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Original Article

A Predictive Nomogram for Intensive Care-Acquired Weakness after Cardiopulmonary Bypass

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Purpose: Intensive care unit-acquired weakness (ICUAW) affects patient prognosis after cardiopulmonary bypass (CPB) surgery, but its risk factors remain unclear. We investigated these risk factors and developed a nomogram for predicting ICUAW after CPB.
Methods: Baseline characteristics, preoperative laboratory data, and intra- and postoperative variables of 473 patients after CPB were determined in this prospective cohort study. Lower limb muscles on bedside ultrasound images were compared 1 day before and 7 days after CPB. Risk factors were assessed using logistic regression models.
Results: Approximately 50.95% of the patients developed ICUAW after CPB. The body mass index (BMI), New York Heart Association (NYHA) class, lactate, albumin, aortic clamping time, operation time, and acute physiological and chronic health evaluation II were determined as independent risk factors. The average absolute error of coincidence was 0.019; the area under the curve, sensitivity, and specificity were 0.811, 0.727, and 0.733, respectively, for the predictive nomogram.
Conclusion: A high BMI, poor NYHA class, preoperative high serum lactate, low serum albumin, long surgical duration, aortic clamping, and high acute physiological and chronic health evaluation II score are risk factors for ICUAW after CPB. This robust and easy-to-use nomogram was developed for clinical decision-making.

Keywords: intensive care unit-acquired weakness, cardiopulmonary bypass, risk factor, nomogram, circulation

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Introduction

Cardiopulmonary bypass (CPB) is frequently performed during cardiovascular surgery.[1)] Routine clinical findings from patients after CPB revealed a high incidence of intensive care unit-acquired weakness (ICUAW). ICUAW is a common neuromuscular dysfunction syndrome, characterized by systemic muscle weakness in intensive care unit (ICU) patients that shows an incidence of 25%–100%.[2,3)] ICUAW results in a range of adverse outcomes, including mechanical ventilator (MV) weaning failure, paresis or quadriplegia, reduced reflexes, various degrees of muscle atrophy, thromboembolic disorders, microcirculation disorders, bedsores, delirium, and varying levels of peripheral neuropathy.[4,5)] It also prolongs hospital stays and increases re-hospitalization, mortality rates, and economic burdens.[2)]

A series of complex physiological processes occurring after CPB stop blood circulation, leading to coagulation dysfunction, proinflammatory responses, and altered redox states.[6] Pathophysiological changes caused by CPB may lead to an increased ICUAW incidence. Therefore, we investigated independent risk factors of ICUAW in patients after CPB. A robust nomogram for predicting the risk of ICUAW after CPB was developed to assist clinical decision-making.

Patients and Methods

Ethical statement

The study was approved by the ethics committee of Fujian Medical University Union Hospital (Ethical Review No. 2020KY015) and was subjected to clinical trial registration in China (registration number: ChiCTR2000034650). Written informed consent for publication was obtained from all participants.

Patients

Patients who had undergone CPB in the Fujian provincial cardiac medical center between December 2019 and November 2020 were enrolled in this study. Inclusion criteria were as follows: patients who underwent cardiac surgery with CPB, aged ≥18 years, and had >24 h of ICU admission. Exclusion criteria were as follows: prior neuromuscular lesions, limb diseases, psychiatric disorders, cerebrovascular diseases, concurrent malignancy, long-term use of neuromuscular blocking agents, and sedatives. (**Supplementary Fig. 1**; all supplementary files are available online.)

Patients enrolled in this study were able to perform normal activities before surgery. Patients on bed rest were admitted to the ICU after CPB. During the consciousness fuzzy period, nurses gave the patients' limb joints passive activity and muscle massage, with each joint activity to be done at least 10 times, twice a day for 10 min each; joint activity order was shoulder, joint-elbow, joint-wrist-finger, joint-hip, joint-knee-ankle-toe joint, with each joint performing flexion, extension, abduction, and adduction activities, respectively. During the awake period, nurses instructed the patients for active limb joint movements twice daily for 15 min. Further, a series of positive measures to regulate the internal environment, control infection, and correct acid–base imbalance and electrolyte disorder, such as appropriate fluid support, pain control, and drug treatment, were administered. Patients were able to move normally in the general ward.

Diagnosis of ICUAW

ICUAW was diagnosed based on standard ultrasound findings.[7] The cross-sectional area (CSA), thickness (TH), and pennation angle (PA) of the bilateral rectus femoris (RF) muscle was measured 1 day before and 7 days after CPB. CSA was defined as the CSA of the RF perpendicular to its longitudinal axis. TH was defined as the distance between the RF and vastus intermedius. PA was measured as the angle at which the muscle fibers entered the aponeurosis. ICUAW was diagnosed when CSA, TH, and PA were decreased by 10%, 20%, and 5%, respectively.[8] Finally, 473 patients were assessed according to the ICUAW diagnostic criteria 1 day before and 7 days after CPB and then assigned to ICUAW or non-ICUAW groups.

These parameters were measured using the Vivid Qportable color Doppler ultrasound instrument (GE Healthcare, Little Chalfont, UK) with a linear array LRS probe (type 9, at a frequency of 10 MHz and a measurable depth of 4 cm). Patients were placed in a supine position with lower extremity braking for 5 min, and the legs splayed at a 30° angle. The operator stood by the right patella of the patient during the measurement and marked the left side of the quadriceps femoris from 1/5 of the left anterior inferior iliac spine to the midpoint of the patella. The probe was covered in gel and placed perpendicularly to the skin surface. A vertical line was drawn from the midpoint of the long axis of the RF to determine CSA. TH was measured using a longitudinal view of the quadriceps femoris. PA was measured with the probe perpendicular to the skin surface. All measurements were repeated and averaged. The right side of the quadriceps femoris was similarly measured (**Fig. 1**).

Variable analysis

We consulted existing ICUAW studies to select factors potentially affecting ICUAW, combined with the opinions of clinical experts, as representative variables as follows. Baseline characteristics, preoperative laboratory parameters, and intra- and postoperative variables were assessed before diagnosis. Demographic data included age, sex, and body mass index (BMI). Data on the history of smoking, alcohol consumption, hypertension, and diabetes mellitus were also included. The Barthel index,[9] Classification of New York Heart Association (NYHA) heart function,[10] and left ventricular ejection fraction (LVEF) were assessed upon admission.

Laboratory values for calcium, glucose, lactate, hemoglobin, albumin, total bilirubin, cardiac troponin, creatine kinase levels, and N-terminal pro-brain natriuretic peptide were determined 1 day before CPB. Intraoperative

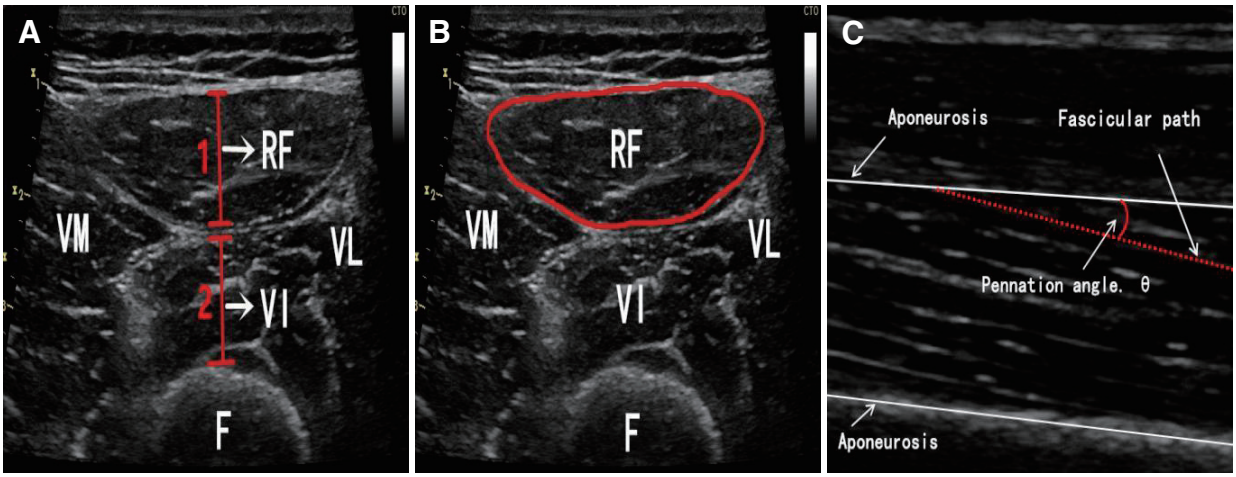

Fig. 1 Muscle measurements based on ultrasound images. (**A**) Muscle TH of RF layer and intermedius femoris layer, (**B**) CSA of RF, and (**C**) PA of RF. CSA: cross-sectional area; F: femur; RF: rectus femoris; VI: vastus internus; VL: vastus lateralis; VM: vastus medialis; PA: pennation angle; TH: thickness

variables included minimum nasal temperature, aortic clamping time, CPB time (operating time of artificial heart–lung machine), operation time (from the beginning to the end of the seam), fluid input and output during surgery duration, and red blood cell infusion. Postoperative variables comprised acute physiology and chronic health evaluation II (APACHE II) scores,[11] duration of MV, duration of critical care, length of stay (LOS) in the hospital, and hospital mortality.

The preoperative laboratory parameters and intra- and postoperative variables were classified by whether they were clinically abnormal or not. The classification method is as follows: LVEF (<50% vs ≥50%), calcium (>1.35 vs ≤1.35 mmol/L), glucose (>6.1 vs ≤6.1 mol/L), lactate (>1.7 vs ≤1.7 mmol/L), hemoglobin (<110 vs ≥110 g/L), albumin (<35 vs ≥35 g/L), total bilirubin (>17.1 vs ≤17.1 μmol/L), cardiac troponin (>0.2 vs ≤0.2 μg/L), and creatine kinase (>198 vs ≤198 U/L).

Statistical analysis

The SPSS version 26.0 software (SPSS, Inc., Chicago, IL, USA) was used for statistical analysis. Bilateral tests were used for all statistical tests, with P <0.05 indicating statistically significant differences. Data showing normal distribution were expressed as mean ± standard deviation and compared by the Student's t-test/analysis of variance. Non-normally distributed data were expressed as median plus quartile spacing and compared by the nonparametric rank-sum test. Enumeration data, expressed as a percentage of cases, were compared between groups using the Chi-square test or Fisher's exact test.

All variables were assessed via univariate logistic regression analysis. Variables with P <0.05 in the univariate logistic regression analysis were included in the multivariate logistic regression analysis. Variables were screened using the stepwise method with P <0.05 indicating significant differences, and the variables with statistically significant differences (P <0.05) were selected as independent risk factors. Based on independent risk factors, the ICUAW risk prediction nomogram model was constructed using the rms package in the R software (http://www.r-project.org/). Internal validation of 1000 bootstrap self-sampling and graphic calibration was used to evaluate the effect of the nomogram model.

Results

Incidence of ICUAW

Among the 473 patients after CPB, the CSA, TH, and PA decreased by varying degrees. The degree of decline is more obvious in groups with ICUAW (**Fig. 2**). A total of 241 (50.95%) patients after CPB developed ICUAW, as measured by ultrasound. The male prevalence of ICUAW was higher than the female prevalence.

Baseline characteristics between patients with and without ICUAW

Among the preoperative, intraoperative, and postoperative variables, sex, BMI, hypertension, Barthel index, NYHA class, LVEF, calcium, lactate, albumin, total bilirubin, creatinine, cardiac troponin, and creatine kinase were different between the ICUAW and non-ICUAW

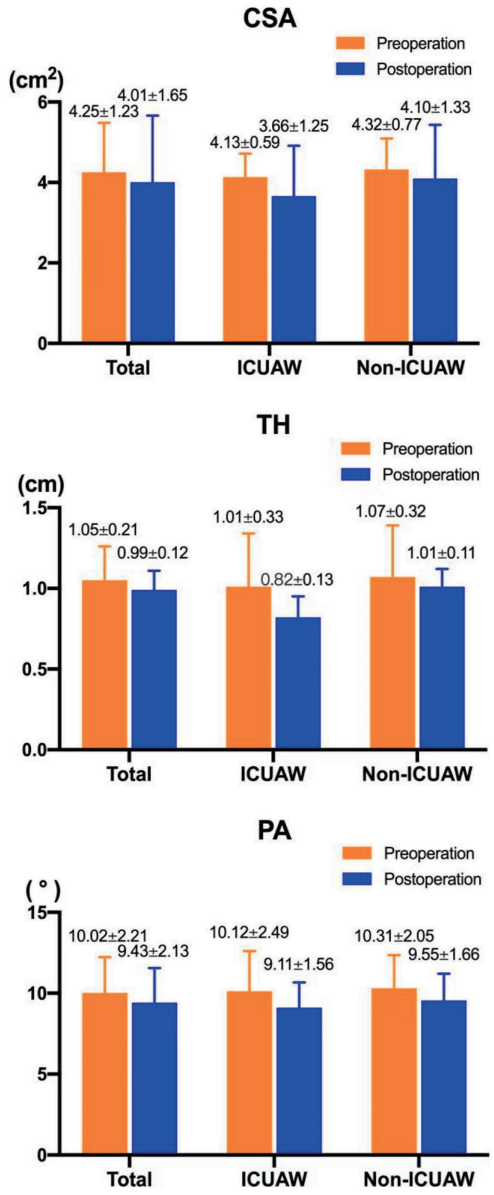

Fig. 2 Comparison of pre-and postoperative muscle decline in patients after CPB. Average CSA, TH, and PA of the bilateral RF muscle at 1 day before and 7 days after CPB. CSA: cross-sectional area; TH: thickness; PA: pennation angle; ICUAW: intensive care unit-acquired weakness; RF: rectus femoris; CPB: cardiopulmonary bypass

patients. Patients diagnosed with ICUAW had a longer aortic clamping time, CPB time, and operation time. Patients with ICUAW usually had a higher APACHE II score, duration of MV, duration of critical care, and LOS in the hospital after CPB (**Table 1**).

Risk factors of ICUAW

Multivariate logistic regression analysis revealed that BMI (95% CI, 1.030–1.189), NYHA class (95% CI, 1.083–2.311), lactate (95% CI, 1.235–4.875), albumin (95% CI, 1.525–5.893), aortic clamping time (95% CI, 1.003–1.026), operation time (95% CI, 1.004–1.015), and APACHE II (95% CI, 1.029–1.197) were independent risk factors for ICUAW after CPB (**Table 2**).

Nomogram model and validation

Based on these independent risk factors, a nomogram model was developed to predict the risk of ICUAW (**Fig. 3**). Based on these seven independent risk factors, a column chart was created using R language. The left side represents the names of the risk factors, and the tick marks on the lines indicate the range of possible values for each factor. The length of the lines reflects the magnitude of the influence of each factor on the occurrence of ICU-acquired weakness. The individual scores, called "Point," corresponding to each risk factor at different values are summed up to calculate the total score, referred to as "Total Point." The corresponding risk is the probability of developing ICU-acquired weakness.

An internal validation of the model was conducted, and the calibration plot for ICUAW probability after CPB indicated the relationship between the actual observation and nomogram predictions, and the average absolute error of coincidence was 0.019. The receiver operating characteristic curve and area under the curve values were obtained by cross-validation. The nomogram was developed with an area under the curve, sensitivity, and specificity of 0.811, 0.727, and 0.733, respectively (**Supplementary Fig. 2**).

Discussion

In our study, patients after CPB exhibiting one or more of the following were prone to developing ICUAW: high BMI, poor NYHA class, high preoperative lactate levels, low albumin, long aortic clamping time, long operation time, and high APACHE II scores. A robust nomogram for predicting ICUAW after CPB was developed based on these findings. The average absolute error of the coincidence was 0.019, with an area under the curve, sensitivity, and specificity of 0.811, 0.727, and 0.733, respectively.

Prevention of ICUAW is critical because there are no clear treatment methods and specific drugs for ICUAW.[12] The nomogram is a simple visual figure that intuitively predicts the risk of an individual disease. It is convenient and easy to popularize for assisting with clinical decision-making. The value of the patient's independent risk

Table 1 Comparison of baseline characteristics between patients with and without ICUAW

	Total	ICUAW	Non-ICUAW	*P*-value
	(n = 473)	(n = 241)	(n = 232)	
Demographics				
Age (years)	57 (48–64)	57 (49–64)	56 (47–64)	0.429
Male sex, n (%)	253 (53.5)	140 (58.1)	113 (48.7)	0.041
BMI (kg/m^2)	22.6 (20–25)	24 (21–25)	22 (20–24)	<0.001
Smoking history, n (%)	111 (23.5)	59 (24.5)	52 (22.4)	0.596
Alcohol consumption, n (%)	26 (5.5)	18 (7.5)	8 (3.4)	0.055
Hypertension, n (%)	150 (31.7)	87 (36.1)	63 (27.2)	0.037
Diabetes, n (%)	49 (10.4)	28 (11.6)	21 (9.1)	0.360
Bathel index, n (%)				<0.001
1	70 (14.8)	56 (23.2)	14 (6.0)	
2	14 (3.0)	8 (3.4)	6 (2.5)	
3	389 (82.2)	179 (74.3)	210 (90.5)	
NYHA class, n (%)				0.004
1	31 (6.6)	11 (4.6)	20 (8.6)	
2	146 (30.9)	62 (25.7)	84 (36.2)	
3	272 (57.5)	151 (62.7)	121 (52.2)	
4	24 (5.1)	17 (7.1)	7 (3.0)	
LVEF (%)	65.0 (59.25–69.8)	63.8 (58.3–68.6)	66.3 (61.1–70.3)	0.003
Calcium (mmol/L)	1.11 (1.08–1.15)	1.10 (1.07–1.15)	1.12 (1.08–1.15)	0.033
Glucose (mmol/L)	6.0 (5.3–7.2)	6.0 (5.3–7.7)	5.9 (5.3–6.9)	0.401
Lactate (mmol/L)	0.9 (0.7–1.4)	1.0 (0.7–1.8)	0.9 (0.7–1.3)	<0.001
Hemoglobin (g/L)	132 (117–143)	132 (117–143)	133 (118–142)	0.603
Albumin (g/L)	40.2 (36.8–43.1)	39.4 (35.6–42.7)	40.8 (38.0–43.8)	<0.001
Total bilirubin (μmol/L)	12.5 (8.5–19.7)	14.3 (9.5–22.8)	11.5 (8.1–17.2)	<0.001
Cardiac troponin (ng/L)	0.002 (0.001–0.010)	0.003 (0.001–0.015)	0.002 (0.001–0.006)	0.002
Creatine kinase (U/L)	69 (50–103)	81 (52–117)	65 (49–92)	0.002
NT-proBNP (pg/L)	865 (455–1983)	913 (423–2012)	841 (412–1988)	0.122
Intraoperative variables				
Minimum nasal temperature (°C)	30.2 (30.0–31.0)	30.0 (29.3–31.0)	30.5 (30.0–31.2)	<0.001
Aortic clamping (min)	63 (41–90)	76 (56–104)	49 (33–73)	<0.001
CPB time (min)	121 (91–153)	136 (113–175)	96 (71–130)	<0.001
Operation time (min)	255 (210–313)	288 (242–335)	225 (183–270)	<0.001
Fluid input (mL)	2600 (2100–3375)	2720 (2300–3410)	2500 (2000–3323)	0.093
Fluid output (mL)	2000 (1700–2500)	2100 (1700–2600)	2000 (1600–2300)	0.006
RBC infusion (U)	2 (0–4)	2 (0–4)	1 (0–3)	0.005
Postoperative variables				
APACHE II	22 (20–24)	23 (21–26)	21 (19–23)	<0.001
Duration of MV (h)	28 (18–62)	43 (21–103)	19 (15–34)	<0.001
Duration of critical care (h)	65 (41–118)	91 (48–176)	45 (31–69)	<0.001
LOS in hospital (days)	20 (15–27)	22 (16–29)	17 (14–24)	<0.001
Mortality, n (%)	12 (2.5)	7 (2.9)	5 (2.2)	0.604

Data are shown as n (%) or median with IQR. APACHE II: acute physiology and chronic health evaluation II; BMI: body mass index; CPB: cardiopulmonary bypass; ICUAW: intensive care unit-acquired weakness; LOS: length of stay; LVEF: left ventricular ejection fraction; MV: mechanical ventilator; NYHA: New York Heart Association; NT-proBNP: N-terminal pro-brain natriuretic peptide; RBC: red blood cell; IQR: interquartile range

factor corresponds to the value of each variable, and the sum of the resulting score added corresponds to the "Total points" value, which is the incidence of ICUAW after CPB. Patients at high risk of ICUAW can be further managed with diversified interventions and fine management.[13] Aside from passive exercise, good limb placement, bedside-seated balance exercises, standing, and walking can be practiced. These specific interventions

Table 2 Logistic regression model to predict ICUAW

Variable	Univariate analysis	*P*-value	Multivariate analysis	*P*-value
	OR (95% CI)		OR (95% CI)	
Demographics				
Age (years)	1.004 (0.991–1.018)	0.539	–	–
Male sex, n (%)	0.661 (0.459–0.952)	0.026	0.703 (0.435–1.36)	0.15
BMI (kg/m^2)	1.116 (1.055–1.180)	<0.001	1.107 (1.030–1.189)	0.006
Smoking history, n (%)	1.154 (0.751–1.774)	0.513	–	–
Alcohol consumption, n (%)	3.600 (1.314–9.865)	0.013	2.336 (0.638–8.550)	0.2
Hypertension, n (%)	1.513 (1.021–2.240)	0.039	0.750 (0.436–1.291)	0.299
Diabetes, n (%)	1.367 (0.747–2.503)	0.311	–	–
Bathel index	0.492 (0.367–0.660)	<0.001	0.797 (0.489–1.300)	0.364
NYHA class, n (%)	1.636 (1.243–2.153)	<0.001	1.582 (1.083–2.311)	0.018
LVEF, <50% vs ≥50%	1.366 (0.637–2.928)	0.423	–	–
Calcium, >1.35 vs ≤1.35 (mmol/L)	1.271 (0.857–1.886)	0.233	–	–
Glucose, >6.1 vs ≤6.1 (mol/L)	1.108 (0.792–1.550)	0.548	–	–
Lactate, >1.7 vs ≤1.7 (mmol/L)	3.139 (1.883–5.233)	<0.001	2.453 (1.235–4.875)	0.01
Hemoglobin, <110 vs ≥110 (g/L)	1.700 (0.988–2.924)	0.055	–	–
Albumin, <35 vs ≥35 (g/L)	3.450 (1.956–6.086)	<0.001	2.998 (1.525–5.893)	0.001
Total bilirubin, >17.1 vs ≤17.1 (μmol/L)	1.918 (1.291–2.851)	0.001	1.129 (0.672–1.897)	0.647
Cardiac troponin, >0.2 vs ≤0.2 (μg/L)	2.857 (1.184–6.893)	0.019	0.899 (0.302–2.675)	0.848
Creatine kinase, >198 vs ≤198 (U/L)	1.309 (0.680–2.521)	0.42	–	–
NT-proBNP (pg/L)	1.158 (0.558-2.346)	0.122	–	–
Intraoperative variables				
Minimum nasal temperature (°C)	0.866 (0.811–0.924)	<0.001	1.041 (0.932–1.162)	0.48
Aortic clamping (min)	1.023 (1.017–1.030)	<0.001	1.014 (1.003–1.026)	0.011
CPB time (min)	1.019 (1.014–1.023)	<0.001	0.997 (0.988–1.007)	0.616
Operation time (min)	1.014 (1.011–1.017)	<0.001	1.010 (1.004–1.015)	<0.001
Fluid input (mL)	1.000 (1.000–1.000)	0.476	–	–
Fluid output (mL)	1.000 (1.000–1.000)	0.009	1.000 (1.000–1.000)	0.661
RBC infusion (U)	1.137 (1.048–1.233)	0.002	0.938 (0.841–1.046)	0.25
Postoperative variables				
APACHE II	1.234 (1.160–1.313)	<0.001	1.110 (1.029–1.197)	0.007
Duration of MV (h)	1.014 (1.009–1.019)	<0.001	1.004 (0.999–1.009)	0.125
Duration of critical care (h)	1.008 (1.005–1.010)	<0.001	1.002 (1.000–1.005)	0.096

CI: confidence interval; OR: odds ratio; APACHE II: acute physiology and chronic health evaluation II; BMI: body mass index; CPB: cardiopulmonary bypass; LVEF: left ventricular ejection fraction; MV: mechanical ventilator; NT-proBNP: N-terminal pro-brain natriuretic peptide; NYHA: New York Heart Association; RBC: red blood cell

may include early guidance for patients, active exercise, neuromuscular electrical stimulation resistance exercise, four-level exercise, personalized nutritional management according to a dietician's evaluation, optimizing the nutritional formula, limiting total energy intake, moderation of high-protein diet, infusion of albumin to patients with hypoalbuminemia, and comprehensive intervention to reduce the occurrence of ICUAW and improve the poor prognosis of ICUAW.[14]

In our study, ICUAW was diagnosed by comparing the pre-and postoperative muscles of patients with ultrasonography. The real "gold standard" diagnostic test for ICUAW was not identified even in the last Clinical Practical Guideline.[15] ICUAW can be diagnosed by manual muscle testing, electrophysiology, and muscle or nerve tissue pathology, but these procedures involve taking human tissue for biopsy, causing pain, injury, infection, and great harm to patients. The most widely used diagnostic test for ICUAW in clinical practice is manually testing muscle strength using the Medical Research Council (MRC) scale. However, the MRC score divides muscle strength into six grades, requires muscle strength assessment at 12 sites in the patient (such as neck flexion, hip flexion, and shoulder abduction), and requires patients to be awake and actively cooperate with manual testing of muscle strength.[3] A current alternative tool, to

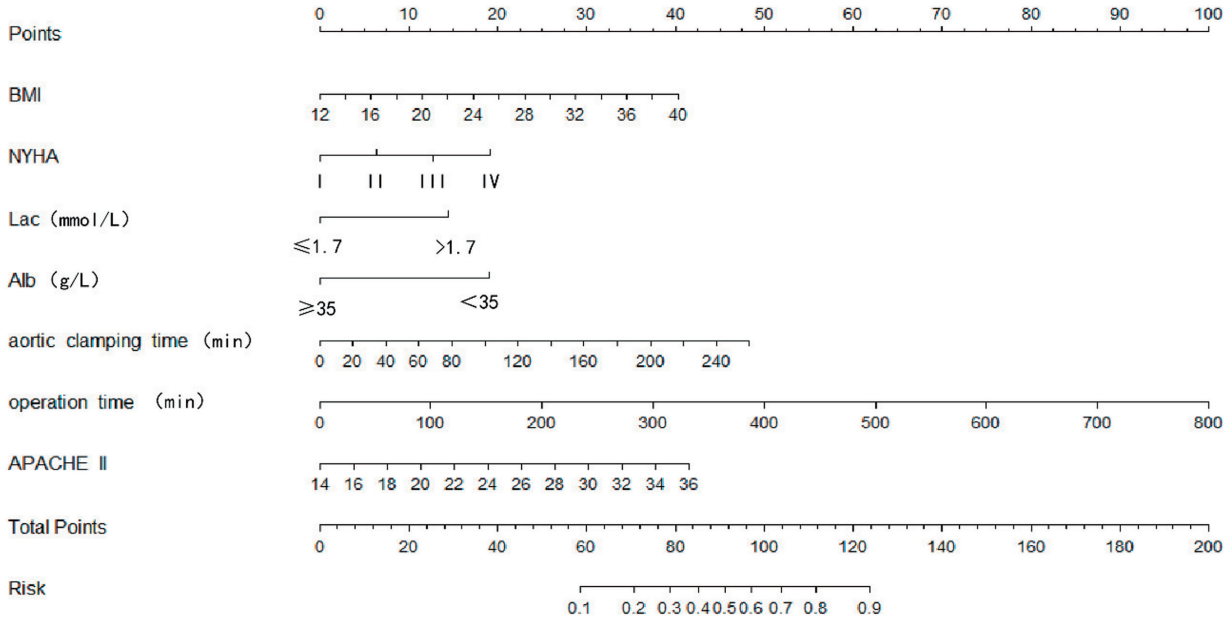

Fig. 3 Nomogram to estimate the risk of ICUAW after CPB. ICUAW: intensive care unit-acquired weakness; CPB: cardiopulmonary bypass; BMI: body mass index; NYHA: New York Heart Association; Lac: lactate; Alb: albumin; APACHE II: acute physiological and chronic health evaluation II

effectively assess the skeletal muscle mass, that may be conveniently used in the ICU is ultrasonography.[8] Ultrasound measurement is a rapid, noninvasive, painless, simple, and reproducible technique for the qualitative and quantitative determination of muscle mass, avoiding patient subjective offset and any harm to the patients. It can reliably detect the pathological changes in the muscle, judge the changes in muscle content in critically ill patients, and has diagnostic value for ICUAW.[8]

Our results demonstrated that 50.95% of patients developed ICUAW after CPB, which is comparable to previous findings in critically ill patients admitted into the ICU.[16,17] It may be associated with cardiac patients adopting CPB. Systemic heparinization in patients before CPB, hemodilution during CPB, aortic clamping time, temperature changes, and neutralization measures after the end of CPB expose patients to a complex range of non-physiological conditions. These lead to muscle ischemia-reperfusion injury, poor perfusion of organs, coagulation dysfunction, systemic inflammation, and other pathophysiological changes.[18] Furthermore, CPB-related factors, including contact with artificial materials, a low perfusion state, shifting body temperatures, drug stimulation, surgical trauma, operation time, and bleeding may cause the occurrence of ICUAW.[19]

We further evaluated the risk factors of ICUAW. First, the APACHE II score is currently the most authoritative critical illness evaluation and can reflect the severity of pathophysiological changes in patients.[20] EuroSCORE is the most well-known system assessing the risk factors before cardiovascular surgery.[21] However, it does not include intraoperative risk factors, such as those for CPB. The APACHE II score is simple and reliable, with reasonable design and accurate prediction. It is a better choice for early ICUAW prediction. Patients with high postoperative APACHE II scores evaluated 1 day after CPB, were more likely to develop ICUAW. Second, the NYHA classification has been used to assess the physical activity, according to the degree of activity that induces heart failure symptoms. It evaluates the degree of impaired cardiac function and the severity of cardiac disease in patients.[22] Our results indicate that patients with a low NYHA class displayed a higher probability of ICUAW. Third, as the main colloid component in the blood, albumin plays an important role in maintaining the human blood osmotic pressure and microcirculation perfusion, preventing ischemia and reperfusion injury, and having a certain positive effect on the blood and oxygen supply of the body.[23] Lower levels of albumin may further disrupt protein synthesis and enhance protein degradation in patients following CPB.[24] Moreover, hypoalbuminemia reflects an inflammatory state,[25] thereby resulting in faster loss of muscle protein, and causes muscle atrophy. Skeletal muscle atrophy is an early and significant characteristic of ICUAW and is mainly caused by unbalanced protein synthesis.[26] Fourth, hyperlacticemia is mainly caused by

defects in pyruvate carboxylase and pyruvate dehydrogenase and is common in cases of poor peripheral circulation due to an inadequate oxygen supply, shock, and sepsis.[27,28] Elevated lactic acid levels may be associated with muscle damage. Finally, high BMI is an independent risk factor, causing death from various cardiovascular diseases.[29] The effect of high BMI on cardiovascular disease leads to the development of ICUAW through the alteration of the underlying morphological and functional hemodynamics.[30]

This study had several limitations. First, all the study samples were from a provincial heart medicine center. The absence of multicenter data for verification may lead to bias. Second, no long-term follow-up was performed for patients with ICUAW after CPB. Finally, the prediction nomogram was not subjected to external validation using an independent dataset. Despite these limitations, these findings provide a basis for future investigations of risk factors for ICUAW after CPB, using a robust and easy-to-use nomogram to assist with clinical decision-making.

Conclusions

Approximately 50.95% of patients after CPB developed ICUAW, as detected by ultrasonography. BMI, NYHA class, lactate, albumin, aortic clamping time, operation time, and APACHE II were independent risk factors for ICUAW after CPB. Based on these risk factors, a robust and easy-to-use nomogram for predicting the risk of ICUAW after CPB was developed to assist in clinical decision-making. Patients at high risk of ICUAW can be further managed with diversified interventions and fine management.

Author Contributions

Yanjuan Lin: conceptualization, methodology, writing – review and editing, funding acquisition, and supervision. Liangwan Chen: conceptualization, methodology, writing – review and editing, and supervision. Fuxiu Zhong: conceptualization, methodology, investigation, formal analysis, and writing – original draft. Haoruo Zhang: investigation, formal analysis, and writing – original draft. Yanchun Peng: investigation, formal analysis, and writing – original draft. Xueying Lin: investigation and formal analysis.

Data and Materials Availability

All data generated or analyzed during this study are included in this published article (and its supplementary information files).

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Disclosure Statement

The authors declare no conflict of interest.

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