to find new ways to recognize shock and initiate early treatment.[1]

Traditionally, shock-onset therapy in the emergency department has focused on normalizing vital signs such as heart rate, mean arterial blood pressure (MAP), and central venous pressure to ensure adequate tissue oxygenation and perfusion. Heart rate and blood pressure findings are not reliable until severe hypotension develops.[2,3] Capnography is widely used in cases of cardiopulmonary resuscitation administered cardiac arrest. Cardiac arrest decreases cardiac output and this results in the carbon dioxide elimination by the lungs. The end tidal carbon dioxide (ETCO2) value shows an increase during successful resuscitation. Therefore, ETCO2 has been proposed and used as a prognostic tool.[3,4] ETCO2 values have also been shown to decrease in volume-related hypotensive conditions with decreased cardiac output.[3]

The primary aim of the study is to evaluate the use of end-tidal carbon dioxide monitoring in predicting 30-day mortality in patients admitted to the emergency department with hypotensive shock. The secondary aim of the study is to determine the relation between ETCO2 and traditional vital signs and laboratory findings, and to show the relation between early measured ETCO2 value and the prognosis of shock patients.

2. MATERIALS AND METHODS

This prospective observational study was performed in the Emergency Department of Gazi University Medical Faculty Hospital which is a tertiary health center. Patients who admitted to the emergency department with clinical signs of shock with systolic arterial blood pressure (SAP) < 90 mm/Hg or MAP < 60 mm/Hg between 1 December 2015 and 30 April 2016 are included in the study. Prior to the study, approval was obtained from the Clinical Research Ethics Committee of Kecioren Training and Research Hospital and (Decision No: 997, Date: 25.11.2015) Helsinki Declaration principles were followed. Necessary information was given to the patients and their relatives and written informed consent was obtained from them or their relatives.

2.1 Inclusion Criteria:

1. Patients over 18 years of age and willing to participate in the study

2. Patients presenting to the emergency department with clinical signs of shock

3. Patients with SAP < 90 mm / Hg or MAP < 60 mm/Hg

2.2 Exclusion Criteria:

1. Patients aged below 18 years

2. Patients who do not want to participate in the study

3. Patients with cardiopulmonary arrest on arrival

4. Patients whose blood pressure or ETCO2 cannot be measured upon arrival

5. Pregnant patients

The age, sex, contact information, medical history, medications, physical examination findings, laboratory results, diagnosis and treatment of the patients included in the study were recorded on the patient registration form. Possible shock type of the patient was recorded. ETCO2 measurement with vital signs including SAP, diastolic arterial blood pressure (DAP), MAP, heart rate, respiration rate, oxygen saturation (SpO2 and SaO2), fever, pulse at 0 and 120 minutes of the patients' admission, and blood gas values including lactate, power of hydrogen (pH), partial arterial oxygen pressure (PaO2), partial arterial carbon dioxide pressure (PaCO2), base excess (BE) and bicarbonate (HCO3) levels parameters on arrival were added to the study form. The final condition of the patients after 30 days (short-term survival on day 30 after admission) was obtained through hospital records and contact information of patients and their relatives and was added to the study form.

In this study, PETAS® KMA®900 series bed-side monitors were used. Non-invasive blood pressure, pulse, respiration rate, oxygen saturation and ETCO2 were measured with this device. ETCO2 was measured with the most appropriate method either with a sidestream with bed-side monitor (Petas KMA®900 bed-side monitor) using the disposable ETCO2 nasal cannula kit or intubated patient kit. Measurements were performed without interfering with the medical necessities and priorities, and routine follow-up was performed according to the procedure, diagnosis and treatment.

2.3 Statistical analysis

The data from the study was uploaded to the computer and evaluated by using “SPSS (Statistical Package for Social Sciences) for Windows 22.0 (SPSS Inc, Chicago, IL)” . The descriptive statistics were presented as mean ± standard deviation, median (25%-75%) and percentage. Yates Corrected Chi-Square Test was used to evaluate categorical variables. Suitability of variables to normal distribution was analysed with using visual (histogram and probability graphs) and analytical methods (Shapiro-Wilk Test). The Mann-Whitney U Test was used as the statistical method for statistical significance between the two independent groups for the variables that were not found to fit the normal distribution. Student’s T Test was used between two independent groups for the variables that were found to fit normal distribution. The relation between variables was evaluated by Spearman Correlation Analysis. In multivariate analysis, independent predictors of mortality condition were analyzed using Logistic Regression analysis using possible factors identified in previous analyzes. Hosmer-Lemeshow test was used for model goodness-of-fit. The diagnostic decision-making feature of ETCO2 value in predicting

mortality condition was analyzed by ROC curve analysis. The sensitivity, specificity, positive and negative predictive values of these cut-off values were calculated in the presence of significant cut-off values. Statistical significance level was accepted as $p<0.05$.

3. RESULTS

A total of 58 patients admitted to the emergency department with shock presentation were evaluated. The mean age of the patients was 70.16 ± 14.48 (min-max: 30-99) years, 53.4% (n = 31) of which were females and 46.6% (n = 27) were males. Seventy seven point six percent (n = 45) of the patients had at least one comorbid disease. Of the patients examined, 46.6% (n = 27) died within the first 30 days after admission, while the remaining 53.4% (n = 31) survived. No statistically significant difference was found between the exitus and survivor groups in terms of age (p=0.103), gender (p=0.623), and comorbidities (p=0.728).

While 48.3% (n = 28) of the patients examined were diagnosed with septic shock, 24.1% (n = 14) were hypovolemic, 12.1% (n = 7) were cardiogenic and 15%, Five (n = 9) were diagnosed as dual combinations.

When the recent condition of the patients inspected in the emergency department was analyzed; while 39.7% (n = 23) were admitted to the intensive care unit, 17.2% (n = 10) were admitted to any service, 15.5% (n = 9) were discharged from the emergency department and 6.9% (n = 4) was referred to another center and 15.5% (n = 9) died in the emergency department, while 5.2% (n = 3) left the emergency department voluntarily.

Of the 31 survivors, 77.4% (n = 24) were followed up at home after 30 days, while the remaining 22.6% (n = 7) were followed up in the ward or intensive care unit.

The ETCO2 values at minute 0 of the exitus patients were significantly lower compared to the survivors ($p <0.001$). No significant difference was observed between the groups in terms of the hemodynamic parameters ($p>0.05$) (Table 1). At minute 0, while pH (p=0.004), HCO3 (p=0.002) and BE (p=0.001) were observed to be significantly lower in the exitus group compared to the survivor group, blood lactate level was observed to be significantly higher ($p<0.001$) No significant difference was observed between the groups in terms of other arterial blood gas parameters at minute 0 ($p>0.05$) (Table 1).

The ETCO2 values at minute 120 of the exitus patients were significantly lower compared to the survivors ($p <0.001$) (Table 2). At the 120th minute, SAP (p=0,029), MAP (p=0.027) and body temperature (p=0.030) were significantly higher in the survivor group compared to the exitus group, while no statistically significant difference was observed between the groups in terms of DAP, pulse, respiratory rate and SpO2 value ($p>0.05$) (Table 2).

While mean SAP (p=0.032), mean MAP (p=0.021) and mean ETCO2 ($p<0.001$) values were observed to be significantly higher in the survivor group compared to the exitus group, no statistically significant

difference was observed between the groups in terms of mean DAP, mean pulse rate, mean respiratory rate, mean SpO2 value and mean body temperature (p> 0.05) (Table 3).

In the exitus group, a moderate positive correlation (r: 0.525 p: 0.005) was observed between the ETCO2 value at minute 0 and PaCO2. The ETCO2 value at minute 120 was observed to be moderately positively correlated with PaCO2 (r: 0.553 p: 0.003), moderately negatively correlated with Lactacte value (r: -0.412 p: 0.033), moderately positively correlated with BE (r: 0.408 p: 0.035), and low to moderately positively correlated with HCO3 (r: 0.391 p: 0.044). The mean ETCO2 value was observed to be moderately positively correlated with PaCO2 (r: 0.539 p: 0.004).

In the survivor group, the ETCO2 value at minute 0 was observed to be moderately positively correlated with PaCO2 (r: 0.457 p: 0.010), moderately negatively correlated with Lactacte value (r: -0.423 p: 0.018), and low to moderately negatively correlated with pH (r: -0.397 p: 0.027). The ETCO2 value at minute 120 was observed to be low to moderately negatively correlated with Lactacte value (r: -0.372 p: 0.039). The mean ETCO2 value was observed to be moderately positively correlated with PaCO2 (r: 0.446 p: 0.012), and moderately negatively correlated with Lactacte value (r: -0.423 p: 0.018).

Multivariate logistic regression analysis was used to evaluate the independent effects of pH, lactate, bicarbonate, base excess and ETCO2 variables at 0 minutes on the mortality condition, which were determined to be significantly different between the mortality condition as a result of the previous univariate analyzes. When other variables were checked; it was observed that pH (p:0.018), lactate (p:0.037) and ETCO2 (p:0.002) variables at 0 minutes had a significant effect on mortality, and bicarbonate and base excess were absent (p>0.05). The increase in lactate values and the decrease in pH increased the risk of exitus while the increase in ETCO2 value at 0 minutes decreased the risk of exitus (Table 4).

In addition, the diagnostic decision-making feature of 0 minutes $ETCO_2$ value in predicting the mortality condition of the patients was evaluated by ROC curve analysis. At minute 0 $ETCO_2$ value was determined to be diagnostic decision-making feature for predicting mortality condition (AUC: 0.863, p<0.001) and two different cut-off values were determined. Sensitivity and specificity of $ETCO_2$ cut-off value of 23.5 mmHg at 0 minutes was 74.1% and 80.6%, positive predictive value (PPV) was 76.9% and negative predictive value (NPV) was 78.1%. The cut-off value of 24.5 was 88.9%, and 71.0%, PPV was 72.7% and NPV was 88.0% (Figure 1).

4. DISCUSSION

In this study which we aimed to evaluate the use of end-tidal carbon dioxide (ETCO2) measurement in predicting 30-day mortality in patients presenting to the emergency department with hypotensive shock, there was a statistically significant difference detected between the exitus and surviving patients when ETCO2 at 0 minutes, ETCO2 at 120 minutes and mean ETCO2 values were compared (p < 0.001). ETCO2 values of the exitus patients were significantly lower than those of survivors. As a result of multivariate

logistic regression analysis to evaluate the independent effect of ETCO2 on mortality condition and when other variables were taken under control, ETCO2 was found to have a significant effect on mortality condition ($p < 0.05$). These findings are consistent with recent literature, including a systematic review and meta-analysis evaluating trauma patients with hemorrhagic shock, which demonstrated that $ETCO_2$ values below approximately 25 mmHg are strongly predictive of mortality and transfusion requirements.[5] Similarly, contemporary prospective trauma studies have confirmed the prognostic value of early $ETCO_2$ monitoring for identifying high-risk patients requiring aggressive resuscitation.[6]

Kheng et al. demonstrated in a study performed in hypotensive patients in the emergency department that ETCO2 can be used to predict mortality in shock. The mean age of the patients included in this study was 54, 52% of the patients were males and 48% were females.[3] Similarly, 46.6% of the patients were males and 53.4% were females in our study. On the other hand, the mean age of the patients was 70.1 in our study. This difference may be due to the fact that the majority of the admissions to our hospital were from the geriatric patient population.

Recent evidence supports ETCO2 as a noninvasive marker of perfusion and prognostication across shock phenotypes. In emergency department sepsis cohorts, lower ETCO2 correlates with higher in-hospital mortality and metabolic derangements, underscoring its role in early risk stratification.[7–9] In trauma, prehospital ETCO2 is depressed in severe injury and predicts death and transfusion needs, extending its utility beyond cardiac arrest.[10] Review data from emergency medicine synthesize capnography's physiologic rationale—tracking ventilation–perfusion and cardiac output changes in real time—which underpins its prognostic signal in shock.[11] Earlier clinical work in hypovolemic and cardiogenic shock also showed lower ETCO2 with worse outcomes and improvement with circulatory support, supporting physiologic plausibility.[12,13] Foundational cardiac arrest literature similarly linked ETCO2 with ROSC and survival, establishing its prognostic relevance in low-flow states.[14]

In our study, 0-minute $ETCO_2$ showed strong discriminative ability for mortality (AUC 0.863; $p < 0.001$). Two actionable cutoffs were identified: 23.5 mmHg (sensitivity 74.1%, specificity 80.6%; PPV 76.9%, NPV 78.1%) and 24.5 mmHg (sensitivity 88.9%, specificity 71.0%; PPV 72.7%, NPV 88.0%). In normotensive trauma patients, the lowest prehospital $ETCO_2$ predicted mortality with an optimal cutoff near 31 mmHg and showed strong negative predictive value.[15] Consistently, prehospital $ETCO_2$ around 30 mmHg identified high-risk patients with high NPV for death, supporting clinically actionable thresholds.[10] $ETCO_2$ also correlates with transfusion needs; a prehospital cutoff of 26 mmHg predicted massive transfusion.[16] Beyond trauma, higher $ETCO_2$ during out-of-hospital resuscitation is associated with greater likelihood of ROSC, underscoring its prognostic utility in low-flow states.[17] Earlier work aligns with these findings, linking low $ETCO_2$ with nonsurvival during emergent trauma surgery and improved outcomes at higher levels, and reporting mean $ETCO_2$ around 25 mmHg among ROSC achievers.[18,19] Overall, convergent prehospital and perioperative evidence suggests clinically relevant thresholds near 26–31 mmHg for risk stratification.

In our study, no statistically significant difference was detected between the exitus and surviving patients in terms of pulse rate at 0 min ($p > 0.05$). Although tachycardia is a sign of acute blood loss, heart rate may mislead the clinician by providing variable responses to internal and external stimuli. Additionally, there was no baseline value for comparison of heart rate before blood loss in patients who were brought to the emergency department with hemorrhagic shock presentation.

In trauma, tachycardia is an unreliable screening sign: a substantial proportion of hypotensive patients lack tachycardia, and many normotensive patients are tachycardic.[20] By contrast, prehospital $ETCO_2$ outperforms systolic blood pressure and shock index for mortality prediction, particularly when HR and BP are equivocal.[15] Reflecting perfusion and metabolic status better than heart rate alone, $ETCO_2$ has been proposed as a "sixth vital sign"[7], and early respiratory metrics can improve identification of serious injury and guide triage beyond traditional vitals.[21]

Serum lactate is a well-established marker of tissue hypoxia and predicts organ failure and mortality across shock states, including septic and cardiogenic shock; serial measurements enhance prognostication.[22–24] In emergency department cohorts, $ETCO_2$ correlates with metabolic disturbances such as lactic acidosis and predicts in-hospital mortality.[7] In penetrating trauma, nasal cannula $ETCO_2$ correlates with serum lactate and the likelihood of operative intervention, indicating complementary use with lactate.[25] Prehospital data further support $ETCO_2$ as continuous, actionable feedback on perfusion that can be integrated with lactate for early risk stratification, with clinically useful thresholds in trauma.[15] In our study, the lactate values at admission were analyzed, but serial lactate monitoring was not performed. Similar to the mentioned studies, in our study, lactate levels were found to be significantly higher in patients who died ($p < 0.05$). As a result of multivariate logistic regression analysis to evaluate the independent effect of lactate on mortality condition and when other variables were under control, lactate variable had a significant effect on mortality condition ($p < 0.05$).

Base deficit is an indirect marker of hypoperfusion and tissue acidosis, and higher initial deficits reflect greater shock severity.[26] Classic trauma studies linked base deficit with transfusion needs and outcomes[24,27] and showed higher mortality with base excess ≤ − 6 in blunt trauma.[28] Contemporary ED data demonstrate that $ETCO_2$ correlates with base excess, stratifies hemorrhagic shock severity (e.g., $ETCO_2$ < 30 mmHg sensitive for stage 2–3; < 22 mmHg specific for stage 4), and a base excess ≤ − 10 mEq/L predicts mortality with high specificity.[6] In non-traumatic circulatory shock, admission $ETCO_2$ positively correlates with base deficit at 0 and 120 minutes and independently predicts in-hospital mortality (AUROC 0.735; cutoff ≤ 23 mmHg).[29] Among mechanically ventilated ICU patients, a widened $Pa–ETCO_2$ gradient—reflecting V/Q mismatch and impaired perfusion—independently associates with higher overall and 28-day mortality and worse acid–base status, underscoring CO_2-derived indices as severity markers.[30] In our study, base excess was significantly lower in non-survivors ($p < 0.05$) and similar to other studies, the mean of the base excess of the exitus was determined to be -9.3 while the mean of the survivors was − 0.2.

This study offers several notable strengths that enhance its contribution to the existing literature. Unlike many retrospective analyses, our prospective observational design allowed for real-time data collection of $ETCO_2$ values at critical time points (0 and 120 minutes), providing a more accurate reflection of dynamic physiological changes in hypotensive shock. Furthermore, by focusing specifically on 30-day mortality as a primary outcome, we provide a clinically relevant and robust measure of long-term prognosis, which is often a limitation in studies with shorter follow-up periods. The comprehensive comparison of $ETCO_2$ with traditional vital signs and a broad panel of laboratory markers (pH, lactate, bicarbonate, base excess) in a multivariate analysis strengthens the evidence for $ETCO_2$ as an independent predictor, highlighting its unique value beyond conventional parameters. Lastly, the determination of specific $ETCO_2$ cut-off values (23.5 mmHg and 24.5 mmHg) with high sensitivity and specificity offers practical, actionable insights for emergency physicians, facilitating earlier risk stratification and potentially guiding more timely interventions in a critically ill patient population.

In conclusion, ETCO2 monitoring is a useful method for predicting mortality in patients with shock. New studies with larger patient groups are needed in this respect. Future research should define standardized thresholds and integration strategies with existing triage tools in large, prospective cohorts.

5. LIMITATIONS

There are some limitations in our study. Power analysis was not performed for sample size estimation before the study. As the number of patients is small, the generalizability of the results is low. Only patients with hypotension were included in the study. However, since hypotension had not yet developed in the early stages of shock, such early-stage shock patients may have been overlooked and may not be included in the study. Another limitation was the monitoring of $ETCO_2$ by two separate methods, these are nasal cannula kit and intubated patient kit. There may be slight measurement differences between the two methods. Heterogeneity in capnography methods and patient selection is a known limitation in the literature, underscoring the need for standardized acquisition and reporting.[31]

6. CONCLUSION

In conclusion, ETCO2 monitoring is a useful method for predicting mortality in shock patients. $ETCO_2$ is considered a reliable, rapid, and noninvasive prognostic tool across different types of shock and should be considered for integration into emergency triage protocols.

Declarations

Author contributions:

OG, AK, AD, FB, UG, and HAD contributed to the study concept and design.
OG, AK, UG, and HAD contributed to data acquisition.
OG and AK contributed to data analysis and interpretation.

OG drafted the manuscript.
AK, AD, FB, UG, and HAD critically revised the manuscript for important intellectual content.
OG had primary responsibility for the final content.

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Conflict of interest:

OG reports no conflict of interest.

AK reports no conflict of interest.

AD reports no conflict of interest.

FB reports no conflict of interest.

UG reports no conflict of interest.

HAD reports no conflict of interest.

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Tables

Table 1. Comparison results between exitus and survivor groups in terms of ETCO2 values, hemodynamic parameters and arterial blood gas parameters at 0th minute

0 minutes	Mortality Condition		p
	Exitus (n = 27)	Survivor (n=31)	
	mean±SD / median (25%-75%)		
$ETCO_2$ (mmHg)	17.93±7.18	28.97±7.23	**<0.001***
SAP (mmHg)	75 (70-80)	80 (70-80)	0.381**
DAP (mmHg)	40 (40-50)	45 (40-50)	0.137**
MAP (mmHg)	53.80±7.07	56.47±6.63	0.143*
Pulse (/min)	101±25	101±27	0.974*
Respiratory Rate (min)	24 (20-28)	22 (20-24)	0.358**
SpO_2 (%)	94 (90-96)	96 (93-98)	0.055**
Tempreature (ºC)	36.6 (36.4-36.7)	36.7 (36.3-36.9)	0.621**
pH	7.32±0.13	7.41±0.11	**0.004***
$PaCO_2$ (mmHg)	35 (25-39)	34 (28-39)	0.827**
PaO_2 (mmHg)	73 (64-97)	68 (59.9-95)	0.464**
SaO_2 (%)	94 (92-97)	94.8 (91-96)	0.673**
Lactate (mmol/L)	3.0 (1.8-5.1)	1.4 (1.0-1.9)	**<0.001****
HCO_3 (mmol/L)	17.0 (15.3-22.3)	22.3 (19.3-24.9)	**0.002****
BE (mmol/L)	-9.3 (-11.6; -1.4)	-0.2 (-5.4; 0.9)	**0.001****

.* Student's T Test;

** Mann-Whitney U Test

Statistical significance level: p<0.05

Abbreviations ETCO2: End tidal carbon dioxide, SAP: systolic arterial blood pressure, DAP: diastolic arterial blood pressure, MAP: mean arterial blood pressure, SpO2: Oxygen saturation with measured a pulse oximeter**,** pH: Power of Hydrogen, PaCO2: Partial Arterial Carbon Dioxide Pressure, PaO2: Partial Arterial Oxygen Pressure, SaO2: Oxygen saturation in arterial blood, HCO3: bicarbonate , BE: base excess

Table 2. Comparison results between exitus and survivor groups in terms of ETCO2 values, hemodynamic parameters and arterial blood gas parameters at 120th minute

120 minutes	**Mortality Condition**		**p**
	Exitus (n = 27)	**Survivor (n=31)**	
	mean±SD / median (25%-75%)		
ETCO2 (mmHg)	16.81±6.15	28.97±6.94	**<0.001***
SAP (mmHg)	80 (75-90)	90 (80-95)	**0.029****
DAP (mmHg)	50 (40-50)	50 (45-60)	0.091**
MAP (mmHg)	58.89±7.10	63.39±7.91	**0.027****
Pulse (/min)	97±25.78	99±24	0.764*
Respiratory Rate (min)	24 (14-26)	22 (20-24)	0.236**
SpO_2 (%)	95 (92-96)	95 (93-96)	0.523**
Tempreature (^oC)	36.4 (36.1-36.7)	36.6 (36.4-36.8)	**0.030****

* Student's T Test;

** Mann-Whitney U Test,

Statistical significance level: p<0.05

Abbreviations ETCO2: End tidal carbon dioxide, SAP: systolic arterial blood pressure, DAP: diastolic arterial blood pressure, MAP: mean arterial blood pressure, SpO2: Oxygen saturation with measured a pulse oximeter.

Table 3. Comparison results of the mean values of ETCO2 values, hemodynamic parameters and arterial blood gas parameters (at 0 and 120 minutes) between the exitus and survivor groups

Mean	Mortality Condition		p
	Exitus (n = 27)	**Survivor (n=31)**	
	mean±SD / median (25%-75%)		
$ETCO_2$ (mmHg)	17.37±6.57	28.97±6.65	**<0.001***
SAP (mmHg)	77.5 (72.5-85.0)	85.0 (77.5-87.5)	**0.032****
DAP (mmHg)	45 (40-50)	47.5 (45-55)	0.068**
MAP (mmHg)	56.35±5.13	59.93±6.22	**0.021***
Pulse (/min)	99±24	100±25	0.892*
Respiratory Rate (min)	24 (19-26)	22 (20-23)	0.377**
SpO_2 (%)	94.5 (90.5-95.5)	95.0 (93.5-97.0)	0.111**
Tempreature (^oC)	36.5 (36.3-36.6)	36.6 (36.4-36.8)	0.189**

* Student's T Test;

** Mann-Whitney U Test,

statistical significance level: p<0.05

Abbreviations ETCO2: End tidal carbon dioxide, SAP: systolic arterial blood pressure, DAP: diastolic arterial blood pressure, MAP: mean arterial blood pressure, SpO2: Oxygen saturation with measured a pulse oximeter.

Table 4. Multivariate Logistic Regression Analysis to Evaluate the Independent Effect of Some Predictors on Mortality Condition

	B	SH	Wald χ^2	sd	OR	95% GA	p
0 minutes pH	-24.690	10.407	5.628	1	0.001	0-0.014	**0.018**
0 minutes Laktat	1.060	0.508	4.344	1	2.885	1.065-7.814	**0.037**
0 minutes HCO_3	0.063	0.351	0.033	1	1.066	0.535-2.121	0.857
0 minutes BE	0.133	0.378	0.123	1	1.142	0.544-2.395	0.726
0minutes $ETCO_2$	-0.315	0.100	9.963	1	0.730	0.600-0.887	**0.002**

Dependent variable: Mortality condition

R^2 (Cox-Snell) = 0.56; R^2 (Nagelkerke) = 0.74; , statistical significance level: p<0.05

Abbreviations ETCO2: End tidal carbon dioxide**,** pH: Power of Hydrogen, HCO3: bicarbonate , BE: base excess.

Figures

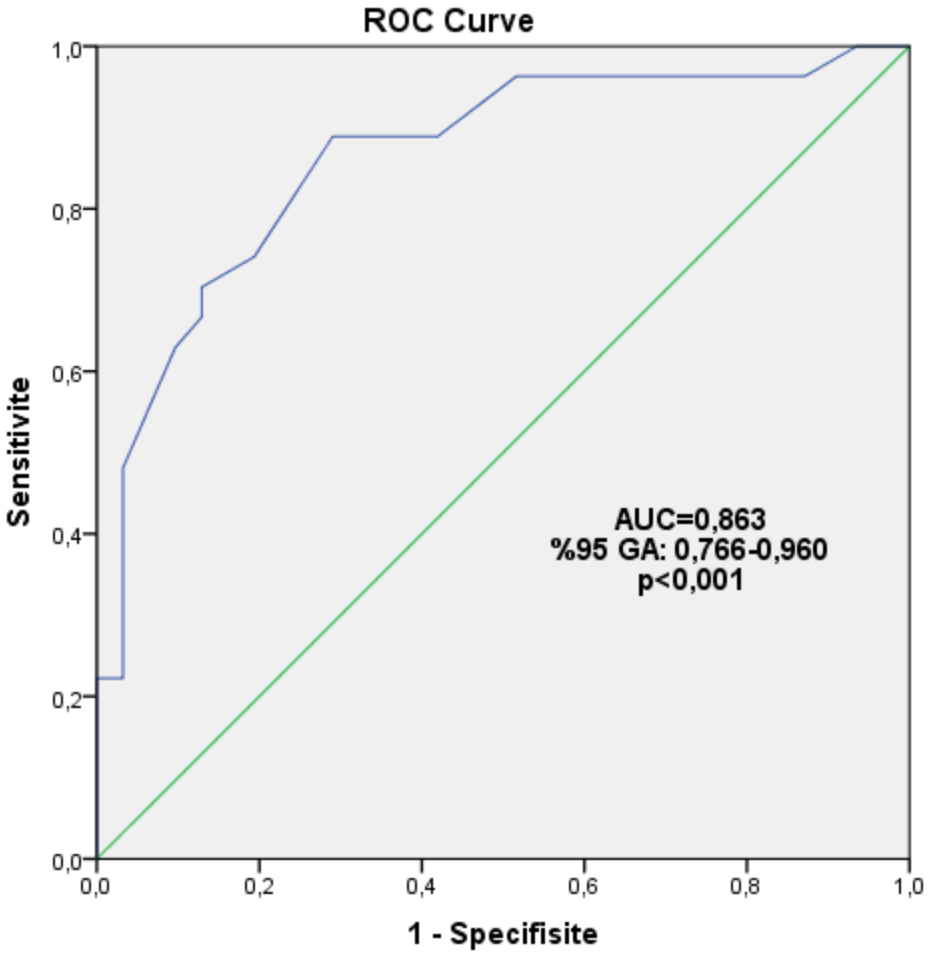


Figure 1

In Predicting Mortality Condition ROC Curve for Diagnostic Decision Making of 0 minutes $ETCO_2$ Value