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Base deficit and triage, not blood ethanol, predict poor outcome after acute alcohol ingestion: a 10-year cohort study

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Abstract

Background

Clinicians often anchor on the blood ethanol concentration when judging the severity of acute alcohol ingestion, yet its prognostic value is uncertain. We compared the blood ethanol concentration with routinely available markers of physiological derangement for predicting a poor outcome in emergency department (ED) patients with acute alcohol ingestion.

Methods

This single-centre retrospective cohort study included adults (≥ 18 years) presenting to a tertiary ED between September 2014 and September 2024 with a blood ethanol concentration > 10.1 mg/dL; co-ingestions and toxic-alcohol exposures were excluded. The primary outcome was a composite of intensive care unit (ICU) admission and/or all-cause mortality at 30 days or 6 months. Discrimination was assessed with the area under the receiver-operating-characteristic curve (AUC); independent predictors were identified by multivariable logistic regression with bootstrap internal validation. Analyses were repeated after stratification by presentation type (trauma versus non-trauma).

Results

Of 2,460 patients (85.4% male; median age 35 years), 90 (3.7%) had a poor outcome. Blood ethanol did not discriminate poor outcome overall (AUC 0.527), whereas base deficit (0.783), bicarbonate (0.768), lactate (0.730) and admission glucose (0.690) discriminated moderately to well. Base deficit performed similarly in non-trauma (0.790) and trauma (0.770) strata. Among non-trauma presentations, patients with a poor outcome had a lower median ethanol concentration (129.9 versus 185.2 mg/dL; p = 0.021). A model combining red triage, base deficit and pathological computed tomography achieved an optimism-corrected AUC of 0.874.

Conclusions

In ED patients with detectable ethanol, the ethanol concentration is a poor predictor of poor outcome, whereas base deficit, lactate, admission glucose and red triage are stronger, readily available markers. Risk stratification should rest on physiological and structural assessment rather than the toxicological level.

Background

Alcohol is among the most common psychoactive exposures managed in the emergency department (ED) and a leading contributor to preventable morbidity and mortality worldwide.[1, 2] Alcohol-related ED

visits have risen over the past two decades and consume a disproportionate share of resources, particularly at night and on weekends.[3–5]

Acute intoxication spans a wide clinical spectrum and can mask or coexist with life-threatening conditions such as traumatic brain injury, occult haemorrhage and metabolic derangement; alcohol also confounds the neurological examination and the Glasgow Coma Scale.[6–8] Because tolerance uncouples the blood ethanol concentration from clinical severity in chronic users, the concentration alone is an unreliable basis for disposition.[9–11]

Contemporary guidance recommends grounding management in physiology rather than in the ethanol level.[9] Klein and colleagues showed that unsuspected critical illness is common among ED patients presenting for apparently simple intoxication.[12] In trauma cohorts, base deficit and lactate retain prognostic value despite the presence of ethanol,[13, 14] and lactate predicts short-term mortality in ethanol-positive ED patients.[15] However, few studies directly compare the discriminative ability of the ethanol concentration with that of routinely available metabolic markers for a patient-centred composite outcome, and long-term data from consecutive, unselected cohorts remain scarce.[16]

We therefore analysed a 10-year cohort of ED patients with acute alcohol ingestion to compare the discrimination of the blood ethanol concentration and markers of physiological derangement for a composite poor outcome, to derive a parsimonious model of independent predictors with internal validation, and to examine whether the findings were consistent across trauma and non-trauma presentations.

Methods

Study design and setting

This single-centre, retrospective cohort study was conducted in the ED of Düzce University Faculty of Medicine (Training and Research Hospital), a tertiary referral centre, and covered a 10-year period from 15 September 2014 to 15 September 2024. The study was approved by the Düzce University Non-interventional Health Research Ethics Committee (approval no. 2024/188), which waived the requirement for informed consent. Reporting follows the STROBE statement.[17]

Participants

We screened all patients aged ≥ 18 years who presented with any complaint and had a blood ethanol concentration measured during the study period. Using a consecutive-case approach, we included patients with a blood ethanol concentration > 10.1 mg/dL. We excluded patients younger than 18 years, those with recreational co-ingestion of other psychoactive substances, those with toxic-alcohol exposure (methanol, ethylene glycol, isopropanol) and those with incomplete clinical records. Of 16,138 screened patients, 13,275 were excluded for an ethanol concentration ≤ 10.1 mg/dL, leaving 2,863 eligible patients;

a further 403 were excluded (351 with incomplete records and 52 with co-ingestion or toxic-alcohol exposure), yielding a final cohort of 2,460 patients (Fig. 1). No formal sample-size calculation was performed; the study size was determined by all consecutive eligible patients during the study period.

Variables and outcomes

Data were extracted from the hospital information system and laboratory and radiology records: demographics; presentation characteristics; triage code; presenting-complaint category; laboratory and blood-gas parameters (ethanol, glucose, pH, lactate, bicarbonate and base excess/deficit); cranial computed tomography (CT); and disposition. Because blood gas was obtained at the discretion of the treating clinician rather than routinely, pH, lactate, bicarbonate and base deficit were available in a subgroup (valid n ≈ 620–630) enriched for higher-acuity patients, and the valid n is reported for each analysis.

Definitions

Triage. Triage was performed at presentation, before registration, in accordance with the three-colour coding system mandated by the Turkish Ministry of Health for emergency departments, which prioritises patients as red, yellow or green according to their need for examination, investigation, and medical or surgical intervention. In keeping with national practice, no fixed physiological cut-off values were applied; the category was assigned by clinical judgement following current guidelines. Walk-in patients were triaged by a nurse, whereas patients arriving by 112 emergency ambulance were received and categorised by a physician; when the ambulance team reported an obviously critical patient, the patient was taken directly to the red area. Red denoted a patient requiring immediate life-saving intervention, yellow a potentially serious but stable condition, and green a minor, non-urgent complaint.

Trauma versus non-trauma. Patients were classified as trauma or non-trauma presentations on the basis of the recorded chief complaint (reason for presentation) at admission, not on the discharge diagnosis. Presentations attributable to physical injury — including road-traffic collisions, falls, assault, and other blunt or penetrating trauma — were classified as trauma; all other presentations (for example, nausea and vomiting, psychiatric complaints, cardiac symptoms and non-specific medical complaints) were classified as non-trauma.

Poor outcome. The primary outcome, poor outcome, was defined a priori as a composite of (1) admission to the intensive care unit (ICU) during the index presentation and/or (2) all-cause mortality at 30 days or 6 months; a patient meeting either criterion was classified as having a poor outcome. Of the 2,460 patients, 90 (3.7%) met this definition.

Pathological CT. A cranial CT was defined a priori as pathological when it showed an acute, clinically significant abnormality expected to influence management (for example, intracranial haemorrhage,

cerebral contusion, or skull fracture). Patients who did not undergo CT were excluded from CT-based analyses.

Statistical analysis

Continuous variables were non-normally distributed (Shapiro–Wilk test) and are presented as median (interquartile range [IQR]) and compared with the Mann–Whitney U or Kruskal–Wallis test; categorical variables are presented as n (%) and compared with the Pearson χ^2 or Fisher exact test. Receiver-operating-characteristic (ROC) analysis was performed for continuous predictors, with cut-offs by the Youden index; the AUC (95% confidence interval [CI]), sensitivity, specificity and odds ratio (OR) are reported. Independent predictors were identified with multivariable binary logistic regression; model selection balanced discrimination, parsimony (Akaike/Bayesian information criteria) and the likelihood-ratio test. Because the model was developed in a subgroup with complete blood-gas and CT data, bootstrap internal validation (1,000 replications) was used to quantify optimism and obtain an optimism-corrected AUC and calibration slope. All principal analyses were repeated after stratification by presentation type (trauma versus non-trauma). Missing data were not imputed; each analysis was performed on the patients with complete data for the variables concerned, and the valid n is reported. Analyses were performed in IBM SPSS Statistics 26, jamovi 2.3 and Python 3.12; a two-sided $p < 0.05$ was considered significant.

Results

Of 2,460 patients, 85.4% were male and the median age was 35 years (IQR 25–46). Most patients were triaged yellow (85.2%) or red (14.3%), and the median blood ethanol concentration was 172.8 mg/dL (IQR 104.6–237.5). Trauma-related complaints accounted for 62.0% of presentations. Overall, 84.9% were discharged without admission, 12.0% were admitted to a ward and 3.1% to the ICU; 30-day and 6-month all-cause mortality were 0.8% and 1.1%, respectively (Table 1).

Table 1
Baseline characteristics of the cohort, overall and by poor outcome

Characteristic	Overall (n = 2,460)	No poor outcome (n = 2,370)	Poor outcome (n = 90)	p
Age, years — median (IQR)	35 (25–46)	35 (25–46)	36 (25–47)	0.432
Male sex — n (%)	2,101 (85.4)	2,025 (85.4)	76 (84.4)	0.911
Red triage — n (%)	353 (14.3)	300 (12.7)	53 (58.9)	< 0.001
Ambulance arrival — n (%)	499 (20.3)	473 (20.0)	26 (28.9)	0.053
Blood ethanol, mg/dL	172.8 (104.6–237.5)	173.8 (104.9–237.7)	149.0 (80.9–237.2)	0.391
Admission glucose, mg/dL	106 (95–124)	106 (95–122)	132 (106–161)	< 0.001
pH	7.4 (7.3–7.4)	7.4 (7.3–7.4)	7.3 (7.2–7.4)	< 0.001
Lactate, mmol/L	2.6 (1.9–3.6)	2.5 (1.9–3.4)	3.8 (2.7–7.3)	< 0.001
Bicarbonate, mmol/L	22.6 (21.0–23.8)	22.8 (21.3–23.9)	19.4 (15.8–22.2)	< 0.001
Base deficit, mmol/L	1.3 (− 0.5–3.6)	1.2 (− 0.7–3.3)	5.8 (2.7–10.3)	< 0.001
Pathological CT — %	30.8	28.5	78.3	< 0.001
ICU admission — n (%)	77 (3.1)	0 (0)	77 (85.6)	—
30-day mortality — n (%)	20 (0.8)	0 (0)	20 (22.2)	—
6-month mortality — n (%)	26 (1.1)	0 (0)	26 (28.9)	—

Data are median (IQR) or n (%). CT computed tomography, ICU intensive care unit, IQR interquartile range. n = 2,120. Blood gas obtained at clinician discretion (valid n: pH 630, lactate 626, bicarbonate 620, base deficit 628). CT available in n = 1,276. Components of the composite outcome; not tested statistically.

A poor outcome occurred in 90 patients (3.7%). In the whole cohort the blood ethanol concentration did not differ between patients with and without a poor outcome (median 149.0 versus 173.8 mg/dL; p = 0.391). The poor-outcome group had a markedly higher frequency of red triage (58.9% versus 12.7%), higher admission glucose (132 versus 106 mg/dL) and more deranged blood gas (all p < 0.001), and far more often a pathological CT (78.3% versus 28.5%; p < 0.001) (Table 1).

The blood ethanol concentration discriminated poor outcome no better than chance (AUC 0.527, 95% CI 0.461–0.592), as did age (0.524). Base deficit discriminated best (AUC 0.783), followed by bicarbonate (0.768), lactate (0.730), admission glucose (0.690) and pH (0.688) (Table 2; Fig. 2A). Red triage (OR 9.88,

95% CI 6.38–15.30) and pathological CT (OR 9.09, 95% CI 4.86–17.01) were strongly associated with poor outcome.

Table 2
Discrimination and diagnostic performance of continuous predictors for poor outcome

Predictor	n	AUC (95% CI)	Cut-off	Sens%	Spec%	OR (95% CI)	p
Base deficit, mmol/L	628	0.783 (0.706–0.860)	≥ 2.9	75.0	72.2	7.58 (3.89–14.78)	< 0.001
Bicarbonate, mmol/L	620	0.768 (0.684–0.852)	≤ 20.0	60.4	86.7	9.82 (5.28–18.26)	< 0.001
Lactate, mmol/L	626	0.730 (0.646–0.813)	≥ 3.30	66.7	72.5	5.18 (2.79–9.63)	< 0.001
Admission glucose, mg/dL	2,120	0.690 (0.625–0.756)	≥ 119	63.5	71.2	4.28 (2.73–6.70)	< 0.001
pH	630	0.688 (0.595–0.781)	≤ 7.30	45.8	91.2	8.76 (4.66–16.46)	< 0.001
Blood ethanol, mg/dL	2,460	0.527 (0.461–0.592)	≤ 145.7	50.0	61.8	1.62 (1.06–2.46)	0.025
Age, years	2,460	0.524 (0.461–0.588)	≥ 42	44.4	65.9	1.55 (1.02–2.37)	0.041

Cut-offs by the Youden index. AUC area under the ROC curve, CI confidence interval, OR odds ratio, Sens sensitivity, Spec specificity.

In the final multivariable model, developed in 268 patients with complete triage, blood-gas and CT data (33 events), red triage (OR 7.31, 95% CI 2.77–19.30), an increasing base deficit (OR 1.25 per mmol/L, 95% CI 1.13–1.39) and pathological CT (OR 2.91, 95% CI 1.11–7.66) were independent predictors (Table 3). The model discriminated well (apparent AUC 0.883; Nagelkerke R^2 = 0.411) (Fig. 2B). Bootstrap internal validation showed minimal optimism (0.009), yielding an optimism-corrected AUC of 0.874 (95% CI 0.820–0.934) and a calibration slope of 0.94.

Table 3
Multivariable logistic regression: independent predictors of poor outcome

Predictor	β	SE	Wald χ^2	p	OR (95% CI)
Constant	−4.147	0.493	70.80	< 0.001	—
Red triage	1.990	0.495	16.14	< 0.001	7.31 (2.77–19.30)
Base deficit (per mmol/L)	0.227	0.053	18.57	< 0.001	1.25 (1.13–1.39)
Pathological CT	1.068	0.494	4.68	0.031	2.91 (1.11–7.66)

n = 268 patients with complete data (33 events). Apparent AUC 0.883; optimism-corrected AUC 0.874 (95% CI 0.820–0.934) after 1,000 bootstrap replications; calibration slope 0.94; Nagelkerke R^2 0.411; likelihood-ratio $\chi^2 = 65.3$, df = 3, p < 0.001. CI confidence interval, CT computed tomography, OR odds ratio, SE standard error.

Because the cohort was assembled on the basis of a measured ethanol concentration rather than a presentation of suspected isolated intoxication, we examined whether the findings reflected the acuity of the underlying presentation. Base deficit discriminated poor outcome almost identically in non-trauma (AUC 0.790) and trauma (0.770) presentations, as did lactate and bicarbonate (Table 4). However, the ethanol concentration behaved differently between strata: among trauma presentations it was unrelated to outcome (p = 0.443), whereas among non-trauma presentations patients with a poor outcome had a significantly lower median ethanol concentration (129.9 versus 185.2 mg/dL; p = 0.021), a difference absent in the pooled cohort (Table 4).

Table 4
Analyses stratified by presentation type (trauma versus non-trauma)

Poor outcome — n (%)	**Non-trauma (n = 935)**	**Trauma (n = 1,525)**
	29 (3.1)	**61 (4.0)**
Ethanol, poor outcome (mg/dL)	129.9 (52.8–227.5)	165.0 (114.3–240.1)
Ethanol, no poor outcome (mg/dL)	185.2 (123.6–251.1)	168.1 (94.2–229.1)
p (Mann–Whitney)	0.021	0.443
AUC — blood ethanol	0.626	0.471
AUC — base deficit	0.790 (n = 389)	0.770 (n = 239)
AUC — bicarbonate	0.792 (n = 382)	0.746 (n = 238)
AUC — lactate	0.718 (n = 388)	0.705 (n = 238)

Values for ethanol are median (IQR). Events: 17 (non-trauma) and 31 (trauma) in the blood-gas subgroups. AUC area under the ROC curve.

Discussion

In this 10-year cohort of more than 2,400 ED patients with acute alcohol ingestion, the blood ethanol concentration did not discriminate poor outcome, whereas readily available markers of physiological derangement did. Base deficit was the best single discriminator (AUC 0.78), and a parsimonious model combining red triage, base deficit and pathological CT retained good discrimination after internal validation (optimism-corrected AUC 0.87). These findings support a shift in bedside reasoning from the toxicological level towards physiology.

A central concern in a cohort assembled on the basis of a measured ethanol concentration is that the observed associations simply reflect the acuity of the underlying presentation: injured or critically ill patients are both more likely to have a blood gas drawn and more likely to have a poor outcome. Two observations argue against this being the whole explanation. First, base deficit, lactate and bicarbonate discriminated outcome to a similar degree in non-trauma and trauma presentations, so their prognostic value is not merely a proxy for injury severity. Second, the relationship between ethanol and outcome differed between strata in a clinically meaningful direction. Among non-trauma presentations — those most closely resembling apparently isolated intoxication — patients with a poor outcome had a lower ethanol concentration, whereas no such relationship existed among trauma presentations; pooling the two strata cancelled this signal.

This stratified finding is the clinically actionable one. When a patient presents obtunded and apparently intoxicated, a concentration lower than expected for the observed degree of depression should raise rather than lower concern, because the mental state is then insufficiently explained by alcohol. This is a quantitative expression of the observation of Klein et al. that critical illness is frequently unsuspected in this population,[12] and it is consistent with evidence that tolerance uncouples the concentration from severity[9, 10] and that the ethanol concentration neither excludes nor predicts intracranial pathology.[6, 7] Dilution of the measured concentration by resuscitation fluids may contribute in shocked patients.[18] A high ethanol concentration should never be accepted as the explanation for depressed consciousness, and a low one should not be reassuring.

Base deficit, bicarbonate and lactate all outperformed ethanol, mirroring trauma data in which these markers retain prognostic value despite the presence of ethanol[13, 14] and ED data in which lactate predicts mortality in ethanol-positive patients.[15] In our patients these markers likely reflect tissue hypoperfusion and the summed metabolic burden of occult injury or critical illness. Although alcohol predisposes to hypoglycaemia, the poor-outcome group was instead hyperglycaemic (median 132 mg/dL), likely reflecting a catecholamine-driven stress response that should prompt evaluation for major trauma or sepsis. Red triage — the systematised form of the clinician’s initial gestalt — was the strongest categorical predictor and remained independent in the model, underscoring that a high-acuity designation should override the “just intoxicated” framing.[19]

Limitations

Several limitations warrant emphasis. First, and most importantly, the cohort was defined by the presence of a measured blood ethanol concentration rather than by a presentation of suspected isolated intoxication; the decision to measure ethanol is clinician-dependent, so the cohort is enriched for patients in whom something was already thought to be wrong, and 62% presented after trauma. The stratified analyses mitigate but do not eliminate this concern, and residual confounding cannot be excluded. Our findings apply to ED patients with detectable ethanol, not to a prospectively defined population with apparent lone intoxication. Second, blood gas and CT were obtained at clinician discretion, so the metabolic-marker and multivariable analyses derive from a higher-acuity subgroup; the model remains internally rather than externally validated. Third, the composite outcome combines ICU admission with mortality, and the triage–ICU association is partly circular. Fourth, the non-trauma stratum contained only 29 events. Finally, this was a single-centre study, limiting generalisability.

Conclusions

In ED patients with detectable blood ethanol, the ethanol concentration is a poor predictor of poor outcome, whereas base deficit, lactate, admission glucose, red triage and pathological CT are stronger and readily available markers whose performance is consistent across trauma and non-trauma presentations. Risk stratification in this population should be driven by physiological and structural assessment rather than by the toxicological level; prospective, multicentre validation is warranted.

Abbreviations

AUC
area under the receiver-operating-characteristic curve
CI
confidence interval
CT
computed tomography
ED
emergency department
ICU
intensive care unit
IQR
interquartile range
OR
odds ratio
ROC
receiver-operating characteristic
SE
standard error
STROBE

Strengthening the Reporting of Observational Studies in Epidemiology.

Declarations

Ethics approval and consent to participate:

The study was approved by the Düzce University Non-interventional Health Research Ethics Committee (16 September 2024; approval no. 2024/188). All methods were carried out in accordance with relevant guidelines and regulations and with the Declaration of Helsinki. The requirement for informed consent was waived by the Düzce University Non-interventional Health Research Ethics Committee owing to the retrospective use of anonymised, routinely collected data.

Consent for publication:

Not applicable.

Clinical trial number

Not applicable.

Competing interests:

The authors declare that they have no financial or non-financial competing interests.

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Author Contribution

Y.H.Y. conceived and designed the study, developed the methodology, collected and curated the data, performed the statistical analysis, interpreted the data, prepared Figures 1–2 and Tables 1–4, and wrote the main manuscript text. S.K. conceived and supervised the study, contributed to the methodology and the interpretation of the data, and critically revised the manuscript for important intellectual content. Both authors reviewed and approved the final manuscript and agree to be accountable for all aspects of the work.

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Use of large language models:

A large-language-model tool was used for language editing and formatting only. No content was generated autonomously; the authors reviewed all text and take full responsibility for the content of the publication.

Data Availability

The datasets analysed during the current study were derived from the hospital information system of Düzce University Faculty of Medicine. They are not publicly available because they contain potentially identifying patient information, but are available from the corresponding author on reasonable request.

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Figures

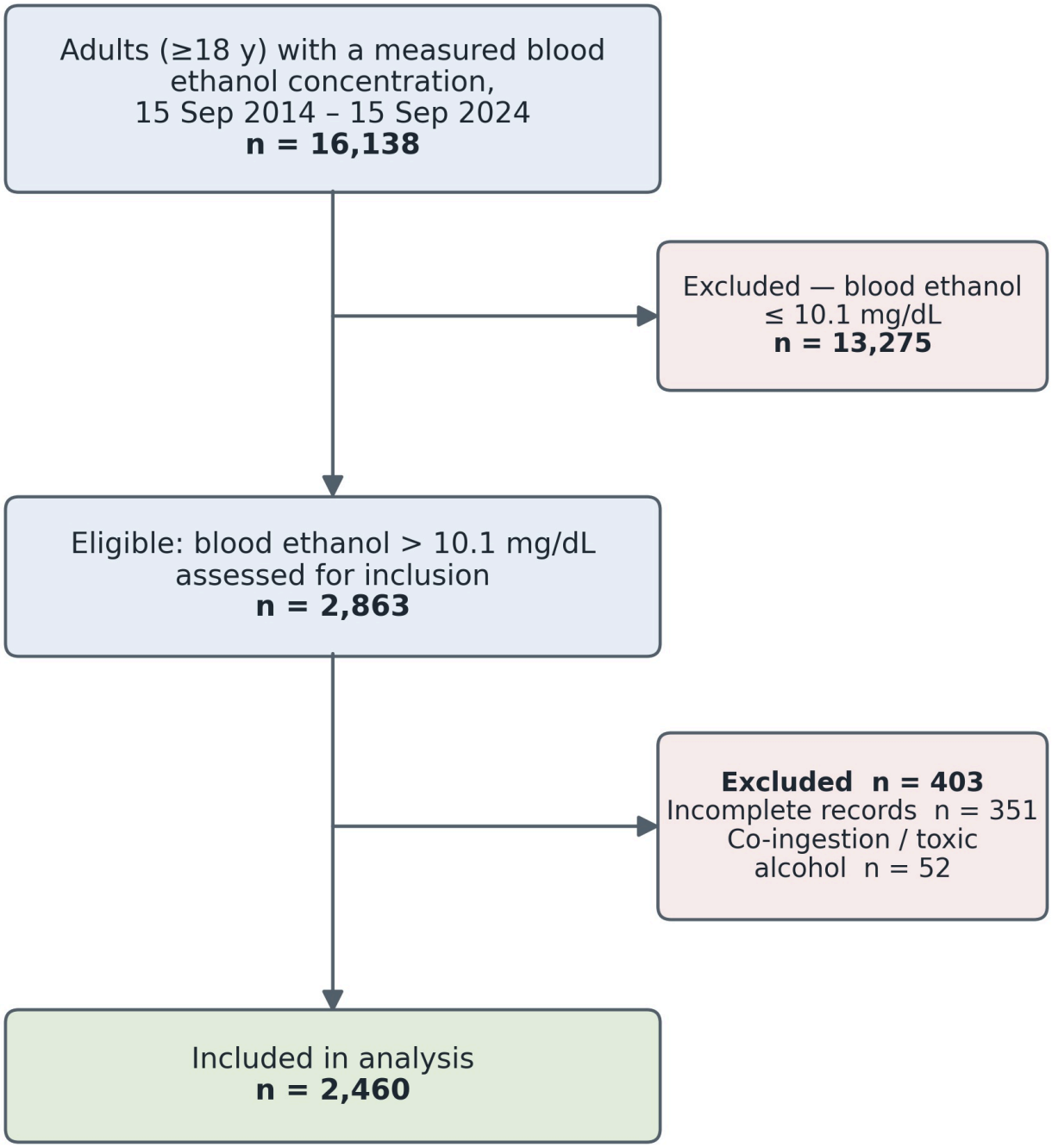


Figure 1

Participant flow diagram. Of 16,138 patients with a measured blood ethanol concentration, 13,275 were excluded for a concentration ≤ 10.1 mg/dL; of 2,863 eligible patients, 403 were excluded (351 with incomplete records and 52 with co-ingestion or toxic-alcohol exposure), leaving 2,460 patients for analysis.

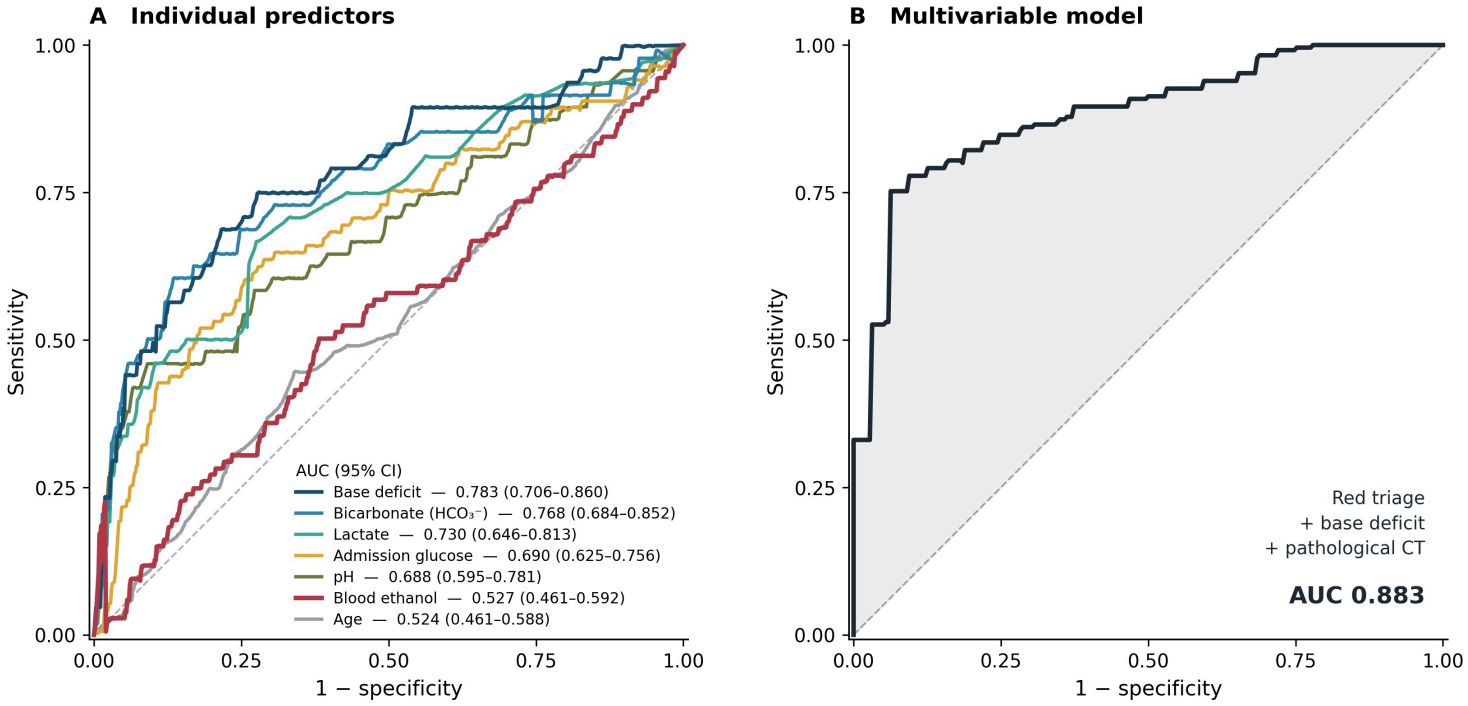


Figure 2

Receiver-operating-characteristic curves for poor outcome. (A) Individual predictors: the blood ethanol concentration (AUC 0.527) and age (0.524) track the line of no discrimination, whereas base deficit (0.783), bicarbonate (0.768) and lactate (0.730) discriminate well. (B) The multivariable model combining red triage, base deficit and pathological computed tomography (apparent AUC 0.883; optimism-corrected 0.874).