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Prognostic Value of Bioelectrical Impedance Analysis Markers in Predicting 28- Day Mortality in Chinese ICU Patients

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Abstract

Bioelectrical impedance analysis (BIA) provides bedside measures of skeletal muscle and fluid distribution, but the joint prognostic value of these measures in critically ill adults remains uncertain. This prospective single-centre cohort included 461 adults, using each patient's earliest eligible ICU admission. Multifrequency BIA was performed within 8 h, and binary day-28 mortality was analysed by logistic regression. Ninety-two patients (20.0%) died by day 28. In the extended APACHE II + BIA model, higher skeletal muscle index (odds ratio [OR] 0.698 per kg/m², 95% CI 0.584–0.833) and phase angle (OR 0.604 per degree, 95% CI 0.473–0.772) were associated with lower mortality, whereas higher extracellular water/total body water ratio (OR 3.169 per 0.05, 95% CI 2.074–4.841) and APACHE II (OR 3.634 per 5 points, 95% CI 2.505–5.270) were associated with higher mortality. The extended model achieved an apparent AUC of 0.900 (95% CI 0.864–0.937) and a bootstrap optimism-adjusted AUC of 0.896. These BIA measures provided complementary prognostic information in the development cohort. External validation is required before clinical use.

Introduction

Critical illness triggers intense catabolism, systemic inflammation, and profound disturbances of body-water distribution, so skeletal muscle is lost rapidly during an ICU stay and malnutrition develops within days [1, 2, 3]. Both malnutrition and reduced muscle mass have been linked to higher mortality, longer mechanical ventilation, and prolonged ICU stays, making early assessment of nutritional status and body composition a priority for risk stratification [4, 5, 6]. Reference techniques for quantifying muscle mass—computed tomography (CT) at the third lumbar vertebra and dual-energy X-ray absorptiometry (DXA)—cannot be applied routinely in the ICU: CT involves radiation exposure and patient transport, and DXA equipment is stationary and unsuitable for bedridden patients [7, 8, 9]. A portable, non-invasive bedside method that can be repeated over time is therefore needed for body-composition monitoring in critical care.

Multifrequency BIA meets these requirements: it is rapid, inexpensive, radiation-free, and yields a whole-body composition estimate within 3–5 minutes [10, 11]. Among the parameters it provides, three are of particular clinical interest: SMI, a BIA-derived algorithmic estimate related to muscle mass that can be influenced by hydration; PhA, a marker of cell-membrane integrity; and ECW/TBW, an index of extracellular fluid accumulation [9, 12, 13, 14]. Because edema and rapid fluid shifts accompany critical illness—early fluid resuscitation can expand the extracellular compartment and distort BIA-derived indices, particularly SMI and ECW/TBW—these measurements warrant cautious interpretation in hemodynamically unstable or fluid-overloaded patients.

Most previous ICU studies have evaluated individual BIA markers in relation to mortality [15, 16], and few have integrated several BIA parameters into composite prediction models for 28-day death. Published BIA cut-offs vary across populations, and thresholds derived in a single development cohort cannot establish ethnicity-specific clinical cut-offs.

The aims of this prospective cohort analysis were to (1) quantify the associations of BIA-derived SMI, PhA, and ECW/TBW with 28-day all-cause mortality and (2) develop a BIA-based prediction model, evaluate the added prognostic value of combining BIA markers with APACHE II, and internally validate the extended model. Reporting follows the STROBE [17] and TRIPOD [18] statements. All performance estimates reflect the development cohort; the study does not present a model ready for clinical implementation.

Results

Study Population and Baseline Characteristics

Of 712 screened ICU admission records, 69 did not meet the eligibility criteria (incomplete BIA data, n = 23; loss to follow-up, n = 18; terminal malignancy, n = 15; pacemaker implantation, n = 13). The remaining 643 eligible admission records represented 461 unique patients. To ensure one observation per patient, 182 subsequent eligible admissions from patients already represented in the cohort were excluded, and the earliest eligible admission was retained. The final analysis therefore included 461 unique patients (369 survivors and 92 non-survivors), with a day-28 all-cause mortality of 20.0%. Baseline characteristics are summarized in Table 1.

Non-survivors were older (median 72 [IQR 68–76] vs. 66 [60–73] years) and more severely ill (APACHE II 19.82 ± 5.49 vs. 13.98 ± 4.37). They also had lower SMI (median 8.77 [IQR 7.59–9.74] vs. 10.19 [8.91–11.82] kg/m²), lower PhA (3.80 ± 1.31 vs. 4.90 ± 1.30 degrees), and higher ECW/TBW (median 0.43 [IQR 0.40–0.45] vs. 0.38 [0.36–0.41]). BMI was lower among non-survivors (23.55 ± 4.16 vs. 25.16 ± 4.51 kg/m²), whereas chronic kidney disease and chronic heart failure were more frequent and daily fluid intake was higher.

Logistic Regression Analysis of 28-Day Mortality

In the extended logistic model, APACHE II, SMI, PhA, and ECW/TBW were each associated with day-28 mortality: adjusted odds ratios were 3.634 per 5 APACHE II points (95% CI 2.505–5.270), 0.698 per 1 kg/m² of SMI (95% CI 0.584–0.833), 0.604 per 1 degree of PhA (95% CI 0.473–0.772), and 3.169 per 0.05 increase in ECW/TBW (95% CI 2.074–4.841), all $P < 0.001$. In the clinically adjusted sensitivity model, which additionally included age, sex, BMI, CKD, chronic heart failure, and daily fluid intake, the corresponding odds ratios were 2.996, 0.629, 0.551, and 2.958; all four predictors remained associated with mortality after adjustment for these clinical covariates, and the sensitivity model achieved an AUC of 0.923 (95% CI 0.892–0.955). Core-predictor estimates are given in Table 2; the full clinically adjusted sensitivity model is reported in Supplementary Table S3, and the core-model estimates are visualized in Fig. 3.

Discrimination of Individual Markers and Prediction Models

Discriminative performance is summarized in Table 3 and Fig. 1. The AUC was 0.794 (95% CI 0.743–0.845) for APACHE II alone, 0.838 (0.793–0.883) for the BIA-only model, and 0.900 (0.864–0.937) for the extended APACHE II + BIA model. The extended model surpassed APACHE II alone (AUC difference 0.106, 95% CI 0.066–0.146, P < 0.001) and the BIA-only model (difference 0.062, 95% CI 0.030–0.094, P < 0.001), whereas BIA-only and APACHE II alone did not differ significantly (difference 0.044, 95% CI -0.021 to 0.109, P = 0.187); detailed comparisons are reported in Table 4. After 1,000 bootstrap refits, the optimism-corrected AUC was 0.896 and the optimism-corrected Brier score was 0.091. The apparent calibration intercept and slope were 0.000 and 1.000, respectively; the optimism-corrected calibration intercept was 0.003 and the optimism-corrected calibration slope was 0.966. At the exploratory development-cohort probability threshold of 0.1734, sensitivity was 85.9% (79/92) and specificity was 80.8% (298/369). Exploratory sex-specific SMI thresholds were < = 9.59 kg/m2 for women and < = 11.09 kg/m2 for men; these are not validated clinical cut-offs. One-variable-at-a-time spline sensitivity analyses showed evidence of nonlinearity for age in the sensitivity model (P = 0.005) and borderline evidence for ECW/TBW in the core model (P = 0.046), but not for APACHE II, SMI, PhA, BMI, or daily fluid intake. Compared with the linear core model, the ECW/TBW spline model changed the optimism-corrected AUC only from 0.896 to 0.898 and the corrected Brier score from 0.091 to 0.088; its corrected calibration intercept and slope were 0.001 and 0.942. The linear specification remained the primary analysis because the performance difference was small and the linear equation is more readily interpretable. Apparent calibration and exploratory apparent decision curves are shown in Supplementary Figures S1 and S2. These calibration and functional-form results are summarized in Supplementary Table S2.

Correlations Among BIA Markers and Clinical Variables

Pearson correlations among SMI, PhA, ECW/TBW, APACHE II, age, BMI, and daily fluid intake were mostly weak (Fig. 2). SMI and PhA were positively correlated (r = 0.22), and ECW/TBW correlated only weakly with APACHE II (r = 0.13); the strongest correlation was that between age and APACHE II (r = 0.40). The maximum variance inflation factor in the clinically adjusted sensitivity model was 1.29, indicating no severe multicollinearity.

Discussion

In this prospective single-centre cohort of 461 critically ill Chinese patients, we evaluated a BIA-only model comprising SMI, PhA, and ECW/TBW and an extended model that combined these markers with APACHE II for prediction of 28-day all-cause mortality. All three BIA markers were associated with day-28 mortality, and the extended model achieved the highest development-cohort discrimination. The results support complementary prognostic information from muscle-related, cellular-integrity, and fluid-distribution measures within this cohort.

Each BIA marker captures a different measurable feature of critical illness. Each 1 kg/m2 of higher SMI was associated with lower adjusted odds of day-28 death (OR = 0.698; single-marker AUC = 0.731),

consistent with prior evidence linking low muscle mass to adverse outcomes [1, 2, 25]. Each additional degree of PhA was similarly associated with lower odds (OR = 0.604; AUC = 0.724), supporting cellular integrity as a prognostic indicator [26, 27]. A 0.05 increase in ECW/TBW was associated with higher odds of death (OR = 3.169), although its single-marker AUC was more modest (0.719) and fluid balance can influence the measurement [13, 14]. Inter-marker correlations were weak to modest (SMI-PhA r = 0.22; SMI-ECW/TBW r = -0.14; PhA-ECW/TBW r = -0.12), suggesting that the measures are not interchangeable. The coexistence of lower BIA-derived SMI, lower PhA, and higher ECW/TBW may therefore convey complementary prognostic information, consistent with the extended model's AUC of 0.900. BIA-derived SMI is an algorithmic estimate related to muscle mass rather than a direct measurement of muscle tissue, and hydration shifts can affect that estimate. These indices should be treated as phenotypic markers rather than proven causal mechanisms because inflammation, organ failure, fluid resuscitation, immobility, and pre-existing frailty can influence both BIA values and mortality. Because muscle strength and physical performance were not assessed, the study describes low SMI and does not diagnose sarcopenia [28]. The sex-specific SMI thresholds were exploratory and establish neither biologic equivalence nor validated clinical cut-offs.

Most previous ICU cohorts have evaluated SMI or PhA individually [16, 25, 29]. This study combined SMI, PhA, and ECW/TBW in a BIA-only model and quantified the incremental discrimination when these measures were added to APACHE II. In the development cohort, exploratory Youden thresholds were SMI < = 9.59 kg/m2 for women and < = 11.09 kg/m2 for men, PhA < = 4.44 degrees, and ECW/TBW > = 0.43. Because every threshold was selected in the same cohort used for model development, the estimates are likely optimistic and cannot be considered ethnicity-specific clinical thresholds. The extended model raised the AUC by 0.106 over APACHE II alone and by 0.062 over the BIA-only model, whereas the BIA-only model did not significantly outperform APACHE II alone (P = 0.187). This pattern suggests that the BIA measures complement rather than replace routine severity assessment. A higher AUC reflects better ranking and does not by itself demonstrate better clinical decisions or outcomes. The decision curve was an apparent development-cohort analysis and does not establish net clinical benefit.

All three BIA indices can be measured non-invasively at the bedside within approximately five minutes, without blood sampling or transport to CT or DXA facilities. The model might support further clinical assessment after external validation, but it should not serve as the sole basis for treatment decisions. Repeated BIA monitoring may be feasible in bedridden ICU patients, although this study evaluated only the first measurement. Functional form deserves particular attention for ECW/TBW: the primary model entered continuous predictors linearly, whereas the restricted-cubic-spline sensitivity analysis showed borderline evidence of nonlinear departure (P = 0.046). The reported OR per 0.05 therefore represents an average linear association over the observed range and may not characterize risk equally at its extremes. The nonlinear ECW/TBW model provided only small changes in optimism-corrected discrimination (AUC 0.898 vs. 0.896) and Brier score (0.088 vs. 0.091), with a lower corrected calibration slope (0.942 vs. 0.966). We therefore retained the linear core model and report the nonlinear model as a sensitivity analysis.

The study's strengths include prospective enrolment, restriction to the earliest eligible ICU admission per patient, standardized positioning and electrode placement for BIA, independent source-document checking of key fields, and bootstrap validation with the model refitted in every resample. Several limitations should be considered. First, this was a single-centre model-development study; external validation across hospitals and ICU case mixes is required. Second, 461 patients and 92 deaths limit the precision of performance estimates and the complexity that can be supported reliably. Third, only the first BIA measurement was analysed. Routine fluids and other ICU care had generally started before BIA, infusions were not routinely paused, and strict fasting was not required. Pre-BIA fluid volume, feeding status at the exact measurement time, oedema grade, diuretic exposure, and detailed treatment timing were unavailable; the findings therefore apply to early routine-care BIA rather than an untreated pre-resuscitation state. Fourth, admission diagnosis was not encoded as a standardized analytic variable. The sensitivity model adjusted for available demographic, body-size, comorbidity, and fluid-intake variables, but residual confounding and differences in case mix remain possible. Fifth, thresholds and decision curves were derived in the development cohort and require external evaluation. Finally, the borderline nonlinear ECW/TBW finding should be assessed in an independent dataset. Because patients with terminal malignancy and expected survival under 28 days were excluded, applicability to end-of-life care is limited; nutrition and hydration decisions in that setting depend primarily on goals of care and patient preferences [30].

Future research should perform multicenter external validation of both the BIA-only model and the extended APACHE II + BIA model, covering calibration, discrimination, thresholds, decision consequences, and the possible need for recalibration. External datasets should also compare the linear formulation with a fully specified nonlinear ECW/TBW alternative. Longitudinal BIA tracking may clarify how body-composition changes relate to patient outcomes and help distinguish transient fluid-related changes from persistent low physiologic reserve.

In conclusion, BIA-derived SMI, PhA, and ECW/TBW measured early during routine ICU care were associated with 28-day mortality. Adding these measures to APACHE II produced an apparent AUC of 0.900 and a bootstrap optimism-adjusted AUC of 0.896, with an adjusted calibration intercept of 0.003 and slope of 0.966. The probability threshold, sex-specific SMI thresholds, calibration, and decision-curve results are development-cohort estimates. External validation and prospective evaluation of clinical utility are required before routine use.

Methods

Study Design and Setting

This prospective observational cohort study was carried out in an 18-bed ICU of a tertiary teaching hospital from January 2022 to December 2025. The protocol was approved by the Medical Ethics Committee of Henan Provincial People’s Hospital (Approval No. 2022-K-L041), and written informed

consent was obtained from every participant or a legal surrogate. The study was conducted in accordance with the Declaration of Helsinki and reported in line with the STROBE guidelines.

Study Participants

Consecutively admitted adults aged 18 years or older were screened. Inclusion required an anticipated ICU stay of at least 24 h, a standardized BIA measurement within 8 h of admission, sufficient clinical records for eligibility assessment, and complete ascertainment of vital status at day 28. Patients were excluded for pregnancy; an implantable cardiac pacemaker or cardioverter-defibrillator; limb amputation or severely damaged skin at electrode sites; terminal malignancy with expected survival under 28 days; loss to follow-up before day 28; or unavailable BIA or core-outcome data. When a patient had more than one eligible ICU admission during the study period, only the first admission was analyzed, so each patient contributed exactly one observation.

Data Collection

BIA Measurement

Body composition was assessed with a segmental multifrequency BIA device (InBody S10; InBody Co., Ltd., Seoul, Republic of Korea) within eight hours of ICU admission, following validated ICU protocols [8, 9, 19]. To limit diurnal variation in hydration status, measurements were obtained between 08:00 and 11:00 whenever clinically feasible; patients admitted outside this window were measured as soon as possible, typically within 4–6 h, and the median interval from admission to BIA was 4.2 h (IQR 2.5–6.8). Before testing, patients rested in a semi-recumbent position with a 30-degree back elevation for at least 10 minutes to reduce posture-related fluid shifts. The skin at both wrists and ankles was cleansed with alcohol pads to remove sweat and sebum, and eight tactile electrodes were placed at standard anatomical sites according to the manufacturer's instructions by two trained research nurses who had completed a standardized training program. The device was calibrated monthly, and measurements with impedance values outside the manufacturer's quality-control range were discarded. The parameters exported directly from the device were the manufacturer-reported skeletal muscle index (SMI, kg/m2), a research parameter based on appendicular segmental lean mass divided by height squared and distinct from the separate total-body SMM output; whole-body phase angle at 50 kHz (PhA, degrees); extracellular water (ECW, L); total body water (TBW, L); and the ECW/TBW ratio. BIA operators were blinded to patients' clinical outcomes, which were not yet known at the time of measurement. Routine ICU care had generally begun before BIA. Infusions were not routinely paused, strict fasting was not required, and some patients were receiving enteral or parenteral nutrition. The volume of fluid administered before BIA and the exact timing of vasopressors, mechanical ventilation, renal replacement therapy, and diuretics relative to BIA were not recorded. The measurements therefore represent early routine ICU care rather than an untreated pre-resuscitation state. Muscle strength and physical performance were not assessed; therefore, low SMI was not used to diagnose sarcopenia.

Clinical Data Collection

Electronic medical records supplied age, sex, height, weight, body mass index (BMI), chronic comorbidities, early critical-care interventions, fluid-related indicators, APACHE II components, and day-28 outcome. APACHE II was calculated from the acute physiology, age, and chronic-health components according to standard scoring rules [20]. Mechanical ventilation, vasopressor use, and renal replacement therapy denoted any use during the first 24 h, and daily fluid intake was the total recorded intake during the first 24 h; these variables did not measure exposure specifically before BIA. SMI, phase angle, ECW, TBW, and ECW/TBW were obtained from the manufacturer-generated BIA reports. Data were abstracted using standardized definitions, and the key BIA and APACHE II fields underwent independent source-document verification before database lock. All analyses used the deidentified 41-variable dataset.

Outcome Definition

The primary endpoint was all-cause mortality within 28 days of ICU admission. Vital status at day 28 was confirmed through electronic medical records and telephone follow-up. Outcome assessment was performed by investigators blinded to the BIA measurements.

Statistical Analysis

The analysis included all 461 eligible unique patients, including 92 deaths. All variables used in the reported analyses were available for every patient; no statistical imputation was performed. Continuous variables were summarized as mean ± standard deviation or median (interquartile range) according to distribution, and categorical variables as number (percentage). Between-group comparisons used independent-samples t tests, Mann–Whitney U tests, chi-square tests, or Fisher's exact tests as appropriate. All tests were two-sided, with $P < 0.05$ considered statistically significant. The recorded outcome was vital status at the common day-28 assessment horizon; exact event and censoring times were not collected. Binomial logistic regression was therefore used. Three models were fitted: APACHE II alone; a BIA-only model containing SMI, PhA, and ECW/TBW; and an extended APACHE II + BIA model containing all four predictors as linear main effects. A clinical sensitivity model additionally included age, sex, BMI, chronic kidney disease, chronic heart failure, and first-24-h total fluid intake. Odds ratios and 95% confidence intervals were expressed per 1 kg/m² SMI, 1 degree PhA, 0.05 ECW/TBW, and 5 APACHE II points. Internal validation used 1,000 bootstrap refits (seed 20260818), with each complete model refitted in every resample [21, 22]. Discrimination, Brier score, calibration intercept, calibration slope, and paired DeLong comparisons were evaluated [23]. Optimism was estimated as the mean difference between bootstrap-sample and original-sample performance and subtracted from apparent performance. Decision-curve analysis over thresholds of 0.01–0.50 was treated as exploratory development-cohort analysis [24]. Restricted cubic splines with four fixed knots were applied to one variable at a time as functional-form sensitivity analyses. The linear core model was compared with a model using a restricted cubic spline for ECW/TBW. Sex-stratified SMI receiver operating characteristic analyses were exploratory.

Declarations

Data availability

The deidentified participant-level data that support the findings of this study are not publicly available because public release of detailed clinical data may pose a residual risk of participant re-identification. Data may be available from the corresponding author (lbw006565@163.com) upon reasonable request for legitimate non-commercial research, subject to approval by the Medical Ethics Committee of Henan Provincial People's Hospital and execution of an appropriate data-use agreement. Requests will normally be reviewed within 30 days.

Code availability

The R code used for data validation, model fitting, internal validation, tables, and figures is provided as Supplementary Analysis Code.

Competing interests

The authors declare no competing interests.

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Author Contribution

Q.Y.: Conceptualization, investigation, data curation, formal analysis, and writing—original draft. S.A.: Investigation, data collection, and data validation. L.X.: Investigation, methodology, data validation, and writing—review and editing. B.L.: Conceptualization, methodology, supervision, project administration, and writing—review and editing. All authors read and approved the submitted version.

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Tables

Table 1. Baseline characteristics of the study population.

Characteristic	Total Cohort (n=461)	Survivors (n=369)	Non-Survivors (n=92)	P-value
Age, years, median (IQR)	68 (62-74)	66 (60-73)	72 (68-76)	<0.001
Male sex, n (%)	265 (57.5)	212 (57.5)	53 (57.6)	0.978
Body mass index (BMI), kg/m2, mean±SD	24.84±4.49	25.16±4.51	23.55±4.16	0.001
APACHE II score, mean±SD	15.14±5.17	13.98±4.37	19.82±5.49	<0.001
Comorbidities, n (%)				
Hypertension	214 (46.4)	166 (45.0)	48 (52.2)	0.216
Type 2 diabetes mellitus	134 (29.1)	104 (28.2)	30 (32.6)	0.403
Chronic kidney disease	70 (15.2)	44 (11.9)	26 (28.3)	<0.001
Chronic heart failure	89 (19.3)	62 (16.8)	27 (29.3)	0.006
Clinical interventions and treatment				
Mechanical ventilation, n (%)	243 (52.7)	193 (52.3)	50 (54.3)	0.725
Vasopressor support, n (%)	158 (34.3)	121 (32.8)	37 (40.2)	0.179
Renal replacement therapy, n (%)	40 (8.7)	29 (7.9)	11 (12.0)	0.212
Daily fluid intake, mL, median (IQR)	1888 (1405-2300)	1815 (1378-2246)	2040 (1673-2547)	0.001
BIA parameters				
Skeletal muscle index (SMI), kg/m2, median (IQR)	9.87 (8.58-11.44)	10.19 (8.91-11.82)	8.77 (7.59-9.74)	<0.001
Phase angle (PhA), degrees, mean±SD	4.68±1.38	4.90±1.30	3.80±1.31	<0.001
ECW/TBW ratio, median (IQR)	0.40 (0.36-0.42)	0.38 (0.36-0.41)	0.43 (0.40-0.45)	<0.001

Continuous variables with a normal distribution are presented as mean ± SD and compared with independent-samples t-tests; non-normal continuous variables as median (IQR) with Mann–Whitney U tests; categorical variables as n (%) with the chi-square or Fisher's exact test.

All statistical tests were two-tailed; $P < 0.05$ was considered significant.

Abbreviations: APACHE II = Acute Physiology and Chronic Health Evaluation II; BIA = bioelectrical impedance analysis; ECW/TBW = extracellular water/total body water ratio.

Table 2. Logistic regression analysis of 28-day all-cause mortality.

Predictor	Core model OR (95% CI)	P-value	Sensitivity model OR (95% CI)	P-value
APACHE II, per 5 points	3.634 (2.505-5.270)	<0.001	2.996 (2.000-4.490)	<0.001
SMI, per 1 kg/m2	0.698 (0.584-0.833)	<0.001	0.629 (0.514-0.770)	<0.001
Phase angle, per 1 degree	0.604 (0.473-0.772)	<0.001	0.551 (0.416-0.729)	<0.001
ECW/TBW, per 0.05	3.169 (2.074-4.841)	<0.001	2.958 (1.893-4.623)	<0.001

The core model included APACHE II, SMI, PhA, and ECW/TBW. Odds ratios are expressed at clinically interpretable scales. Full equation: logit(p) = −9.284145 + 0.258060 × APACHE II − 0.359757 × SMI − 0.503543 × PhA + 23.067307 × ECW/TBW, where the individual probability is p = 1/[1 + exp(−logit(p))]. The clinically adjusted sensitivity model additionally included age, sex, BMI, CKD, chronic heart failure, and daily fluid intake.

OR = odds ratio; CI = confidence interval. The exploratory development-cohort operational probability threshold was p ≥ 0.1734 (sensitivity 85.9%; specificity 80.8%).

All tests were two-tailed; P < 0.05 was considered statistically significant.

Table 3. Discriminative performance of individual predictors and prediction models for 28-day all-cause mortality.

Indicator	AUC (95% CI)	Exploratory Development-Cohort Cut-Off	Sensitivity (%)	Specificity (%)	Youden Index
Skeletal muscle index (SMI)	0.731 (0.677-0.784)	<=9.45 kg/m2	69.6	66.1	0.357
Phase angle (PhA)	0.724 (0.669-0.779)	<=4.44 degrees	71.7	64.2	0.360
ECW/TBW ratio	0.719 (0.654-0.785)	>=0.43	51.1	90.0	0.411
APACHE II alone	0.794 (0.743-0.845)	>=0.2585 probability	63.0	79.7	0.427
BIA-only model	0.838 (0.793-0.883)	>=0.1464 probability	87.0	68.0	0.550
Extended APACHE II + BIA model	0.900 (0.864-0.937)	>=0.1734 probability	85.9	80.8	0.666

Optimal cut-off values were determined by the maximum Youden index. For model-probability rows, the displayed ≥ thresholds preserve the classification achieved at the exact unrounded cut-points.

AUC = area under the receiver operating characteristic curve; CI = confidence interval.

Table 4. Pairwise comparison of AUC values among the three models using DeLong's test.

Comparison	AUC Difference	95% CI	Z-value	P-value
Extended APACHE II + BIA model vs APACHE II alone	0.106	0.066-0.146	5.17	<0.001
Extended APACHE II + BIA model vs BIA-only model	0.062	0.030-0.094	3.83	<0.001
BIA-only model vs APACHE II alone	0.044	-0.021 to 0.109	1.32	0.187

DeLong's test is the standard method for comparing correlated ROC AUC values.

A negative AUC difference indicates better predictive performance of the latter model.

Figures

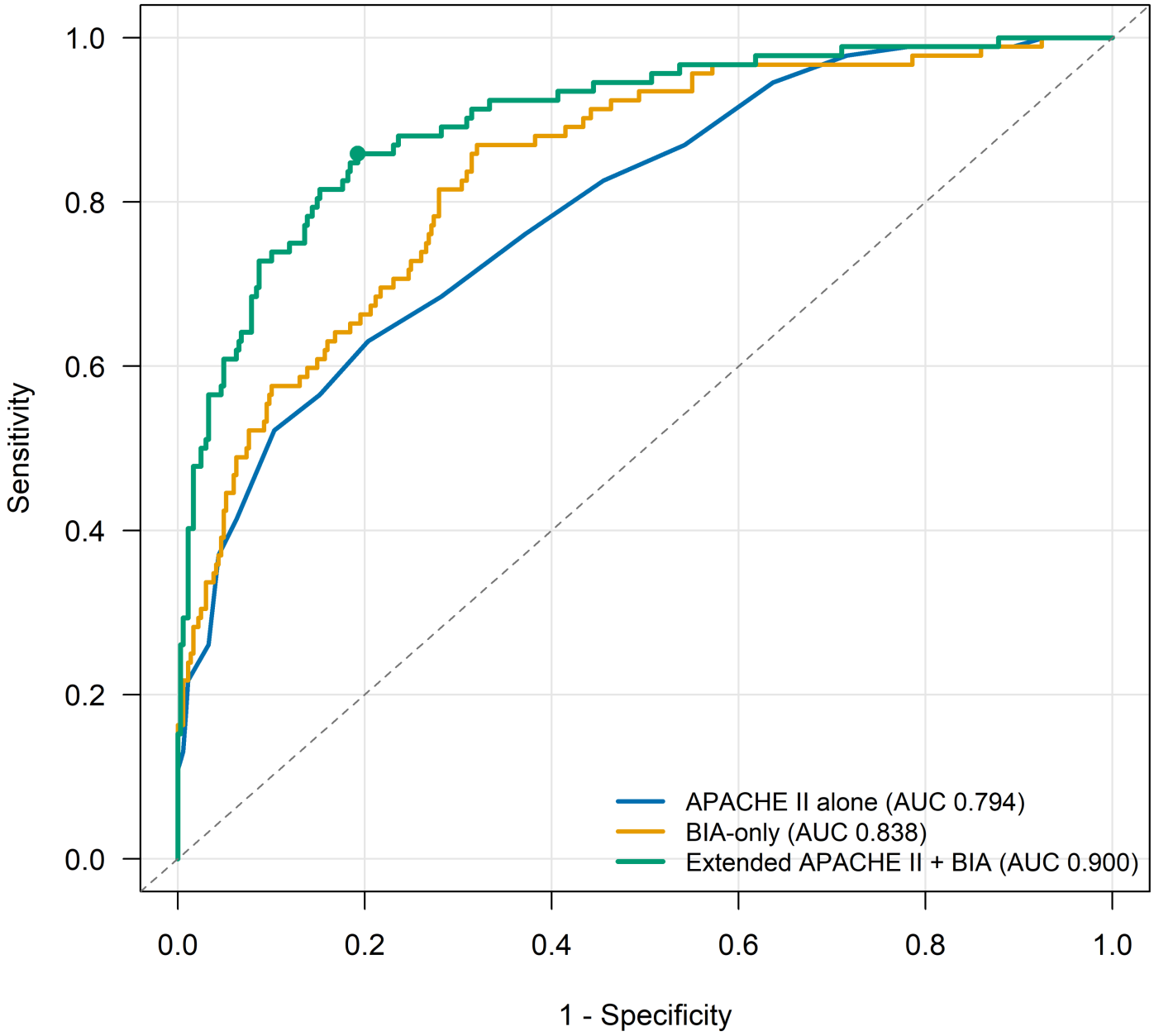


Figure 1

Receiver operating characteristic (ROC) curve analysis for predicting 28-day all-cause mortality.

ROC curves show apparent development-cohort discrimination for APACHE II alone, the BIA-only model, and the extended APACHE II + BIA model. The x-axis is 1-specificity and the y-axis is sensitivity. AUCs were 0.794, 0.838, and 0.900, respectively. The diagonal dashed line represents non-discrimination (AUC = 0.5).

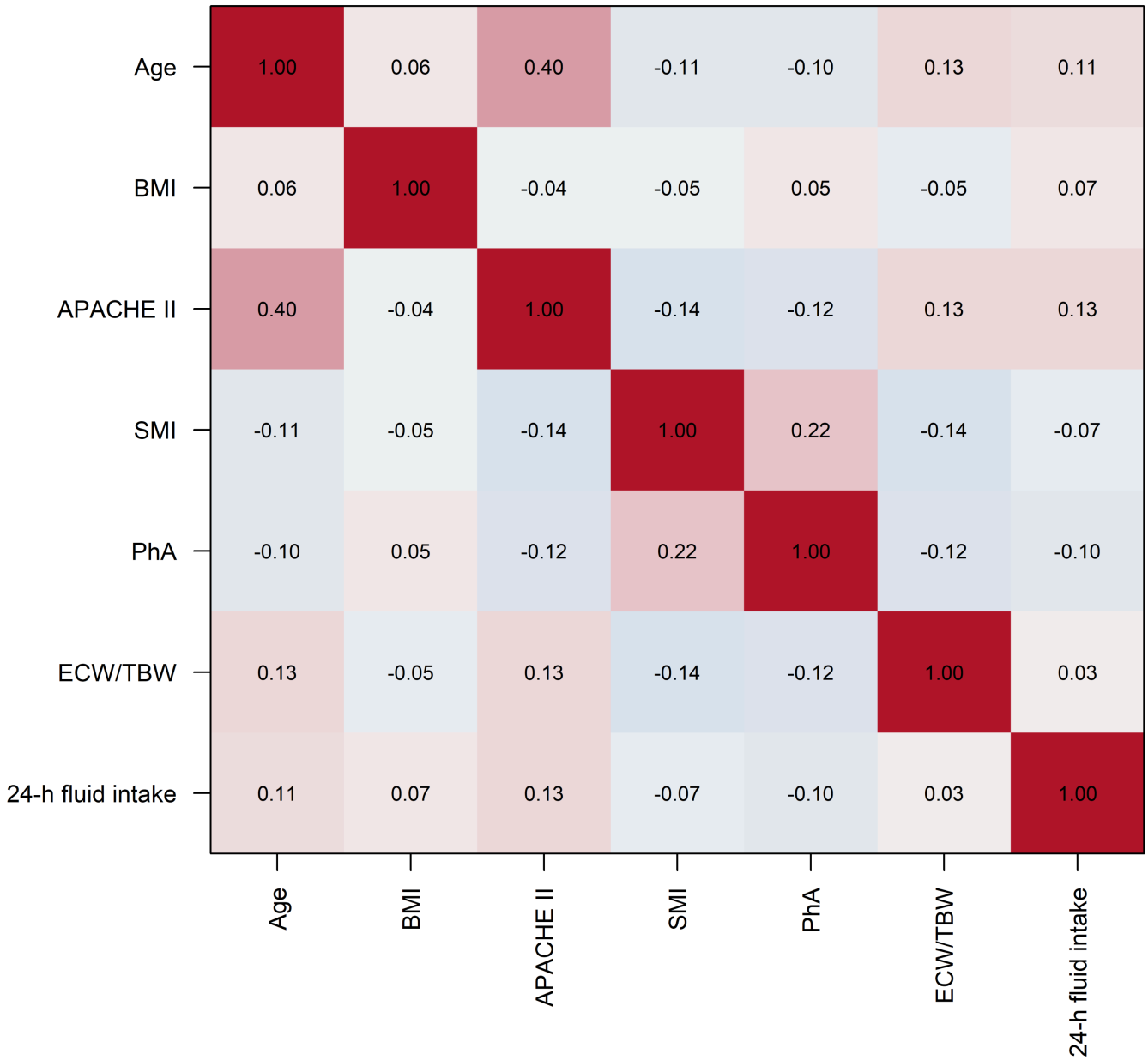


Figure 2

Pearson correlation heatmap of the study variables.

Pearson correlation coefficients are shown for SMI, PhA, ECW/TBW, APACHE II, age, BMI, and first-24-h total fluid intake. The color scale ranges from -1 to +1; positive and negative values indicate correlations in the corresponding direction. SMI and PhA were positively correlated (r = 0.22), ECW/TBW correlated weakly with APACHE II (r = 0.13), and the strongest correlation was between age and APACHE II (r = 0.40).

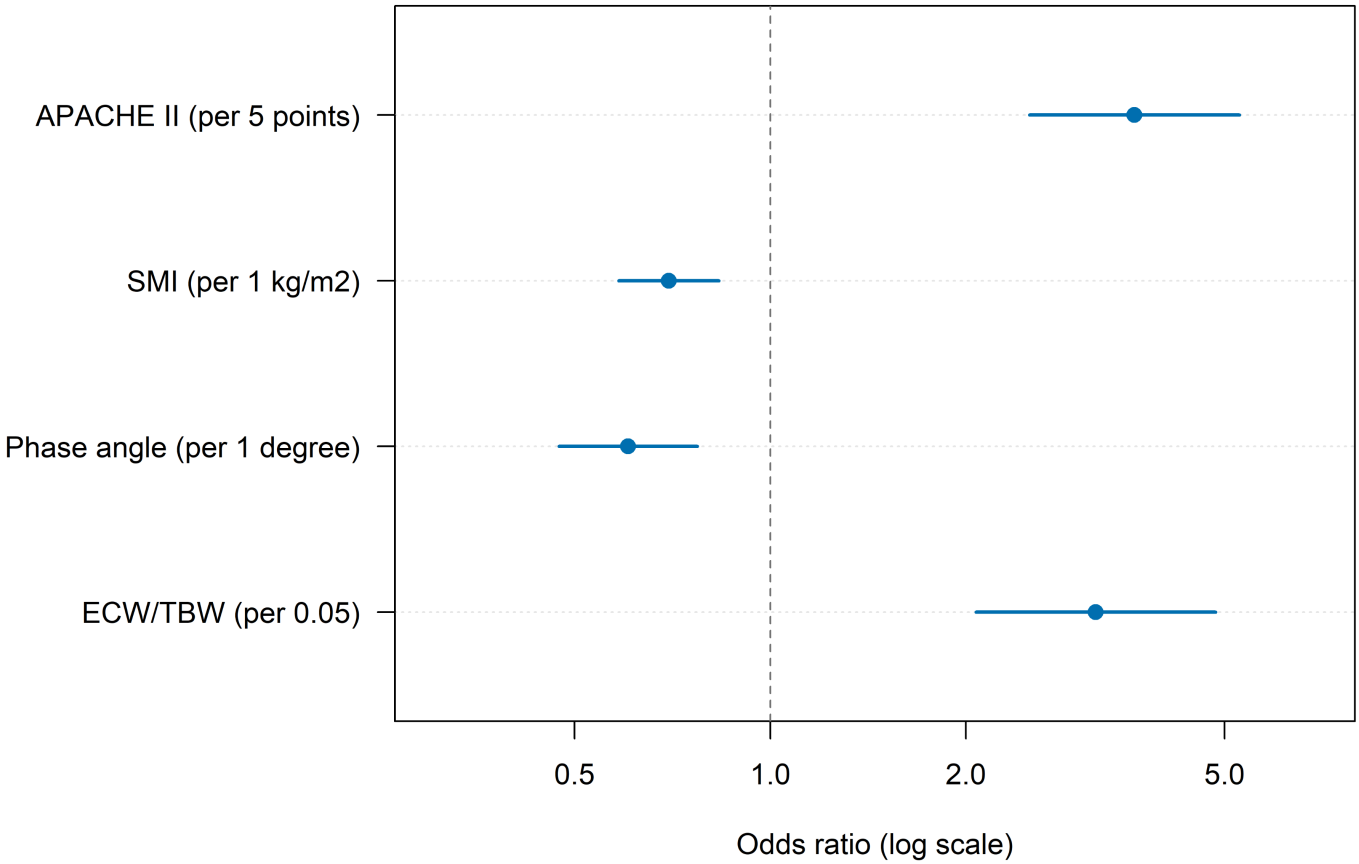


Figure 3

Forest plot of the extended logistic model for 28-day all-cause mortality.

Adjusted odds ratios and 95% CIs from the fixed four-predictor extended logistic model are shown on a logarithmic x-axis. Effects are reported per 5 APACHE II points, 1 kg/m2 SMI, 1 degree PhA, and 0.05 ECW/TBW. The vertical line at OR = 1 is the null value. Estimates describe associations with binary day-28 mortality in the development cohort.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryInformation.pdf
- SupplementaryAnalysisCode.r