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Outcome of Cardiac Arrest and Non-Cardiac Arrest Patients with Severe Acidosis in the Emergency Department: A Retrospective Cohort Study

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Abstract

Aim: We aimed to evaluate the outcome of cardiac arrest and non-cardiac arrest patients with severe acidosis admitted to the emergency department (ED) and to analyze the relationship between in-hospital mortality and clinical factors.

Materials and Methods: Patients with severe acidosis (pH <7.1) presenting to the ED were included in the study. Patients were divided according to arrest status and outcomes and analyzed accordingly.

Results: The study included 540 patients with severe acidosis. The 30-day mortality rate was 74.8% in all patients. In the non-cardiac arrest subgroup, the 30-day mortality rate was 21.4%. Non-arrest and non-survivors were more likely to be older, male, and have a higher prevalence of hypertension and coronary artery disease. Mortality was significantly higher among patients with sepsis, metabolic causes, and isolated respiratory arrest, while it was lower, in those with neurological etiologies, diabetic ketoacidosis, and seizures ($p<0.05$). Although no significant differences were observed in blood gas parameters, non-survivors had significantly higher lactate and creatinine levels lower hemoglobin levels ($p<0.05$). Univariate analysis identified advanced age, male sex, sepsis, metabolic causes, isolated respiratory arrest, hypertension, coronary artery disease, elevated partial carbon dioxide pressure (pCO_2) and creatinine, and reduced hemoglobin as significant predictors of 30-day mortality ($p<0.05$ for all).

Conclusion: Severe acidosis is associated with high 30-day mortality, particularly in cardiac arrest patients. However, non-arrest patients also exhibit considerable mortality. Advanced age, male sex, cardiovascular comorbidities, sepsis, and elevated lactate, pCO_2, and creatinine levels were identified as key predictors. Early recognition and management of these factors may improve outcomes.

Keywords: Severe acidocis, emergency, mortality

Introduction

The maintenance of blood pH within a narrow physiological range (7.35-7.45) is essential for the optimal activity of intracellular enzymes and the preservation of cellular membrane integrity (1). This homeostasis is regulated by buffer systems in conjunction with respiratory and renal mechanisms, which collectively prevent significant deviations in pH and thereby support the normal function of all cellular and organ systems (2).

Acid-base disorders are common among critically ill patients admitted to the emergency department (ED) and are observed in 97.3% of critical care areas such as the resuscitation area (3). Severe acidosis is a life-threatening emergency that is often associated with poor outcomes. Severe metabolic acidosis is typically defined as a pH level below 7.1. This condition may arise due to various etiologies, including lactate accumulation resulting from a shift to anaerobic cellular metabolism, acute or chronic renal insufficiency, systemic hypoperfusion, or hypoventilation (4).

The most significant issues linked to acidosis include hemodynamic instability, respiratory failure, renal and hepatic failure, severe infections, trauma, various metabolic disorders,

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and toxic ingestion. Consequently, acidemia has been identified as a poor prognostic factor (1). The presence of these conditions, along with the fact that acidemia is a symptom of a critical illness, explains why severe acidemia, generally (defined as a pH value below 6.8-7.0), is considered to be incompatible with life (5,6). However, there are rare reports of patients surviving even when the pH drops to as low as 6.7 (7-11). Determining the prognostic impact of severe acidemia could significantly influence critical patient care and decision-making in the initial hours of resuscitation, especially the mortality rate in this patient group is still unknown.

During cardiac arrest, tissue perfusion ceases entirely, leading to a shift toward anaerobic metabolism and the development of lactic acidosis. Following reperfusion, metabolic acidosis may further worsen. In patients with cardiac arrest, severe acidosis impairs myocardial contractility and diminishes the responsiveness to vasopressors, thereby negatively affecting resuscitation outcomes. Several studies have demonstrated that a pH level below 7.0 is associated with reduced rates of successful defibrillation and return of spontaneous circulation (12).

In non-cardiac arrest patients, severe acidosis is commonly observed in conditions such as diabetic ketoacidosis (DKA), acute and/or chronic renal failure (CRF), profound hypoxia, sepsis, and drug or alcohol intoxications (e.g., salicylates, metformin), as well as following epileptic seizures. In these cases, acidosis except in seizure-related presentations tends to develop more gradually and involves more complex pathophysiology. Although the underlying etiology may vary, a pH <7.2 in critically ill non-cardiac patients has been associated with increased mortality. However, early and targeted interventions in the ED, tailored to the specific cause, may improve patient outcomes (13). A review of the literature reveals that most existing studies have been conducted on cardiac arrest patients and within intensive care unit settings, primarily focusing on clinical and prognostic factors (5-13). This study primarily aimed to evaluate patients presenting to the ED with severe acidosis and to investigate the association between 30-day mortality and clinical factors, particularly in the subgroup of patients with non-cardiac arrest-related severe acidosis. A better understanding of the determinants of mortality in this patient population may contribute to the development of early diagnostic and therapeutic strategies in clinical practice. In this context, optimizing the clinical trajectory and treatment approaches for patients with severe acidosis represents a critical opportunity to improve patient management and outcomes.

Materials and Methods

This is a retrospective study. The Ethics Committee of University of Health Sciences Türkiye, Ankara Atatürk Sanatorium Training and Research Hospital approved the study protocol in accordance with (decision number: 2024-BÇEK/11, date: 14.02.2024) the ethical principles of the Declaration of Helsinki and current Good Clinical Practice guidelines. Since our study was retrospective, the requirement for informed consent was waived.

Between 01.01.2021 and 01.12.2023, patients aged ≥18 years who were admitted to the ED of the 780-bed University of Health Sciences Türkiye, Ankara Atatürk Sanatorium Training and Research Hospital (Ankara, Türkiye) whose blood gas analysis obtained within the first hour of ED admission showed a pH value below 7.1 at least once were included in the study. Patients were excluded if they had unreliable blood gas analysis results (e.g., markedly inconsistent values in tests performed immediately before or after), lacked blood gas sampling within the first hour of admission, or had missing key parameters, lactate levels, complete blood count, international normalized ratio (INR), or biochemical values.

Demographic data, comorbidities [hypertension (HT), diabetes mellitus (DM), coronary artery disease (CAD), chronic obstructive pulmonary disease, CRF, cerebrovascular disease], laboratory results (including biochemical values, venous blood gas analysis, etc.) and hospital outcomes were obtained through a retrospective review of patient files.

Patients were classified into the following categories based on the reason for admission: cardiac arrest, bleeding/trauma, intoxication, sepsis, metabolic, neurological, and unknown/other. Patients admitted with DKA and epileptic seizures were classified separately. Patients with multiple diagnoses were noted accordingly in the tables. Among non-arrest patients, those who developed severe acidosis due to DKA or epileptic seizures were evaluated separately.

Patients who were documented in the hospital records as having cardiac arrest and received cardiopulmonary resuscitation (CPR) were classified under the cardiac arrest group. Patients who did not undergo CPR and diagnosed with respiratory arrest in the system records were classified as having isolated respiratory arrest. Initially, a cohort was established comprising all patients with severe acidosis. Subsequently, a distinct subgroup of patients with severe acidosis but without cardiac arrest was identified. Statistical analyses were conducted to evaluate the factors associated with 30-day mortality in both groups.

Statistical Analysis

All data obtained throughout the study and recorded on the study form were analyzed using the IBM SPSS 20.0 statistical program (Chicago, IL, USA). The Kolmogorov-Smirnov test was used to determine whether the distribution of discrete and

continuous numerical variables followed a normal distribution. Continuous numerical variables were expressed as median interquartile range (IQR: 25-75), while categorical variables were expressed as the number of cases and percentages. Categorical variables were analyzed using the chi-square test, and continuous variables were analyzed using the Mann-Whitney U test. To identify the risk factors predicting mortality in patients with non-arrest severe acidosis, univariate regression analysis was performed. Results were considered statistically significant when $p<0.05$.

Results

A total of 540 patients were included in the study (Figure 1). The median age was 69 years (IQR: 57-79), and 43.1% of the patients were female. Among the study population, 71.5% presented to the ED following cardiac arrest. The overall 30-day mortality rate was 74.8%.

When 30-day mortality rates were compared across all patients, those who died were significantly older and had a higher prevalence of HT and CAD, whereas DM was more common among survivors ($p<0.05$ for all). Mortality rates were significantly elevated in patients with cardiovascular etiologies, while patients with metabolic or neurological etiologies exhibited lower mortality rates. Laboratory findings revealed that non-survivors were more acidotic and had significantly higher Partial carbon dioxide pressure (pCO_2), lactate, INR, creatinine, and potassium ($p<0.05$ for all parameters) (Table 1).

In the non-arrest group, the 30-day mortality rate was 21.4%. Among these patients, those who died were more likely to be older and male, and have a higher prevalence of HT and CAD. Analysis of diagnostic categories revealed that mortality was significantly higher in patients with sepsis, metabolic causes, and isolated respiratory arrest, whereas it was lower in those with neurological etiologies, DKA, and seizure-related presentations ($p<0.05$ for all values). There were no statistically significant differences in arterial blood gas parameters between survivors and non-survivors. However, non-survivors exhibited significantly higher levels of lactate and creatinine, along with lower hemoglobin concentrations ($p<0.05$ for all values) (Table 2).

In the subgroup of patients with non-arrest-related severe acidosis, univariate analyses were initially conducted to assess the impact of the variables listed in Table 3 on 30-day mortality. This analysis identified advanced age, male sex, presence of sepsis, metabolic causes, isolated respiratory arrest, HT, CAD, elevated pCO_2 and creatinine levels, and reduced hemoglobin as significant factors associated with increased mortality ($p<0.05$ for all values).

Discussion

Although the adverse effects of severe acidosis are well-known, its impact on mortality and the factors influencing it remain unclear. In this study, conducted to examine the factors that may affect mortality, we identified several key variables that influence patient outcomes. The 30-day mortality rate in patients with severe acidosis was 74.8%, while the mortality rate for non-arrested patients was 21.4%. This rate was 68% in one study and 83% in another (4,5). In another study, the mortality rate for patients with a history of arrest was 90% (14). Although mortality rates are generally high, the significant survival rate observed in this patient group highlights the importance of early diagnosis of potential influencing factors and a timely treatment approach.

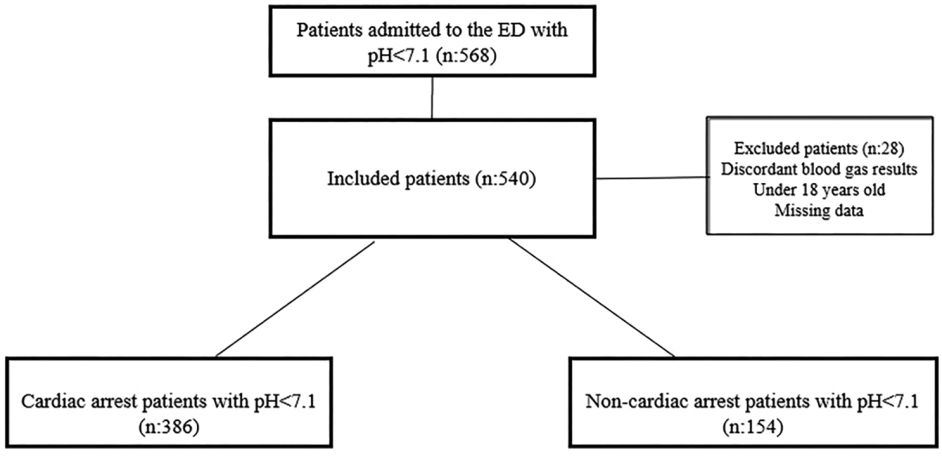

Figure 1. Flow chart of patients
ED: Emergency department

Table 1. Comparison of demographic and laboratory values of all patients according to 30-day mortality			
	Survived (n=136)	**Deceased (n=404)**	**p value**
Age, years, median (IQR: 25-75)	56.5 (34-70.75)	72 (62-81)	<0.001
Gender, female, (n %)	54 (39.7%)	179 (44.3%)	0.349
*Diagnosis, (n %)			
Cardiopulmonary arrest	15 (11.0%)	371 (91.8%)	<0.001
Hemorrhage/trauma	10 (7.4%)	24 (5.9%)	0.558
Intoxication	3 (2.2%)	7 (1.7%)	0.718
Sepsis	13 (9.6%)	49 (12.1%)	0.416
Metabolic	29 (21.3%)	32 (7.9%)	<0.001
Neurological	24 (17.6%)	4 (1.0%)	<0.001
Unknown	0 (0.0%)	138 (34.2%)	<0.001
Isolated respiratory arrest	9 (6.6%)	7 (1.7%)	0.007
Comorbidities, (n %)			
Hypertension	50 (36.8%)	188 (46.5%)	0.047
Diabetes	70 (51.5%)	150 (37.1%)	0.003
COPD	17 (12.5%)	75 (18.6%)	0.104
CAD	29 (21.3%)	145 (35.9%)	0.002
CRF	17 (12.5%)	27 (6.7%)	0.032
CVD	8 (5.9%)	27 (6.7%)	0.743
Malignancy	12 (8.8%)	46 (11.4%)	0.404
Laboratory, median (IQR: 25-75)			
pH	7.02 (6.93-7.06)	6.92 (6.81-7.01)	<0.001
pCO_2, mmHg	37.9 (25.6-54.5)	67.65 (49.22-86.47)	<0.001
HCO_3, mmol/L	9.4 (7.3-11.7)	9.3 (6.85-11.7)	0.661
BE, mmol/L	20.55 (16.42-24.2)	19.3 (15.3-23.5)	0.064
Anion gap, mEq/L	25.45 (18.02-31.45)	22.5 (17.27-27.52)	0.029
Lactate, mmol/L	4.74 (2.73-8.77)	9.98 (6.58-12.79)	<0.001
Hemoglobin, g/dL	14.3 (11.8-16.17)	11.8 (9.8-13.72)	<0.001
INR	1.28 (1.13-1.47)	1.53 (1.26-1.85)	<0.001
Glucose, mg/dL	218 (143.75-504.5)	236 (137-360)	0.136
Creatine, mg/dL	1.32 (1.1-1.87)	1.53 (1.21-2.3)	0.002
Potassium, mmol/L	4.94 (4.24-5.74)	5.52 (4.56-6.61)	<0.001

*Some patients were assigned more than one diagnosis, COPD: Chronic obstructive pulmonary disease, CAD: Coronary artery disease, CRF: Chronic renal failure, CVD: Cerebrovascular disease, pCO_2: Partial carbon dioxide pressure, BE: Base excess HCO_3: Bicarbonate, INR: International normalization rate, IQR: Interquartile range

Table 2. Comparison of demographic and laboratory values of non-cardiac arrested patients according to 30-day mortality			
	Survived (n=121)	**Deceased (n=33)**	**p value**
Age, years, median (IQR: 25-75)	53 (31-70)	75 (68-84)	<0.001
Gender, female, (n %)	48 (39.7%)	20 (60.6%)	0.032
*Diagnosis, (n %)			
Cardiovasculer disease	17 (14.0%)	8 (24.2%)	0.159
Hemorrhage/trauma	10 (8.3%)	1 (3.0%)	0.459
Intoxication	3 (2.5%)	2 (6.1%)	0.291
Sepsis	9 (7.4%)	15 (45.5%)	<0.001
Metabolic	29 (24.0%)	16 (48.5%)	0.006
Neurological	23 (19.0%)	1 (3.0%)	0.025
Isolated respiratory arrest	9 (7.4%)	7 (21.2%)	0.047
Diabetic ketoacidosis	38 (31.4%)	4 (12.1%)	0.027
Epileptic seizure	23 (19%)	0 (0.0%)	0.004
Comorbidities, (n %)			
Hypertension	44 (36.4%)	19 (57.6%)	0.028
Diabetes	67 (55.4%)	21 (63.6%)	0.395
COPD	15 (12.4%)	7 (21.2%)	0.259
CAD	26 (21.5%)	15 (45.5%)	0.006
CRF	17 (14.0%)	6 (18.2%)	0.584
CVD	7 (5.8%)	3 (9.1%)	0.447
Malignancy	10 (8.3%)	4 (12.1%)	0.501

Table 2. Continued			
	Survived (n=121)	**Decased (n=33)**	**p value**
Laboratory, median (IQR: 25-75)			
pH	7.02 (6.93-7.07)	7.03 (6.91-7.06)	0.824
pCO_2, mmHg	35.8 (24.55-52.6)	42.6 (30.7-60.4)	0.082
HCO_3, mmol/L	9.0 (7.22-11.4)	9.8 (6.3-12.05)	0.646
BE, mmol/L	20.9 (17.25-24.6)	19.1 (15.4-25.4)	0.248
Anion gap	25.95 (18.25-31.87)	23.6 (16.8-27.8)	0.156
Lactate, mmol/L	4.25 (2.57-7.66)	6.78 (4.52-9.5)	0.041
Hemoglobin, g/dL	14.3 (11.9-16.3)	11.7 (9.5-13.35)	<0.001
INR	1.26 (1.12-1.46)	1.35 (1.17-1.65)	0.089
Glucose, mg/dL	211 (140-545.5)	217 (139.5-295)	0.359
Creatinine, mg/dL	1.33 (1.13-2.13)	2.31 (1.54-5.45)	<0.001
Potassium, mmol/L	4.97 (4.27-5.81)	5.44 (4.62-5.9)	0.174

*Some patients were assigned more than one diagnosis, COPD: Chronic obstructive pulmonary disease, CAD: Coronary artery disease, CRF: Chronic renal failure, CVD: Cerebrovascular disease, pCO_2: Partial carbon dioxide pressure, HCO_3: Bicarbonate, BE: Base excess, INR: International normalization rate, IQR: Interquartile range

Table 3. Univariate regression model to predict the 30 day mortality in noncardiac arrest patients			
	Wald	**p value**	**Odds ratio (95% CI)**
Age, median (IQR: 25-75)	18.187	<0.001	1.062 (1.033-1.092)
Gender, female, (n %)	4.475	0.034	0.427 (0.194-0.939)
*Diagnosis, (n %)			
Cardiovasculer disease	1.933	0.164	1.958 (0.759-5.047)
Intoxication	0.992	0.319	2.538 (0.406-15.857)
Sepsis	22.582	<0.001	10.370 (3.952-27.212)
Metabolic	7.179	0.007	2.986 (1.341-6.646)
Isolated respiratory arrest	4.851	0.028	3.350 (1.142-9.826)
Comorbidities, (n %)			
Hypertension	4.683	0.030	2.375 (1.085-5.199)
Diabetes mellitus	0.719	0.396	1.410 (0.637-3.122)
COPD	1.607	0.205	1.903 (0.704-5.143)
CAD	7.241	0.007	3.045 (1.353-6.851)
CRF	0.347	0.556	1.359 (0.489-3.779)
Malignancy	0.461	0.497	1.531 (0.448-5.235)
Laboratory, median (IQR: 25-75)			
pH	0.286	0.593	0.419 (0.017-10.124)
pCO_2, mmHg	3.676	0.045	1.017 (1.000-1.034)
HCO_3, mmol/L	0.304	0.581	1.031 (0.924-1.151)
BE, mmol/L	1.124	0.289	0.967 (0.909-1.029)
Anion gap	1.465	0.226	0.971 (0.927-1.018)
Lactate, mmol/L	2.182	0.140	1.054 (0.983-1.131)
Hemoglobin, g/dL	12.233	<0.001	0.775 (0.672-0.894)
INR	0.167	0.683	0.962 (0.800-1.157)
Glucose, mg/dL	3.070	0.080	0.998 (0.996-1.000)
Creatinine, mg/dL	3.972	0.046	1.154 (1.002-1.328)
Potassium, mmol/L	1.520	0.218	1.230 (0.885-1.708)

*Some patients were assigned more than one diagnosis, COPD: Chronic obstructive pulmonary disease, CAD: Coronary artery disease, CRF: Chronic renal failure, pCO_2: Partial carbon dioxide pressure, HCO_3: Bicarbonate, BE: Base excess, INR: International normalization rate, IQR: Interquartile range

When the diagnoses of the patients were analyzed, cardiovascular mortality rates were high in the general patient group, while metabolic mortality rates were low. In the non-arrested patient group, mortality rates were significantly higher in cases of sepsis and metabolic causes compared to patients with DKA.

In one study, the primary disorder was not associated with mortality, whereas in another study, the mortality rate was found to be low in acidosis with metabolic causes (1-4). Mortality rates in DKA have been found to be low in various studies. We believe that the relatively low mortality rate in DKA is due to the contribution of underlying secondary pathologies to the condition. The rate may have been higher in our study because cardiovascular diseases are among the common causes of death. However, early aggressive treatment in patients with suspected sepsis, among those without a history of arrest, but with severe acidosis, may serve as an important intervention to reduce mortality.

Analysis of blood gas parameters revealed that non-survivors were more acidotic and had significantly elevated levels of pCO_2 and lactate. Previous research has suggested that the severity of acidosis may be associated with patient prognosis (15). In the non-arrest subgroup, no significant differences were observed in pH or pCO_2 levels between survivors and non-survivors; however, lactate levels remained persistently elevated in those who died. Interestingly, although elevated lactate levels were also observed in patients presenting with epileptic seizures, no mortality occurred in this subgroup. This finding is consistent with a previous study reporting elevated lactate levels in seizure patients, attributed to transient anaerobic metabolism (16). Several studies have proposed that hyperlactatemia may be linked to increased mortality risk (1-14). In this context, we suggest that elevated lactate levels when interpreted in conjunction with the underlying etiology may serve as a valuable marker in guiding clinical decision-making in critically ill patients.

In the study by Gutgold et al. (4), it was reported that high CO_2 levels may be associated with mortality, while no relationship was found between pH and mortality. In another study, a high CO_2 level has been found to be associated with mortality (17). However, Allyn et al. (14) did not find a relationship between CO_2 levels and mortality. Since elevated CO_2 levels may reflect increased physiological dead space, they could indicate a prolonged duration since the onset of cardiac arrest, potentially leading to higher mortality because of irreversible tissue damage. This discrepancy may also be explained by differences in study settings, as our study was conducted in the ED, whereas the comparison study was conducted in an intensive care unit.

We found that potassium, creatinine, INR, and hemoglobin among laboratory parameters, as well as hemoglobin and creatinine in the non-arrest group, were associated with mortality. Although creatinine was found to be associated with mortality in the study by Allyn et al. (14), it was noted that explaining this relationship would be difficult. Paz et al. (5) reported that hyperkalemia could be a determinant of mortality. In a study conducted in patients diagnosed with acute renal failure, it was shown that both creatinine levels and hyperkalemia may be associated with mortality (18,19). Another study found that low hemoglobin levels could be associated with mortality (20). Therefore, we believe that high creatinine, high potassium, and low hemoglobin levels may serve as predictors of mortality in critically ill patients.

In our univariate analysis, sepsis, metabolic causes, and elevated pCO_2 levels were significantly associated with increased mortality. Advanced age was also found to be an independent predictor of mortality [odds ratio (OR): 1.062; 95% confidence interval: 1.033-1.092; $p<0.001$], which is consistent with the decline in physiological reserve and the increased burden of comorbidities observed in older patients. When evaluated by diagnostic categories, sepsis (OR: 10.37; $p<0.001$), isolated respiratory causes (OR: 3.35; $p=0.028$), and metabolic causes (OR: 2.99; $p=0.007$) emerged as significant risk factors for mortality. The markedly high mortality observed in the sepsis group may be explained by mechanisms such as multi-organ dysfunction, cytokine storm, and lactic acidosis. These findings suggest that the systemic impact of sepsis-induced acidosis may be more devastating than that of other etiologies (21).

Among comorbid conditions, CAD was significantly associated with mortality (OR: 3.045; $p=0.007$). This finding may be attributed to reduced cardiovascular reserve and increased susceptibility to myocardial ischemia during episodes of systemic hypoperfusion. HT also emerged as a significant risk factor (OR: 2.375; $p=0.030$), suggesting that chronic vascular damage may exacerbate clinical outcomes during acute physiological stress. Regarding laboratory parameters, low hemoglobin was inversely associated with mortality (OR: 0.775; $p<0.001$). This association likely reflects impaired oxygen delivery capacity in the setting of reduced hemoglobin concentration, contributing to tissue hypoxia and adverse outcomes (22). In addition, elevated pCO_2 levels (OR: 1.017; $p=0.045$) and increased creatinine levels (OR: 1.154; $p=0.046$) were found to be significantly associated with mortality. These findings highlight the prognostic significance of respiratory failure and acute or chronic renal dysfunction, respectively (7). These findings may serve as a cautionary note in predicting mortality in patients with severe acidosis in the ED.

Study Limitations

This study has several limitations. First, its retrospective design led to instances of missing data, which may have introduced selection bias. Second, we did not subclassify the specific types of acid-base disturbances (e.g., high anion gap vs. normal anion gap metabolic acidosis), which limited our ability to draw definitive conclusions regarding the underlying etiologies. Third, overlapping primary diagnoses in some patients may have confounded outcome comparisons across diagnostic categories; for instance, a patient diagnosed with both DKA and sepsis was included in both groups, potentially distorting mortality estimates. Additionally, all blood gas samples analyzed were venous rather than arterial, precluding accurate assessment of oxygenation status. Finally, although a multivariable regression model was initially planned to identify independent predictors of mortality in non-cardiac arrest patients with severe acidosis, the analysis could not be performed due to statistical limitations. Specifically, modeling attempts resulted in perfect separation, where certain predictors (e.g., sepsis, age) nearly perfectly distinguished survivors from non-survivors. This issue, which commonly arises in small sample sizes or sparsely distributed

data, prevents reliable estimation of regression coefficients. Therefore, only unadjusted odds ratios from the univariate analysis are reported.

Conclusion

Although the majority of patients presenting to the ED with severe acidosis are those who have experienced cardiac arrest, a notable mortality rate also exists among patients with non-arrest-related severe acidosis. This study highlights that severe acidosis, particularly in the ED setting, is associated with high 30-day mortality, especially among patients presenting with cardiac arrest. However, even in the non-arrest subgroup, a substantial mortality rate of 21.4% was observed. Key predictors of mortality included advanced age, male sex, HT, CAD, sepsis, metabolic etiologies, isolated respiratory arrest, elevated pCO_2 and creatinine levels, and low hemoglobin. These findings underscore the importance of early recognition and targeted management of high-risk clinical and laboratory features to improve outcomes in patients with severe acidosis.

Ethics

Ethics Committee Approval: The Ethics Committee of University of Health Sciences Türkiye, Ankara Atatürk Sanatorium Training and Research Hospital approved the study protocol in accordance with (decision number: 2024-BÇEK/11, date: 14.02.2024) the ethical principles of the Declaration of Helsinki and current Good Clinical Practice guidelines.

Informed Consent: This is a retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Z.S.Ö., H.Ö.O., Concept: E.E., Design: E.E., Data Collection or Processing: Z.S.Ö., H.Ö.O., S.A., Analysis or Interpretation: E.E., Y.C., Literature Search: E.E., Writing: Z.S.Ö.

Conflict of Interest: No conflict of interest was declared by the authors.

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