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A novel model for early prediction of in-hospital mortality in seawater drowning: the SNOP score

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Abstract

Background Drowning is a leading cause of preventable mortality worldwide; however, early in-hospital risk stratification remains limited. Although tools such as the Szpilman score assist in early severity assessment, they may not fully capture the evolving clinical status after admission. This study aimed to develop a simplified and objective model based on readily available parameters to predict in-hospital mortality following seawater drowning.

Methods This retrospective study was conducted at a referral emergency department (ED) in northern Turkey between July 1, 2011, and December 31, 2024. Of 190 patients initially included, 166 with complete clinical and laboratory data were analyzed. Data were obtained from institutional and national health information systems. Clinical, physiological, and biochemical variables were assessed. Predictors of in-hospital mortality were identified using receiver operating characteristic (ROC) analysis and multivariable logistic regression. Variables with near-perfect discrimination (e.g., GCS, pH, Szpilman score) were excluded to avoid overfitting.

Results Among the 166 patients, 34 (20.5%) died during hospitalization. CPR and endotracheal intubation rates were significantly higher among non-survivors (CPR: 97.1% vs. 0%; intubation: 97.1% vs. 2.3%; both $p < 0.001$). Non-survivors also presented with lower GCS (median 3 vs. 15), lower arterial pH, and higher Szpilman scores (all $p < 0.001$). ROC analysis identified four potential predictors with AUC values between 0.90 and 0.95—pCO_2, lactate, SpO_2, and sodium—all showing significant discriminatory capacity ($p < 0.001$). These variables were entered into a binary logistic regression model, from which serum sodium (OR = 2.110; 95% CI: 1.310–3.401; $p = 0.002$) and SpO_2 (OR = 0.902; 95% CI: 0.847–0.961; $p = 0.001$) emerged as independent predictors. These formed the basis of the SNOP score (Saturation and Natremia-based Outcome Predictor), a two-parameter logistic model demonstrating excellent performance: AUC = 0.996, sensitivity = 99.0%, specificity = 96.2%, and overall accuracy = 98.4%.

Conclusion: The SNOP score is a simple, ED-specific tool for early prediction of in-hospital mortality in seawater drowning. It complements existing assessment systems by incorporating objective, admission-based parameters. Prospective multicenter validation is warranted to confirm its clinical applicability and support broader implementation.

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Keywords Drowning, Hospital mortality, Seawater, Sodium, Oxygen saturation, Emergency medicine, Prognostic model

Introduction

Drowning remains a substantial yet underrecognized global health issue, consistently ranked among the leading causes of unintentional injury-related deaths worldwide [1–3]. According to the World Health Organization's 2024 Global Status Report on Drowning Prevention, an estimated 300,000 people died from drowning in 2021, with 92% of these deaths occurring in low- and middle-income countries [1]. Children under five years of age and young adult males are disproportionately affected, highlighting the urgent need for targeted prevention strategies and improved acute care systems [1, 4].

To promote clarity and standardization, the 2002 World Congress on Drowning—later endorsed by the WHO—defined drowning as "the process of experiencing respiratory impairment from submersion or immersion in liquid," encompassing both fatal and non-fatal outcomes [5, 6]. This definition replaced outdated terms such as "near-drowning," which are no longer recommended in clinical or scientific contexts [5, 7].

Despite advances in terminology and prevention, in-hospital prognostication following drowning remains challenging. The Szpilman classification, introduced in 1997, stratifies drowning severity based on initial cardiopulmonary status and has been widely adopted due to its simplicity and utility in prehospital triage [8, 9]. However, hospital-based risk assessment often requires additional physiological and biochemical parameters due to dynamic metabolic and respiratory changes.

Recent studies have identified objective markers such as the Glasgow Coma Scale (GCS), arterial pH, lactate, peripheral oxygen saturation (SpO_2), and serum sodium (Na^+) as significant predictors of mortality [10–14]. While each has clinical value, no validated scoring system currently integrates these variables into a cohesive model tailored for emergency department (ED) use.

This gap has meaningful clinical implications, particularly in early emergency care. Physicians are often required to make high-stakes decisions about intensive care admission or escalation of treatment in the face of limited prognostic information [15]. A robust, objective, and easily implementable scoring system could enhance early risk stratification, guide clinical decision-making, and improve resource allocation [5, 7, 14].

In this context, the present study aimed to develop a simplified and objective prognostic model to predict in-hospital mortality in patients presenting to the emergency department following seawater drowning.

Materials and methods

Study design, setting, and ethical considerations

This retrospective, cross-sectional observational study was conducted in the emergency department of a referral center located in the northern coastal region of Turkey. All patients diagnosed with drowning between July 1, 2011, and December 31, 2024, were eligible for inclusion.

In the study setting, patients were managed within Turkey's publicly funded, physician-supervised EMS system. Advanced Life Support (ALS)-equipped ambulances, staffed by paramedics and emergency medical technicians (without on-site physicians), typically reached urban and coastal areas within 10 min.

Ethical approval was obtained from the Sinop University Ethics Committee for Human Research (Approval No: 2025/113, Date: February 28, 2025). The study was conducted in accordance with the principles of the Declaration of Helsinki. Informed consent was waived due to the retrospective design and anonymized nature of the data.

Study population and data collection

A total of 190 cases were initially screened. Of these, 15 patients were excluded due to missing survival outcome and essential laboratory data. An additional 9 patients had known outcomes but lacked key predictor variables (e.g., serum sodium, SpO_2, arterial pH) and were excluded based solely on data completeness. All exclusions were made prior to statistical modeling and independent of outcomes to minimize selection bias. Two independent researchers assessed eligibility and data completeness by triangulating emergency department notes, physician documentation, and registry records. The final cohort comprised 166 patients aged 2 to 77 years, with no age-based exclusion criteria.

All available clinical data were extracted from the institutional electronic health record (EHR) system using a standardized case report form. Collected variables included patient demographics (age, sex, province of origin), event characteristics (presence of trauma, prehospital and/or in-hospital cardiopulmonary resuscitation (CPR), endotracheal intubation), and clinical severity indices such as Glasgow Coma Scale (GCS), Szpilman score, and length of hospital stay. The primary outcome was in-hospital mortality, defined as death occurring after emergency department presentation but prior to discharge. The variable was recorded as binary: deceased vs. survived to discharge.

Data on CPR were obtained from both prehospital EMS documentation and emergency department records. We

recorded whether CPR was initiated in the field (by EMS teams) or after hospital arrival (ED-based CPR). However, information regarding bystander-initiated CPR prior to EMS arrival was not systematically recorded in either EMS transfer reports or hospital records and was therefore not included in the analysis.

Biochemical and physiological parameters obtained at initial presentation included arterial blood gas results (pH, pCO_2, pO_2, HCO_3^-, SaO_2, lactate), serum electrolytes (sodium, potassium, chloride), renal function tests (urea, creatinine), hepatic enzymes (AST, ALT, LDH), cardiac biomarkers (troponin, CK, CK-MB), and hematologic indices (white blood cell count, hemoglobin, hematocrit).

Outcome data for patients managed entirely at the study center were extracted from institutional records. For patients transferred to other facilities, outcomes were verified using the national electronic health record system, which provides cross-institutional access to longitudinal clinical data.

Table 1 Baseline characteristics and emergency interventions according to clinical outcome

	Discharged (*n* = 132)	**Deceased (*n* = 34)**			**Missing data**
	$\bar{X}$±SD/n (%)	$\bar{X}$±SD/n (%)	t/Chi-square	p-value	n (%)
Age (years)	30.05 ± 19.31	36.2 ± 19.21	1.659	0.099	0 (0)
Sex			0.072	0.835	0 (0)
Male	94 (71.2)	25 (73.5)			
Female	38 (28.8)	9 (26.5)			
Province of origin			1.325	0.305	11 (6.6)
Sinop (local)	46 (37.1)	15 (48.4)			
Other	78 (62.9)	16 (51.6)			
Trauma			2.178	0.166	0 (0)
Absent	123 (93.2)	29 (85.3)			
Present	9 (6.8)	5 (14.7)			
CPR			159.906	< 0.001	0 (0)
Not Performed	132 (100)	1 (2.9)			
Performed	0 (0)	33 (97.1)			
Endotracheal intubation			143.022	< 0.001	0 (0)
Intubated	3 (2.3)	33 (97.1)			
Not Intubated	129 (97.7)	1 (2.9)			

Exact p-values were calculated for Chi-square tests where appropriate

$\bar{X}$ Mean, *SD* Standard deviation, *CPR* Cardiopulmonary resuscitation

"Drowning" was used in line with WHO/ILCOR definitions to refer to all cases involving respiratory impairment due to submersion or immersion, regardless of outcome. The obsolete term "near-drowning" was intentionally avoided to maintain consistency with current international terminology.

Statistical analysis

Nominal variables were summarized as frequencies and percentages, ordinal variables as medians (min–max), and continuous variables as means ± standard deviations. Between-group comparisons were performed using the Chi-square test (with exact p-values) for nominal variables, the Mann–Whitney U test for ordinal variables, and either the independent-samples t-test or Welch's t-test for continuous variables, depending on variance homogeneity. Effect sizes (Cohen's d for continuous and rank-biserial correlation for ordinal variables) were calculated to complement significance testing.

Receiver operating characteristic (ROC) analysis was conducted to assess the discriminative ability of candidate predictors, with the optimal cut-off values determined by the Youden index. Variables with AUC > 0.90 were included in a multivariable logistic regression model using the Backward Likelihood Ratio method, in which predictors with $p > 0.10$ were sequentially excluded.

To evaluate model performance, the dataset was randomly split into a training set (70%) and a test set (30%), and classification accuracy was reported for both. All analyses were conducted using SPSS version 27, with statistical significance set at $p < 0.05$.

Results

Patient characteristics

A total of 190 patients with drowning were initially identified. Of these, 15 patients were excluded due to missing survival outcome and essential laboratory data. An additional 9 patients had known survival status but lacked key predictor variables (e.g., serum sodium, arterial pH, SpO_2) and were excluded based solely on data incompleteness. Because the extent and non-random pattern of missingness precluded imputation, these cases were not included in modeling. The final analytic cohort comprised 166 patients, of whom 132 (79.5%) survived to hospital discharge and 34 (20.5%) died during hospitalization (Table 1; Fig. 1).There were no statistically significant differences between survivors and non-survivors in terms of mean age (30.05 ± 19.31 vs. 36.2 ± 19.21 years, $p = 0.099$), sex (male: 71.2% vs. 73.5%, $p = 0.835$), province of origin (37.1% vs. 48.4%, $p = 0.305$), or presence of trauma (6.8% vs. 14.7%, $p = 0.166$) (Table 1).

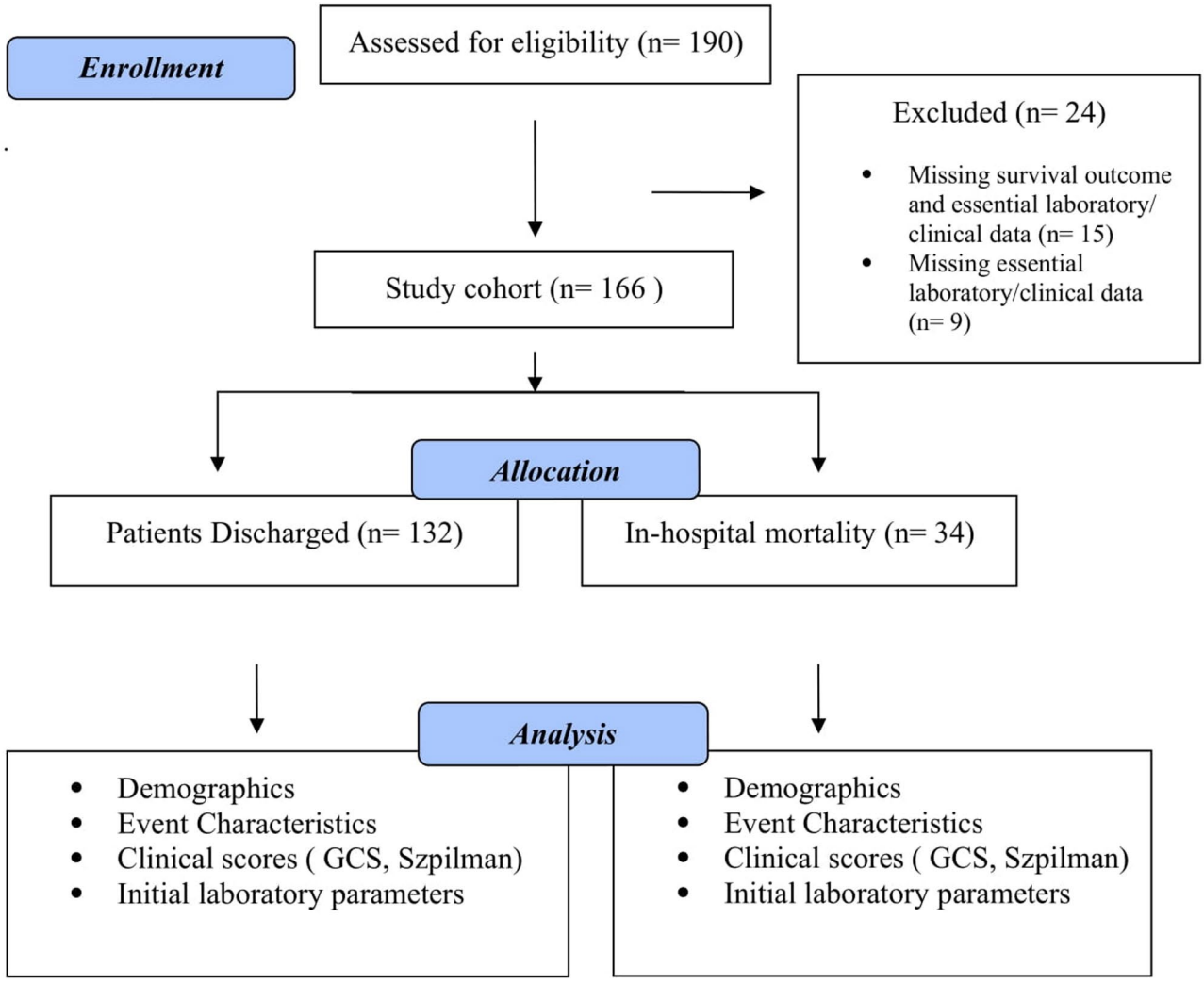

Fig. 1 Flowchart of patient inclusion and analysis this figure outlines the patient screening and enrollment process. The flow diagram follows the CONSORT format and includes stages of eligibility assessment, exclusion reasons, and final analytic cohort distribution

Incident characteristics and prehospital interventions

Prehospital CPR and endotracheal intubation were both strongly associated with in-hospital mortality. CPR was performed in 97.1% of non-survivors and in none of the survivors ($p < 0.001$). Similarly, endotracheal intubation was required in 97.1% of non-survivors compared to 2.3% of survivors ($p < 0.001$) (Table 2).

Of the 164 patients analyzed, CPR was performed in 33 (19.9%), predominantly initiated by EMS and continued in the ED ($n = 29$); the remaining 4 cases were initiated in the ED. ROSC was not achieved in 12 patients (36.4%), while 21 (63.6%) regained circulation, most commonly after > 15 min (36.4%). Endotracheal intubation was performed in 36 patients (21.7%), including 2 by EMS and 36 by ED teams; both EMS-intubated cases required re-intubation upon ED arrival (Table 2).

Clinical parameters and laboratory findings

Non-survivors had significantly longer hospital stays (median 5.5 vs. 0.97 days, $p = 0.041$), lower Glasgow Coma Scale scores (median 3.0 vs. 15.0, $p < 0.001$), and higher Szpilman scores (median 5.0 vs. 1.0, $p < 0.001$) (Table 3).

Arterial blood gas analysis revealed more pronounced respiratory and metabolic derangements among non-survivors, including lower pH (6.68 vs. 7.27, $p < 0.001$), higher pCO_2 (102.3 vs. 39.8 mmHg, $p < 0.001$), and lower HCO_3^- (11.8 vs. 18.3 mmol/L, $p < 0.001$). SaO_2 (71.6% vs. 94.8%, $p = 0.002$) and SpO_2 (63.2% vs. 95.4%, $p < 0.001$) were also significantly lower in non-survivors (Table 3).

Non-survivors had significantly higher serum levels of lactate (18.5 vs. 6.8 mmol/L, $p < 0.001$), creatinine ($p < 0.001$), AST ($p = 0.033$), ALT ($p = 0.034$), LDH ($p = 0.006$), sodium ($p < 0.001$), chloride ($p = 0.001$), and potassium ($p < 0.001$). No statistically significant

Table 2 Resuscitation interventions and patients outcomes

Total Patients	*n* = 166	100%
CPR		
No CPR	133	80.1
CPR performed	33	19.9
Initiated by EMS and continued in ED	29	-
Initiated by ED	4	-
Time to ROSC (among *n* = 34)		
No response	12	36.4
5 min	2	6.0
10 min	6	18.2
13 min	1	3.0
>15 min	12	36.4
Endotracheal Intubation		
Not intubated	130	78.3
Emergency Medical Service	2	1.2
Emergency Department	36*	21.7

CPR cardiopulmonary resuscitation, *EMS* Emergency Medical Service, *ED* Emergency Department, *ROSC* Return of spontaneous circulation

*Two patients intubated in EMS were also re-intubated in the ED

differences were found in WBC, hemoglobin, hematocrit, troponin, CK, or CK-MB levels (all $p > 0.05$).

ROC analysis of prognostic markers

ROC analysis was conducted to evaluate the predictive performance of individual clinical and biochemical markers for in-hospital mortality. The Glasgow Coma Scale (AUC = 1.000), Szpilman score (AUC = 0.989), and arterial pH (AUC = 0.975) demonstrated excellent discriminatory performance (AUC > 0.95) and were therefore presented separately due to their near-perfect accuracy (Table 4; Fig. 2).

To reduce the risk of overfitting, variables with AUC > 0.95 were not included in multivariable logistic regression. Candidate variables with AUC values between 0.90 and 0.95 included pCO_2 (AUC = 0.932), lactate (AUC = 0.923), SpO_2 (AUC = 0.918), and sodium (AUC = 0.907), all of which showed statistically significant discriminatory capacity ($p < 0.001$) (Table 4; Fig. 3).

Table 3 Clinical scores and initial laboratory parameters according to in-hospital outcome

	Discharge		Deceased		t/z	p	Cohen d	Missing Data
	n	$\bar{X}$±SD/Me (Min-Max)	n	$\bar{X}$±SD/Me (Min-Max)				n (%)
Length of Stay (days)	123	0.97 ± 1.6	28	5.5 ± 11.15	2.147	0.041*	0.914	15 (9)
GCS	132	15.0 (8.0–15.0)	34	3.0 (3.0–9.0)	−11.358	< 0.001[a]	0.999[b]	0 (0)
Szpilman Score	131	1.0 (0.0–4.0)	34	5.0 (2.0–6.0)	−9.174	< 0.001[a]	−0.979[b]	1 (0.6)
Initial Laboratory Findings								
pH	128	7.27 ± 0.16	32	6.68 ± 0.25	−12.771	< 0.001*	−3.256	6 (3.6)
pCO_2 (mmHg)	128	39.81 ± 11.72	32	102.32 ± 43.48	8.059	< 0.001*	2.849	6 (3.6)
pO_2 (mmHg)	128	53.64 ± 31.78	32	62.88 ± 64.11	0.791	0.434*	0.230	6 (3.6)
HCO_3 (mmol/L)	128	18.29 ± 5.56	32	11.83 ± 4.42	−6.101	< 0.001	−1.206	6 (3.6)
SaO_2 (ABG)	126	69.71 ± 23.74	29	45.91 ± 37.39	−3.279	0.002*	−0.889	11 (6.6)
SpO_2 (Pulse) (%)	102	93.85 ± 9.26	26	29.69 ± 34.36	−9.435	< 0.001*	−3.686	38 (22.9)
CO (%)	104	1.59 ± 2.11	25	1.09 ± 0.99	−1.150	0.252	−0.256	37 (22.3)
Lactate (mmol/L)	88	6.81 ± 5.47	16	18.48 ± 5.87	7.762	< 0.001	2.110	62 (37.3)
WBC ($\times 10^9$/L)	131	12.59 ± 15.36	32	11.62 ± 4.68	−0.354	0.724	−0.070	3 (1.8)
Hemoglobin (g/dL)	131	14.36 ± 1.79	32	14.68 ± 2.27	0.726	0.472*	0.165	3 (1.8)
Hematocrit (%)	131	42.38 ± 5.48	32	45.66 ± 7.04	2.461	0.018*	0.564	3 (1.8)
Urea (mg/dL)	131	29.34 ± 9.52	33	30 ± 7.51	0.368	0.713	0.072	2 (1.2)
Creatinine (mg/dL)	131	0.86 ± 0.24	33	1.25 ± 0.3	7.837	< 0.001	1.526	2 (1.2)
AST (U/L)	131	32.47 ± 40.47	33	201 ± 433.37	2.231	0.033*	0.860	2 (1.2)
ALT (U/L)	131	23.73 ± 19.69	33	216 ± 499.13	2.212	0.034*	0.864	2 (1.2)
LDH (U/L)	129	303.48 ± 124.24	31	629.49 ± 612.6	2.948	0.006*	1.126	6 (3.6)
Troponin (ng/mL)	90	9.28 ± 26.13	24	43.77 ± 140.01	1.201	0.242*	0.510	52 (31.3)
CK-MB (U/L)	95	1.81 ± 1.62	26	2.37 ± 1.99	1.472	0.144	0.326	45 (27.1)
CK (U/L)	126	496.27 ± 2841.29	31	203.54 ± 122.49	−0.572	0.568	−0.115	9 (5.4)
Sodium (mmol/L)	131	140.58 ± 3.4	33	150.42 ± 6.26	8.722	< 0.001*	2.388	2 (1.2)
Chloride (mmol/L)	131	108.49 ± 3.67	33	110.97 ± 3.64	3.476	0.001	0.677	2 (1.2)
Potassium (mmol/L)	131	4.18 ± 0.54	32	6.04 ± 2.01	5.167	< 0.001*	1.843	3 (1.8)

$\bar{X}$ Mean, *SD* Standard deviation, *Me* Median, *Min* Minimum, *Max* Maximum. *GCS* Glasgow Coma Scale, *SaO_2 (ABG)* Arterial oxygen saturation measured via arterial blood gas analysis. *SpO_2 (Pulse)* Peripheral oxygen saturation measured by pulse oximetry, *CO* Carboxyhemoglobin, *WBC* White blood cell count, *AST* Aspartate aminotransferase, *ALT* Alanine aminotransferase, *LDH* Lactate dehydrogenase, *CK* Creatine kinase, *CK-MB* Creatine kinase myocardial band, *Urea* Blood urea nitrogen. Welch's t-test

[a] Mann–Whitney U test

[b] Rank-biserial correlation coefficient

Table 4 ROC curve analysis of clinical and laboratory predictors of in-hospital mortality

					%95 CI					Cut-off Value
	Pos/Neg	AUC	SE	p-value	CI lower	CI upper	Se	Sp	Youden Index	
GCS	34/132	1.000	< 0.001	< 0.001	0.999	1.000	1.000	0.992	0.992	≤ 9.5
Szpilman Score	34/131	0.989	0,010	< 0.001	0.969	1.000	0.971	0.985	0.956	≥ 3.5
pH	32/128	0.975	0.011	< 0.001	0.955	0.996	0.969	0.891	0.860	≤ 7.095
	26/102	0.918	0.044	< 0.001	0.832	1.000	0.846	0.922	0.768	≤ 81.5
HCO_3 (mmol/L)	32/128	0.818	0.038	< 0.001	0.743	0.892	0.906	0.680	0.586	≤ 16.55
LDH (U/L)	31/129	0.801	0.050	< 0.001	0.703	0.899	0.774	0.767	0.541	≥ 343.65
Creatinine (mg/dL)	33/131	0.842	0.035	< 0.001	0.773	0.911	0.667	0.870	0.537	≥ 1.145
Potassium (mmol/L)	32/131	0.830	0.058	< 0.001	0.717	0.942	0.813	0.847	0.660	≥ 4.65
Lactate (mmol/L)	16/88	0.923	0.027	< 0.001	0.871	0.975	1.000	0.750	0.750	≥ 9.515
	33/131	0.907	0.036	< 0.001	0.836	0.979	0.788	0.969	0.757	≥ 146.5
pCO_2 (mmHg)	32/128	0.932	0.034	< 0.001	0.866	0.998	0.875	0.945	0.820	≥ 54.85

Cut-off values were determined according to the Youden Index (Se + Sp − 1)

Pos Number of deceased cases, *Neg* Number of discharged cases, *AUC* Area under the ROC curve, *SE* Standard error, *CI* Confidence interval, *Se* Sensitivity, *Sp* Specificity, *GCS* Glasgow Coma Scale, *SpO_2* Peripheral oxygen saturation measured by pulse oximetry (%), *LDH* Lactate dehydrogenase (U/L)

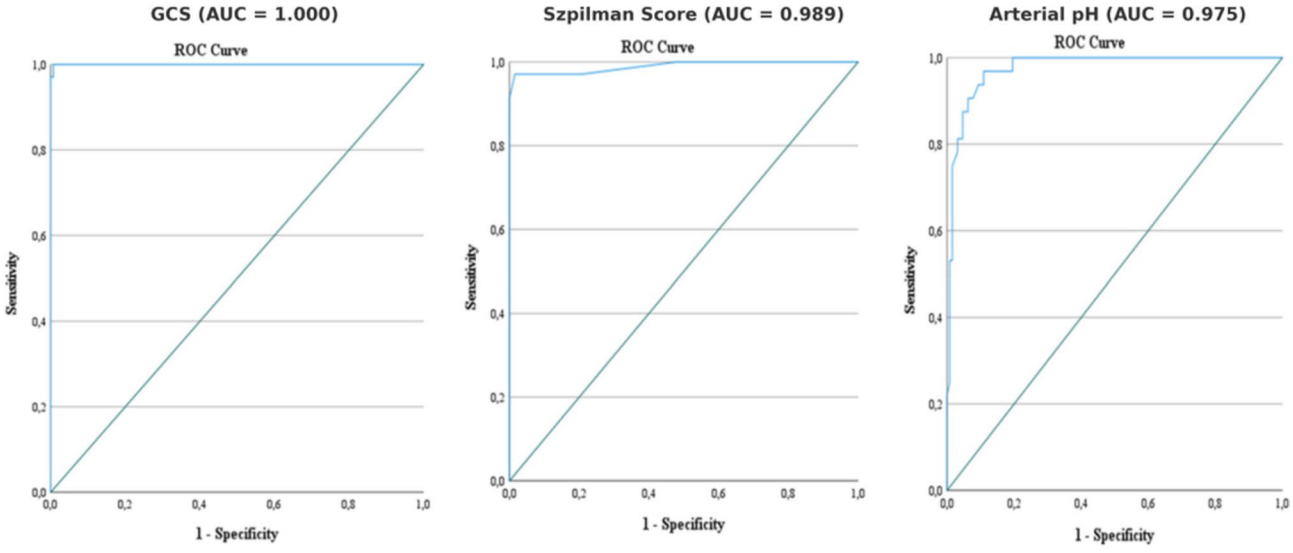

Fig. 2 ROC curves of variables demonstrating near-perfect discriminatory performance (AUC > 0.95) ROC curves of the top-performing predictors with AUC > 0.95. The Glasgow Coma Scale (AUC = 1.000), Szpilman score (AUC = 0.989), and arterial pH (AUC = 0.975) demonstrated outstanding discriminatory ability for in-hospital mortality. Each curve was derived from original ROC analysis using nonparametric estimates. The diagonal line represents random classification (AUC = 0.5)

Lactate was excluded due to missing data in a substantial proportion of cases.

Variables with AUC < 0.90 (e.g., potassium [0.830], creatinine [0.842], HCO_3^- [0.818], LDH [0.801]) were excluded from the predictive model due to their limited discriminative utility.

Multivariable logistic regression and prognostic model development

To minimize overfitting and improve generalizability, variables were selected for multivariable analysis based on univariate discriminatory performance. Predictors with AUC values above 0.95— specifically the GCS, Szpilman score, and arterial pH—were excluded due to their near-complete separation and potential to reduce external validity [16, 17].

Binary logistic regression was conducted using SpO_2, serum sodium, and pCO_2 as candidate predictors, selected based on AUC > 0.90 and data availability. Backward likelihood ratio elimination was applied to reach the final model (Train set: $n = 91$). Only sodium and SpO_2 remained significant in the final model (Table 5).

Interpretation of the logistic regression model indicates that each 1% increase in SpO_2 was associated with an approximate 8.3% reduction in the odds of in-hospital mortality (OR = 0.917; 95% CI: 0.867–0.969). Conversely, each 1 mmol/L increase in serum sodium was associated

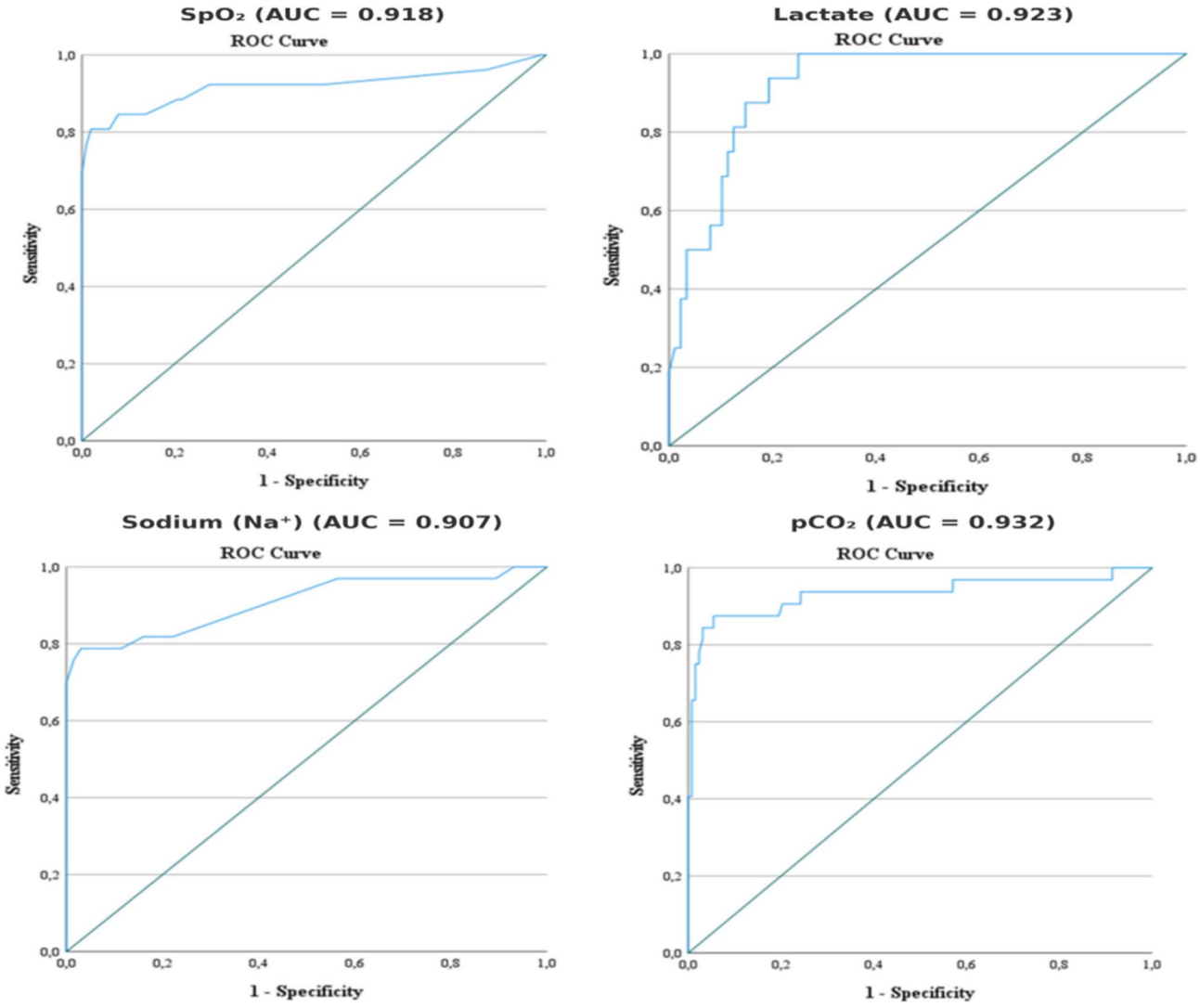

Fig. 3 ROC curves of clinical markers with strong prognostic discrimination (AUC = 0.90–0.95) Comparative ROC curves of selected clinical and biochemical markers with AUC values between 0.90 and 0.95. SpO_2 (AUC = 0.918), lactate (AUC = 0.923), serum sodium (AUC = 0.907), and pCO_2 (AUC = 0.932) demonstrated strong discriminatory ability for predicting in-hospital mortality. Each panel displays the ROC curve derived from nonparametric estimates. The diagonal line represents the line of no discrimination (AUC = 0.5)

Table 5 Multivariate logistic regression model (Training set, *n* = 91)

						95% CI for OR	
	B	**SE**	**Wald**	**p**	**OR**	**Lower**	**Upper**
SpO_2	–0,087	0,028	9,53	0,002	0,917	0,867	0,969
Na	0,702	0,253	7,68	0,006	2,018	1,228	3,316
Constant	–97,178	35,923	7,318	0,007			

Nagelkerke R Square = 0.929, Hosmer&Lemeshow test: χ^2 (df = 8) = 1.597, p = 0.991.

B Regression coefficient, *SE* Standard error, *Wald* Wald chi-square statistic, *p-value* Significance level, *OR* Odds ratio, *CI* Confidence interval, SpO_2 Peripheral oxygen saturation measured by pulse oximetry (%), *Na* Serum sodium (mmol/L)

with a 2.02-fold increase in the odds of death (OR = 2.018; 95% CI: 1.228–3.316) (Table 4).

Model performance was assessed using standard metrics, demonstrating excellent calibration (Hosmer–Lemeshow $p = 0.991$), strong discrimination (Nagelkerke R^2 = 0.929), and high overall accuracy in the training set (97.8%), consistent with established principles of logistic regression modeling (Table 5) [16, 17]. Model development and reporting adhered to established guidelines for prognostic research, including TRIPOD recommendations [18].

The final predictive equation based on Table 5 was:

Table 6 Comparative classification performance of the SNOP Model, GCS, and Szpilman scores in training and test sets[a]

		Train SetPrediction		Test setPrediction		Train+TestPrediction		GCS Prediction		Szpilman Prediction	
		DC	DE	DC	DE	DC	DE	DC	DE	DC	DE
Observed	**DC**	**69**	**1**	**32**	**0**	**101**	**1**	**131**	**1**	**129**	**2**
DE	**1**	**20**	**0**	**5**	**1**	**25**	**0**	**34**	**1**	**33**	
Performance measures											
Accuracy		0.978		1.000		0.984		0.994		0.982	
Sensitivity		0.986		1.000		0.990		0.992		0.985	
Specificity		0.952		1.000		0.962		1.000		0.971	
PPV		0.986		1.000		0.990		1.000		0.992	
NPV		0.952		1.000		0.962		0.971		0.943	
F-measure		0.986		1.000		0.990		0.996		0.989	

DC Discharged, *DE* Deceased, *PPV* Positive Predictive Value, *NPV* Negative Predictive Value

[a]The cut value is 0.500

Table 7 Comparative classification performance of the SNOP model, GCS, and Szpilman scores in training and test sets

		Train Set Prediction		Test set Prediction		Train + Test Prediction		GCS Prediction		Szpilman Prediction	
		DC	DE	DC	DE	DC	DE	DC	DE	DC	DE
Observed	**DC**	**69**	**1**	**32**	**0**	**101**	**1**	**131**	**1**	**129**	**2**
	DE	**1**	**20**	**0**	**5**	**1**	**25**	**0**	**34**	**1**	**33**
Performance measures											
Accuracy		0.978		1,000		0,984		0,994		0,982	
Sensitivity		0.986		1,000		0,990		0,992		0,985	
Specificity		0.952		1,000		0,962		1,000		0,971	
PPV		0.986		1,000		0,990		1,000		0,992	
NPV		0.952		1,000		0,962		0,971		0,943	
F-measure		0.986		1,000		0,990		0,996		0,989	

DC Discharged, *DE* Deceased, *PPV* Positive Predictive Value, *NPV* Negative Predictive Value

[a] The cut value is 0.500

$$\mathrm{P(Death)} = 1 \,/\, (1 + \mathrm{e}\hat{}(-(-97.178 + 0.702 \times \mathrm{Na} - 0.087 \times \mathrm{SpO_2})))$$

To support clinical implementation, a web-based SNOP score calculator is available at www.snopscore.com, which enables clinicians to estimate mortality risk in real time.

Additional details regarding predictor selection, regression diagnostics, and validation procedures are provided in Supplementary Appendix A.

Comparative classification performance of the SNOP model

Classification performance of the final logistic regression model was evaluated using both the training ($n = 91$) and test ($n = 39$) sets (Table 6). In the training set, the model misclassified one survivor and one non-survivor, yielding an overall accuracy of 97.8%. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and F-measure were 0.986, 0.952, 0.986, 0.952, and 0.986, respectively. In the test set, the model achieved perfect classification across all metrics.

To benchmark predictive performance, we compared the SNOP model to the Glasgow Coma Scale (GCS) and Szpilman score using the combined dataset ($n = 166$). The SNOP model achieved an accuracy of 98.4%, with sensitivity of 0.990, specificity of 0.962, PPV of 0.990, NPV of 0.962, and an F-measure of 0.990. GCS-based classification yielded slightly higher sensitivity (0.992) and perfect specificity (1.000), with an F-measure of 0.996. The Szpilman score showed slightly lower performance, with an accuracy of 98.2%, sensitivity of 0.985, specificity of 0.971, and F-measure of 0.989.

To reduce the risk of overfitting, we followed the recommended events-per-variable (EPV) criterion during model development. With 21 events (deaths) and two predictors (SpO_2 and serum sodium), the resulting EPV was 10.5—meeting the conventional minimum threshold of 10 for logistic regression modeling [18].

Discussion

Drowning remains a major cause of injury-related mortality worldwide, yet risk stratification at hospital admission remains poorly defined [1, 19]. Although survival has improved in many high-resource settings [20], early prognostic assessment in the emergency department (ED) is often limited [21]. Existing tools such as the Szpilman classification offer valuable prehospital guidance but

lack integration of objective laboratory parameters relevant to in-hospital care.

This study sought to address this gap by developing a simple, ED-based prognostic model incorporating clinical and biochemical markers. Prior studies have emphasized factors such as altered consciousness, respiratory failure, and acid-base imbalance as key prognostic indicators [10–12]. However, few models have combined these with objective laboratory values into a structured, reproducible framework for early risk assessment [14, 15].

Our cohort mirrored known epidemiological trends, with male predominance and seasonal clustering linked to recreational seawater exposure [1, 7, 22]. The wide age range (2–77 years) underscores the need for age-independent triage strategies. In line with previous findings [10–12, 23], non-survivors were more likely to have undergone CPR or intubation and presented with lower GCS, lower pH, and higher sodium and lactate levels—reflecting greater physiological compromise.

Although the SNOP score was not specifically designed for resuscitated patients, nearly all non-survivors in our cohort had undergone CPR or intubation. This likely reflects the clinical severity of fatal cases rather than selection bias. The score's predictive utility may therefore be strongest in patients with significant physiological derangement.

ROC analysis confirmed that several parameters—GCS, Szpilman score, and pH—had excellent predictive value (AUC > 0.97), consistent with their role in triage [8, 9, 12, 14]. However, their near-perfect accuracy increases the risk of overfitting in multivariable models. Following the principle of parsimony and current modeling guidelines [16, 17], we excluded these variables in favor of others with strong yet independent predictive capacity. GCS also lacks discriminatory value during ongoing CPR, where it is uniformly scored as 3.

All cases in our study involved seawater submersion along Black Sea coast of Turkey. This setting contributed to elevated serum sodium levels due to aspiration of hypertonic fluid, which drives osmotic shifts, volume depletion, and cerebral dehydration [2, 7, 24, 25]. Accordingly, hypernatremia has been linked to poorer outcomes in saltwater drownings [9, 25, 26]. In particular, the inclusion of serum sodium as a predictor reflects the specific pathophysiology of seawater drowning, which predominated in our cohort, and may limit the applicability of the SNOP score to freshwater or non-marine drowning scenarios.

SpO_2 was significantly lower in non-survivors, underscoring the role of hypoxia in drowning-related mortality. Saltwater aspiration disrupts alveolar-capillary integrity, leading to pulmonary edema, surfactant loss, and impaired gas exchange [11, 19]. SpO_2 thus serves as a non-invasive marker of pulmonary injury and systemic hypoxia, both critical to early outcomes [10, 13].

Serum sodium levels and SpO_2 emerged as the two strongest independent predictors of in-hospital mortality and were used to construct the SNOP score (Saturation and Natremia-based Outcome Predictor). The model demonstrated excellent performance (accuracy = 97.8%) and uses routinely available parameters, supporting its feasibility for ED implementation.

While Szpilman staging and GCS remain important, their limitations in in-hospital contexts highlight the value of complementary tools. The SNOP score adds an ED-specific perspective, using objective inputs for early risk stratification. In addition to mortality prediction, it may also assist in identifying low-risk patients suitable for short-term observation rather than hospital admission, potentially alleviating ED and ICU burden. This could help streamline triage decisions and optimize ED and ICU resource utilization, particularly in high-volume settings.

It should be noted that the model is designed to complement—rather than replace—existing tools. While Szpilman's staging remains highly relevant in prehospital triage, it does not account for post-admission laboratory data or dynamic physiological deterioration.

In summary, the SNOP score provides a practical, objective, and ED-specific approach for early mortality risk stratification following seawater drowning. Its simplicity and reliance on readily available parameters support real-world applicability, particularly in resource-limited or high-volume settings. Although the model demonstrated excellent internal performance, external validation across diverse geographic and environmental contexts—including freshwater submersion—is essential to confirm its broader utility. Additionally, while the SNOP score shows strong discriminatory power, it currently functions as a logistic regression–based tool rather than a simplified point-based score. Future studies should focus on deriving an integer-based version suitable for bedside use. Prospective research is also needed to evaluate its integration into clinical workflows and its potential impact on triage decisions and resource allocation. As drowning remains a time-critical emergency with unpredictable outcomes, tools like the SNOP score may help translate early physiological data into timely, evidence-informed care.

Conclusion

This study introduces the SNOP score, a simple, objective, and accurate two-parameter model designed for early prediction of in-hospital mortality in drowning patients. By leveraging universally available clinical variables—SpO_2 and serum Na^+—this prognostic tool provides a rapid and reproducible means of supporting

emergency physicians in making timely triage decisions, anticipating intensive care needs, and identifying high-risk cases. While established tools such as the Glasgow Coma Scale and the Szpilman classification remain valuable in specific clinical contexts, the SNOP score offers a complementary, evidence-based approach tailored to early hospital care. Future prospective multicenter validation and integration into electronic clinical decision support systems will be essential steps toward confirming its broader clinical utility.

Limitations

This study has several limitations. Its retrospective, single-center design may limit the generalizability of findings to broader patient populations, and the sample size—although larger than in many previous studies—may not fully capture the variability of drowning scenarios across different age groups, environments, and water types. The availability of certain laboratory parameters, such as lactate, was inconsistent due to historical differences in analyzer technology, potentially limiting the inclusion of additional relevant predictors. Moreover, the absence of reliable documentation on submersion duration prevented its inclusion in the analysis, despite its known prognostic significance. Additionally, 24 patients were excluded due to missing critical data, including outcome or key laboratory parameters. While these exclusions were necessary for valid statistical modeling, we acknowledge that some of the excluded cases may have represented the most severe presentations (e.g., dead-on-arrival), which could have influenced the overall risk profile of the study cohort. Finally, while the exclusion of highly discriminative variables like GCS and arterial pH was methodologically justified to avoid overfitting, this choice may have influenced the model's performance in other clinical contexts. Future multicenter, prospective studies with external validation cohorts are needed to refine and further confirm the predictive strength of the SNOP score.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12245-025-00977-2.

Supplementary Material 1.

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Authors' contributions

K.Ö. (corresponding author) and Ö.O. contributed to all aspects of the study, including study design, data acquisition, analysis, interpretation, and manuscript preparation. Ş.Ş.K. was involved in data collection and provided substantial input in analysis, interpretation, and critical revision of the manuscript. A.P. contributed to the conceptual design, statistical modeling, and software development related to the SNOP score. T.E. contributed to all phases of the study, including conceptualization, methodology, data interpretation, and final manuscript approval. All authors reviewed and approved the final version of the manuscript and agreed to be personally accountable for their own contributions and the integrity of the entire work.

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Data availability

Yes, I have research data to declare.The data that support the findings of this study are not publicly available due to privacy and ethical restrictions. Anonymized data may be made available from the corresponding author (K.Ö.) upon reasonable request and with approval from the relevant institutional review board.Data AvailabilityDetailed statistical analyses related to the predictive modeling of in-hospital mortality—based on the study dataset—are provided as Supplementary Appendix A in Excel format to ensure academic transparency.The experimental risk prediction model derived from our logistic regression analysis is also available in a web-based calculator format at www.snopscore.com. This online tool is intended for academic purposes only and is based exclusively on the dataset and findings presented in this manuscript.The website and its contents are copyrighted by the authors and serve as a prototype implementation of the SNOP score. No clinical or legal responsibility is assumed for its use in patient management without further validation.

Declarations

Ethics approval and consent to participate

Ethical approval was obtained from the Sinop University Ethics Committee for Human Research (Approval No: 2025/113, Date: February 28, 2025). The study was conducted in accordance with the principles of the Declaration of Helsinki. Informed consent was waived due to the retrospective design and anonymized nature of the data.

Consent for publication

Not applicable. This manuscript does not contain any individual person's data in any form (including images or videos).

Competing interests

The authors declare no competing interests.

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