

Mechanistic Differences in Early Allograft Dysfunction Following OLT versus ELRA: Analysis and Predictive Model Construction

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Abstract

Objective

This study aimed to analyze the risk factors associated with early allograft dysfunction (EAD) following orthotopic liver transplantation (OLT) and ex vivo liver resection combined with autologous liver transplantation (ELRA), and to develop predictive models with the ultimate goal of optimizing clinical pathways and improving patient outcomes.

Methods

This retrospective study analyzed patients who underwent LT in the ICU of between January 2013 and April 2024. Patients were categorized based on EAD occurrence and surgical procedure. Comparative analyses were conducted across the groups to identify the independent risk factors for EAD, which were subsequently used to develop a predictive model.

Results

In OLT recipients, postoperative day 0 (POD0) procalcitonin, POD0 interleukin-6 (IL-6), and POD0 prothrombin time were identified as the primary risk factors for EAD. In ELRA recipients, intraoperative packed red blood cell transfusion volume, POD0 international normalized ratio (INR), and postoperative epinephrine use were significantly associated with EAD development. Based on these findings, separate nomograms were constructed for each surgical cohort. Both nomogram models demonstrated satisfactory goodness-of-fit, and the decision curve analysis (DCA) together with the clinical impact curve (CIC) confirmed their clinical utility, thereby enhancing the early identification of EAD.

Conclusions

This study suggests that the pathogenic mechanisms underlying EAD differ according to the type of liver transplantation performed. In OLT, EAD appears to be predominantly driven by inflammatory-immune cascades, whereas in ELRA, it is primarily governed by perioperative hemodynamic and circulatory instability. The category-specific predictive models developed in this study demonstrated superior accuracy in identifying high-risk patients along distinct pathological pathways. Accordingly, differentiated clinical management strategies are warranted: OLT recipients may benefit from early and targeted anti-inflammatory intervention, while ELRA patients require meticulous monitoring and optimization of volume status and hemodynamic parameters.

INTRODUCTION

There is an increasing number of patients awaiting liver transplantation (LT), and the severe imbalance between liver supply and demand has led to an increased utilization of marginal donor livers, resulting in a decline in graft quality and an increased incidence of early allograft dysfunction (EAD).^{1,2} EAD is a critical factor affecting patient prognosis, as it increases the risk of complications, prolongs hospitalization, and increases medical costs.³ The incidence of EAD ranges from 10.8% to 36.3%, with an associated more mortality rate.⁴ Additionally, multicenter studies have shown that the one-year graft survival rate in patients with EAD may decrease from 72–90% to approximately 43–64%.⁵ Current research has predominantly focused on identifying the risk factors for EAD, mainly involving donor liver quality and intraoperative cold ischemia time.^{6–8} However, predictive models for post-LT EAD remain scarce. Existing models often rely on a single intraoperative or postoperative risk factor,⁹ lacking a comprehensive assessment incorporating preoperative, intraoperative, and postoperative variables, thereby limiting their predictive capability. Furthermore, most studies have focused on allogeneic LT (OLT), with limited research on the risk factors for EAD after ex vivo liver resection and autotransplantation (ELRA), leading to a lack of individualized predictive models tailored to different surgical approaches.

This study aimed to retrospectively analyze clinical data from patients undergoing different types of liver transplantation (LT) to systematically assess the risk factors associated with early allograft dysfunction (EAD) and to develop nomogram-based predictive models integrating preoperative, intraoperative, and postoperative indicators. By doing so, we sought to provide novel insights and practical tools for elucidating the underlying mechanisms of EAD across distinct LT modalities, as well as for its prevention and clinical management, ultimately advancing the standard of care in liver transplantation.

METHODS

Study design, setting, and population

This retrospective study analyzed the clinical data of patients who underwent LT admitted to the intensive care unit (ICU) of the First Affiliated Hospital of Xinjiang Medical University between April 2013 and April 2024. Patients with severe postoperative complications (e.g., major cardiovascular events), age <18 years, and incomplete clinical data were excluded. During the study period, 293 patients who underwent LT were admitted to the ICU. After excluding 45 pediatric patients and 18 patients with incomplete clinical data, 230 patients were included in the final analysis.

Ethical statements

All research data underwent strict anonymization and de-identification processing to remove personally identifiable information. The study was approved by the Ethics Committee of Xinjiang Medical University prior to data collection and analysis (approval no. K202412-29) and conducted in accordance with the Declaration of Helsinki and relevant ethical guidelines. The need for informed consent was waived, considering the minimal risk to participants and the inability to obtain consent from all participants.

Data collection

Clinical data were extracted from the hospital's electronic medical records system, including the following.

Demographic and baseline characteristics

Age, sex, ethnicity, occupation, educational level, body weight, body mass index (BMI), Acute Physiology and Chronic Health Evaluation II (APACHE II) score, MELD score, Charlson Comorbidity Index (CCI), history of chronic diseases, liver disease complications, and prior medical interventions.

Preoperative variables

Complete blood count, including white blood cell (WBC) count, neutrophil percentage (NE%), hemoglobin level, and platelet (PLT) count; electrolyte levels, including sodium and potassium levels; renal function parameters such as creatinine (Cr) and blood urea nitrogen; liver function parameters such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), direct bilirubin, indirect bilirubin, and albumin; coagulation parameters such as activated partial thromboplastin time, prothrombin time, international normalized ratio (INR), fibrinogen, and thrombin time (TT) were measured.

Perioperative and treatment variables

Surgical procedure type; operation duration; anhepatic phase duration; cold ischemia time; warm ischemia time; intraoperative blood loss; transfusion volume; and ICU interventions, including vasopressor use, antibiotic therapy, mechanical ventilation duration, continuous renal replacement therapy support, immunosuppressive therapy, and nutritional support strategies, were assessed.

Postoperative variables

Blood gas analysis; complete blood count; liver function; renal function, coagulation parameters; and inflammatory markers, including procalcitonin (PCT), IL-6 (interleukin-6), and C-reactive protein were measured on postoperative day 0 (POD0) and 1 (POD1). Additionally, bacterial and fungal cultures of the postoperative drainage fluid, ICU length of stay, total hospitalization duration, and hospital costs were recorded.

Grouping and analysis

Patients were stratified by surgical procedure into the OLT group and the ELRA group, and further classified into the EAD group and the non-EAD group according to established diagnostic criteria. Comparative analyses were conducted across these groups to identify the independent risk factors for EAD, which were subsequently used to develop a predictive model.

EAD was diagnosed when the patient met one or more of the following criteria on POD 7¹⁰:

1. Total bilirubin level $\geq 171 \mu\text{mol/L}$
2. INR ≥ 1.6
3. ALT or AST level $> 2,000 \text{ U/L}$

Definitions of LT types

(1) ELRA is a surgical technique in which the liver is temporarily or partially removed, subjected to hypothermic preservation and continuous hypothermic perfusion, followed by reimplantation after lesion resection and graft reconstruction.¹¹(Fig. S1)

(2) OLT is the transplantation of a healthy donor liver, commonly used for end-stage liver disease and acute liver failure. Due to immune system differences between donor and recipient, long-term immunosuppressive therapy is required to prevent graft rejection.¹²

Statistical analysis

Statistical analyses were performed using R software (version 4.2.3) and SPSS 27.0. The MICE package in R was used for multiple imputations of missing values ($< 10\%$ missing rate), and variables with excessive missing data were excluded. Spearman's correlation analysis and variance inflation factor (VIF) were used to assess multicollinearity and interactions between variables.

Categorical variables are presented as frequencies and proportions and compared using the χ^2 test. Ordinal variables were analyzed using the rank-sum test. Continuous variables were assessed for normality: Normally distributed data are expressed as mean $\pm$ SD ($\bar{x} \pm s$) and compared using the independent sample t-test. Non-normally distributed data are expressed as medians (P25–P75) and compared using the Mann–Whitney U test. A binary logistic regression model was used to identify independent risk factors for EAD. To construct the predictive model, R software (version 4.2.3) was used to generate a nomogram. The predictive performance of the model was evaluated using the AUROC. Goodness-of-fit tests were used to assess model calibration, and clinical net benefit analysis was used to evaluate the clinical utility of the model.

RESULTS

A total of 230 patients who underwent LT were included in this study. Of these, 127 underwent ELRA and 103 underwent OLT.

Baseline characteristics

A total of 103 OLT patients were enrolled, of whom 45 (42.45%) developed EAD postoperatively. Among the 127 ELRA patients, 35 (27.56%) developed EAD. In the OLT group, comparison of clinical and preoperative characteristics between the EAD and non-EAD groups revealed that only alanine aminotransferase (ALT) differed significantly between the two groups ($P=0.041$). In the ELRA group,

serum creatinine and aspartate aminotransferase (AST) levels were significantly different between the EAD and non-EAD groups ($P<0.05$), while no statistically significant differences were observed in the remaining variables (Table 1).

An analysis of preoperative variables indicated that most parameters did not differ significantly between the EAD and non-EAD groups. The distribution of surgical procedure type differed significantly between the EAD and non-EAD groups ($P=0.011$), with a higher proportion of OLT patients observed in the EAD group. Among OLT patients, intraoperative comparison revealed statistically significant differences in anhepatic phase duration, total fluid input, total blood product transfusion, red blood cell transfusion units, and plasma transfusion volume between the EAD and non-EAD groups ($P<0.05$). Among ELRA patients, total blood product transfusion, red blood cell transfusion units (PRBCs), and plasma transfusion volume were significantly different between the two groups ($P<0.05$) (Table 2).

Among OLT patients, postoperative ICU and laboratory indicators revealed statistically significant differences between the EAD and non-EAD groups in POD0 lactate (Lac), procalcitonin (PCT), interleukin-6 (IL-6), platelet count (PLT), serum creatinine (Cr), albumin (Alb), prothrombin time (PT), and INR, as well as POD1 PLT, Cr, fibrinogen (Fib), and thrombin time (TT). Among ELRA patients, statistically significant differences between the EAD and non-EAD groups were observed in POD0 partial pressure of oxygen (PO_2), pH, Lac, IL-6, Alb, PT, and INR, as well as POD1 PLT, Fib, TT, ICU length of stay, and hospitalization costs (Table 3).

Spearman's correlation analysis showed no strong correlations between the related factors (Fig. 1); the duration of the anhepatic phase was moderately correlated with surgical duration. The collinearity results indicated that the VIF of all related factors was <5 (Table 4), suggesting no significant interaction between the factors.

Predictive model construction

Screening for risk factors for EAD after LT and construction of a nomogram model

Variables showing statistically significant differences in the univariate analysis (Tables 1–3) were separately included in binary logistic regression analyses for each surgical procedure (Table 4). For OLT, the analysis of risk factors for EAD revealed that POD0 PCT (OR=1.046, 95% CI: 1.012–1.081, $P=0.007$), POD0 IL-6 (OR=1.003, 95% CI: 1.001–1.006, $P=0.003$), and POD0 PT (OR=1.063, 95% CI: 1.014–1.224, $P=0.011$) were significant independent risk factors for EAD following OLT. For ELRA, intraoperative PRBCs (OR=1.186, 95% CI: 1.007–1.305, $P<0.001$), POD0 INR (OR=2.154, 95% CI: 1.180–3.898, $P=0.011$) and postoperative adrenaline use (OR=5.982, 95% CI: 1.256–27.974, $P=0.025$) were identified as significant risk factors for EAD following ELRA.

Nomogram model for predicting EAD risk after LT

Nomogram models were constructed separately to predict the risk of EAD following OLT and ELRA (Figure 2). Risk factors for EAD after OLT included POD0 PCT, POD0 IL-6, and POD0 TT. Risk factors for

EAD after ELRA included intraoperative PRBCs, POD0 INR, and postoperative adrenaline use.

Evaluation of the nomogram model for predicting EAD risk after LT

The ROC curve for the OLT EAD prediction nomogram yielded an AUC of 0.79 (Figure 3A), and the calibration curve demonstrated satisfactory goodness-of-fit (Figure 3B). The ROC curve for the ELRA EAD prediction nomogram yielded an AUC of 0.76 (Figure 4A), and the calibration curve similarly demonstrated satisfactory goodness-of-fit (Figure 4B). The clinical utility of both predictive models was further evaluated using decision curve analysis (DCA) and clinical impact curves (CIC). The results showed that the net benefit of the model (red curve) exceeded that of the other two reference lines beyond a threshold probability of 2%, indicating that the model's net benefit was substantially superior to both extreme strategies. The decision curves confirmed clinically meaningful net benefit across the corresponding threshold probability ranges (Figures 3C–D and 4C–D).

DISCUSSION

EAD is a critical contributor to early postoperative mortality in liver transplant recipients⁴, and its pathogenic mechanisms have remained a central focus of clinical investigation. Previous studies have established that EAD arises from the complex interplay of multiple perioperative factors, including donor organ quality, recipient baseline status, intraoperative surgical technique, and intraoperative hemodynamic fluctuations¹³. With the advancement of surgical techniques in recent years, growing attention has been directed toward the differential impact of distinct transplantation procedures on early graft functional recovery. Prior literature has demonstrated that different surgical approaches give rise to markedly distinct mechanisms of ischemia-reperfusion injury, attributable primarily to differences in intraoperative vascular reconstruction strategies, ex vivo organ preservation duration, and hemodynamic profiles¹⁴. In the present study, by developing procedure-specific predictive models, we found that the pathogenic mechanisms underlying EAD differ between OLT and ELRA: EAD following OLT appears to be predominantly driven by inflammatory-immune cascades, whereas EAD following ELRA is primarily governed by perioperative hemodynamic instability and the extent of surgical insult.

The Mechanism of EAD After OLT: Inflammatory Activation and Immune Response

Elevated POD0 PCT and IL-6 levels were significant risk factors for EAD (PCT OR=1.046, P=0.007; IL-6 OR=1.003, P=0.003). PCT and IL-6 are key markers of inflammation, and their elevated levels may reflect postoperative inflammation or an increased risk of infection in the transplanted liver.¹⁵ EAD is associated with various inflammatory factors, and elevated postoperative IL-6 levels have been significantly linked to its increased incidence.¹⁶ This underscores the importance of early monitoring of inflammatory markers and timely initiation of anti-inflammatory treatment to reduce the risk of EAD.¹⁷

Elevated POD0 PT was identified as an independent risk factor for EAD following OLT (OR=1.063, P=0.011), suggesting that the recovery of coagulation factor synthetic capacity plays an important role

in the pathophysiological process of EAD^{18,19}. Compared with isolated transaminase elevation, coagulopathy more directly reflects insufficient functional hepatocyte mass and global hepatic dysfunction, indicating that restoration of coagulation function is closely associated with the development of EAD²⁰. In clinical practice, a marked elevation of PT in the early postoperative period often indicates inadequate graft functional recovery, insufficient microcirculatory perfusion, or significant ischemia-reperfusion injury^{21,22}. Therefore, close monitoring of hepatic arterial and portal blood flow, lactate clearance, bilirubin trends, and postoperative complications is warranted in this setting.

The Mechanisms of EAD After ELRA: Hemodynamic Instability and Surgical Insult

Intraoperative RBCs transfusion was a significant risk factor for EAD occurrence (OR=1.186, P<0.001). A large number of intraoperative transfusions may reflect a prolonged surgery time or intraoperative hemodynamic instability, among other issues.²³ Excessive transfusions can lead to graft microcirculation disorders and inflammatory responses,²⁴ thereby worsening graft function. Therefore, indications for intraoperative blood transfusion should be strictly controlled to reduce the risk of EAD.

Elevated POD0 INR (OR=2.154, P=0.011) was closely related to the occurrence of EAD. This indicator reflects postoperative coagulopathy and may be an important indicator of EAD. Hudcova J⁴ found that elevated postoperative INR significantly increased the incidence of EAD and was associated with poor graft survival. Postoperative coagulopathy may result from graft dysfunction, poor liver quality, or improper intraoperative hemodynamic management. An elevated INR particularly when accompanied by increased total bilirubin levels, is a strong predictor of early graft loss, underscoring its value in EAD risk assessment.²⁵ Therefore, close monitoring of the coagulation function and timely intervention in the early postoperative period are essential.

Postoperative epinephrine use was associated with a significantly elevated risk of EAD (OR=5.982, P=0.025). The requirement for epinephrine may reflect underlying hemodynamic instability, which can impair graft reperfusion and compromise early functional recovery of the transplanted liver²⁶. Therefore, rigorous postoperative hemodynamic management is essential, including optimization of fluid resuscitation and judicious use of vasoactive agents, to maintain circulatory stability and thereby reduce the risk of EAD development²⁷.

POD1 fibrinogen demonstrated a potential protective effect against EAD (OR=0.599, P=0.085); however, given the wide confidence interval and borderline P value, the reliability of this finding warrants further validation. Elevated fibrinogen levels reflect preserved hepatic synthetic function and may contribute to the maintenance of microcirculatory stability^{28,29}. Previous studies have confirmed that preoperative fibrinogen levels are closely associated with intraoperative blood loss and early graft function, and that maintaining adequate fibrinogen levels may reduce transfusion requirements and improve transplanted liver survival^{30,31}.

The major risk factors for EAD following OLT include POD0 PCT, POD0 IL-6, and POD0 INR. The underlying mechanisms are likely related to the immune responses and inflammatory activation triggered by allogeneic liver transplantation. Elevated POD0 PCT and IL-6 levels indicate enhanced postoperative inflammatory responses [16], while elevated POD0 INR reflects postoperative coagulopathy¹⁰, both of which increase the risk of EAD development. Furthermore, studies have demonstrated that patients who develop EAD exhibit significantly reduced short-term survival rates and graft survival compared with those who do not³². Therefore, targeted optimization of postoperative management can effectively reduce the incidence of EAD and improve transplantation success rates³³. In clinical practice, risk assessment and individualized management should be guided by the surgical approach and patient-specific characteristics³⁴. For patients undergoing ELRA, particular attention should be directed toward coagulation function, hepatic functional recovery, and the maintenance of hemodynamic stability to ensure adequate graft perfusion. For patients undergoing OLT, the management focus should be placed on early anti-inflammatory intervention and immune modulation, with close monitoring of inflammatory marker trends to facilitate timely identification of EAD.

Advantages of the prediction model in this study

This study developed and validated nomogram models to predict EAD following both OLT and ELRA, confirming their feasibility and clinical applicability. Unlike previous models, the present models incorporate a comprehensive set of predictors spanning preoperative, intraoperative, and postoperative domains. By performing stratified analyses according to surgical procedure type, these models enable a multidimensional reflection of the primary mechanisms underlying EAD across distinct transplantation modalities. The model construction involved a thorough analysis of all potential postoperative EAD risk factors, with particular emphasis on early postoperative indicators that are readily obtainable at POD0, such as PCT and IL-6 levels. The key strengths of this model lie in its integration of preoperative, intraoperative, and postoperative variables into a unified framework, alongside the provision of a visual nomogram tool that facilitates rapid risk stratification in clinical settings. The favorable decision curve performance further validates the clinical utility of these models for EAD risk prediction and provides an important reference for guiding postoperative management.

Study limitations

Although this study provides insights into the differential mechanisms of EAD across distinct surgical procedures, several limitations should be acknowledged. First, as a single-center retrospective study with a relatively limited sample size and the absence of external validation, the findings may be subject to selection bias. Second, due to data constraints, certain potentially relevant variables — including cold ischemia time and graft-to-recipient weight ratio — could not be incorporated, which may have limited the completeness of the mechanistic analysis. Furthermore, this study focused primarily on early graft dysfunction and did not explore the long-term implications of these mechanistic differences on recipient survival outcomes. Future studies employing multicenter, large-sample, prospective designs with more

comprehensive variable inclusion are warranted to further validate and refine the predictive models developed herein.

CONCLUSIONS

In conclusion, this study suggests that the pathophysiological mechanisms underlying EAD differ between OLT and ELRA. In clinical practice, procedure-specific monitoring and intervention strategies should be adopted accordingly: early anti-inflammatory management should be prioritized for OLT recipients, whereas maintaining hemodynamic stability should be emphasized for ELRA patients. Future multicenter, large-sample, prospective studies are warranted to further elucidate the molecular biological mechanisms of EAD across distinct transplantation modalities, thereby continuously advancing the individualized management of liver transplant recipients.

Abbreviations

APACHE II

Acute Physiology and Chronic Health Evaluation II

APTT

activated partial thromboplastin time

BMI

body mass index

BUN

blood urea nitrogen

CCI

Charlson Comorbidity Index

Cr

creatinine

DBiL

direct bilirubin

EAD

early allograft dysfunction

ELRA

ex vivo liver resection and autotransplantation

Hb

hemoglobin

ICU

intensive care unit

IL-6

interleukin-6

INR

international normalized ratio

Lac
lactate
LT
liver transplantation
NE%
neutrophil percentage
OLT
allogeneic liver transplantation
PCT
procalcitonin
PLT
platelet
POD
postoperative day
PRBCs
packed red blood cells
PT
prothrombin time
RBC
red blood cell
ROC
receiver operating characteristic curve
TT
thrombin time
UCB
indirect bilirubin
VIF
variance inflation factor
WBC
white blood cell

Declarations

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Author Contributions

Jingnan Xu, Jingjie Wang, Yi Wang, Xiangyou Yu designed the study; Jingjie Wang, Wenzhe Li, Zhihua Li, Qihang Zheng retrieved the data; Qihang Zheng, Jingyan Wang, Jingnan Xu Participated in data analysis; Jingjie Wang, Jingnan Xu, Wenzhe Li wrote the manuscript; Xiaoming Gao, Chunbo Yang, Ruifeng Chai, Hao Wen, Yingmei Shao, Tuerganaili Aji supervised this study, Xiangyou Yu, Yi Wang funded this study. All authors affirm their contributions to the manuscript reviews, involving critical revisions for significant intellectual content, and their approval of the final text for submission. Every author bears the responsibility for the content of the manuscript.

Conflict of Interest Statement

The authors declare no conflicts of interest

Data Availability

All data generated or analyzed during this study are included in this article and its online supplementary material files. Further inquiries can be directed to the first author. The datasets collected and analyzed during the current study are not publicly available due to patient privacy restrictions under the General Data Protection Regulation. Anonymized data are available from the corresponding author upon reasonable request, pending approval from the Institutional Review Board of The Ethics Committee of Xinjiang Medical University and a signed data use agreement.

Statement of Ethics

Study approval statement: This study was approved by the Ethics Committee of Xinjiang Medical University prior to data collection and analysis (approval no. K202412-29).

Consent to participate statement:

The study has been granted an exemption from requiring written informed consent by the Ethics Committee of Xinjiang Medical University prior.

Clinical trial number statement:

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Tables

Table 1 Comparison of baseline characteristics and preoperative variables between the EAD and non-EAD groups in OLT and ELRA patients

	EAD group	Non-EADgroup	Z/ χ^2 /t	P
OLT group	n=45	n=58		
Age (years)	51.00(43.00-56.50)	49.50(43.00-54.25)	0.759	0.448
Sex[n(%)]			0.822	0.365
Male	25(55.56)	27(46.55)		
Female	20(44.44)	31(53.45)		
Weight (kg)	61.00[59.00-75.00)	62.00[55.00-75.00)	0.453	0.651
MELD	15.33[8.66-24.13)	16.50[8.06-24.98)	0.213	0.831
CCI	3.00[3.00-6.50)	3.00[3.00-4.00)	0.688	0.491
Complicated with diabetes mellitus [n (%)]			2.915	0.088
Yes	8(17.78)	4(6.90)		
Complicated with hepatic encephalopathy [n (%)]			14.275	<0.001
Yes	10(22.22)	0(0.00)		
Preoperative variables				
ALT(U/L)	31.00(21.70-55.50)	47.72(27.00-109.12)	-2.048	0.041
AST(U/L)	52.00(35.15-81.39)	72.58(39.16-180.64)	-1.762	0.078
ELRA group	n=35	n=92		
Sex[n(%)]			0.000	0.982
Male	14(40.00)	37(40.22)		
Female	21(60.00)	55(59.78)		
Weight(kg)	56.00(46.20-64.00)	58.00(47.30-62.75)	-0.678	0.498
MELD	6.00(6.00-7.46)	6.00(6.00-7.16)	0.097	0.922
CCI	3.00(3.00-3.00)	3.00(3.00-3.00)	0.589	0.556
History of prior hepatic surgery [n (%)]			1.164	0.281
Yes	30(85.71)	15		
Preoperative variables				
Cr(μ mol/L)	49.44(40.19-63.47)	56.00(47.00-56.00)	-2.091	0.037
ALT(U/L)	45.08(29.00-77.00)	31.46(18.13-66.27)	1.929	0.054

AST(U/L)	43.60(32.73-70.23)	31.93(23.95-56.05)	2.482	0.013
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Table 2 Comparison of intraoperative variables between the EAD and non-EAD groups

	EAD group	Non-EADgroup	χ^2 Z/t	P
Surgical procedure			6.523	0.011
OLT	45	58		
ELRA	35	92		
OLT group				
Operation duration (h)	10.92(9.08-13.67)	10.46(8.78-14.04)	0.096	0.923
Anhepatic phase duration (h)	1.87(1.48-2.38)	1.45(1.00-2.28)	2.791	0.005
Total fluid input (L)	9.05(6.64-12.30)	6.95(5.51-9.96)	2.407	0.016
Total blood product transfusion (L)	4.30(2.71-5.82)	2.79(1.85-3.91)	3.384	<0.001
PRBCs (U)	9.00(6.00-13.50)	7.50(4.00-10.00)	2.306	0.021
Plasma transfusion (L)	1.22(0.90-1.92)	1.01(0.69-1.40)	2.191	0.028
ELRAgroup				
Operation duration (h)	16.17(14.67-20.45)	15.67(14.47-17.21)	1.219	0.223
Anhepatic phase duration (h)	6.50(5.25-7.50)	6.51(5.25-7.50)	0.439	0.660
Total fluid input (L)	9.46(8.60-13.23)	9.46(8.54-11.12)	0.838	0.402
Total blood product transfusion (L)	2.92(1.85-4.44)	2.19(1.56-2.90)	2.815	0.005
PRBCs (U)	7.00(4.00-12.00)	4.00(3.00-6.00)	3.494	<0.001
Plasma transfusion (L)	0.80(0.59-1.15)	0.60(0.44-1.01)	1.958	0.050

Table 3 Univariate comparison of postoperative variables between the EAD and non-EAD groups in OLT and ELRA patients

	EAD group	Non-EADgroup	$Z/\chi^2/t$	P
OLT group	n = 45	n = 58		
APACHE II	12.00(8.00-15.00)	11.00(7.00-15.00)	1.177	0.239
POD0				
Lac(mmol/L)	4.80(2.40-10.50)	3.24(1.70-5.83)	2.960	0.003
PCT(ng/mL)	10.58(4.35-24.31)	3.31(0.96-7.36)	3.757	<0.001
IL-6(pg/mL)	212.10(103.20-543.95)	119.40(48.93-206.60)	3.592	<0.001
PLT(109/L)	37.00(24.00-62.50)	55.50(27.75-90.75)	-2.215	0.027
Cr(μ mol/L)	91.00(65.89-117.41)	77.35(60.62-95.53)	2.118	0.034
ALB(g/L)	33.58 $\pm$ 6.28	34.33 $\pm$ 6.33	0.601	0.009
PT(s)	28.70(21.75-38.50)	23.85(19.50-29.13)	2.480	0.013
INR	2.50(1.84-3.38)	2.06(1.65-2.54)	2.627	0.009
POD1				
PLT(109/L)	30.00 (21.50-53.00)	45.00(28.50-88.50)	-2.627	0.009
Cr(μ mol/L)	121.17(71.88-188.54)	77.91(59.71-109.19)	2.962	0.003
PT(s)	22.70(16.75-35.05)	18.85(15.68-25.08)	2.154	0.031
INR	2.05(1.43-3.05)	1.57(1.29-2.15)	2.430	0.015
FIB(g/L)	1.42(1.06-2.04)	1.77(1.37-2.38)	-2.391	0.017
TT(s)	26.50(24.10-30.75)	23.80(22.20-26.40)	2.933	0.003
Postoperative administration of adrenaline[n(%)]			6.679	0.010
Yes	10	3		
Hospitalization costs (10,000 CNY)	29.87(23.46-43.81)	24.41(17.88-28.76)	2.729	0.006
ELRA group	n=35	n=92		
APACHE II	11.00[7.00-14.00]	9.50[7.00-12.00]	1.581	0.114
POD0				
PO2(mmHg)	160.00(125.00-196.00)	187.50(146.25-222.75)	-2.094	0.036
pH	7.40(7.35-7.46)	7.43(7.38-7.49)	-2.200	0.028

Lac(mmol/L)	6.80(4.1-9.90)	5.30(3.40-8.10)	2.008	0.045
IL-6(pg/mL)	215.80(87.30-605.60)	105.05(67.18-225.40)	2.453	0.014
ALB(g/L)	37.17±6.99	40.76±6.85	2.628	0.010
PT(s)	27.10 (21.90-34.30)	22.70(20.43-26.80)	2.598	0.009
INR	2.20(1.84-3.01)	1.96(1.74-2.35)	2.199	0.028
POD1				
PLT(109/L)	102.00(82.00-132.00)	143.50(90.25-196.00)	-2.350	0.019
FIB(g/L)	1.94(1.51-2.58)	2.62(1.77-3.17)	-2.682	0.007
TT(s)	25.90(23.00-28.90)	22.60(21.00-25.28)	2.547	0.011
Postoperative administration of adrenaline[n(%)]			12.307	<0.001
Yes	8	3		
Length of hospital stay (days)	41.00(31.58-51.50)	39.50(29.50-58.16)	0.027	0.978
ICU length of stay (days)	5.00(3.00-11.00)	3.00(2.00-5.00)	2.963	0.003
Hospitalization costs (10,000 CNY)	22.77(18.11-30.30)	19.51(15.91-24.90)	2.299	0.022

Table 4 Binary Logistic Regression Analysis of OLT and ELRA Patients: EAD Group vs. Non-EAD Group

	β	SE	Wald	<i>P</i>	OR	95%CI	VIF
OLT group							
POD0 PCT	0.045	0.017	7.309	0.007	1.046	1.012-1.081	1.006
POD0 IL-6	0.003	0.001	8.769	0.003	1.003	1.001-1.006	1.079
POD0 PT	0.061	0.024	6.493	0.011	1.063	1.014-1.224	1.077
Constant	-3.255	0.795	16.753	<0.001	0.039		
ELRAGroup							
PRBCs(U)	0.171	0.049	12.137	<0.001	1.186	1.077-1.305	1.021
POD0 INR	0.767	0.303	6.426	0.011	2.154	1.180-3.898	1.009
POD1 FIB(g/L)	-0.513	0.297	2.976	0.085	0.599	0.334-1.072	
Postoperative administration of adrenaline	1.780	0.792	5.054	0.025	5.928	1.256-27.974	1.0282
Constant	-2.855	1.108	6.642	0.010	0.058		

Figures

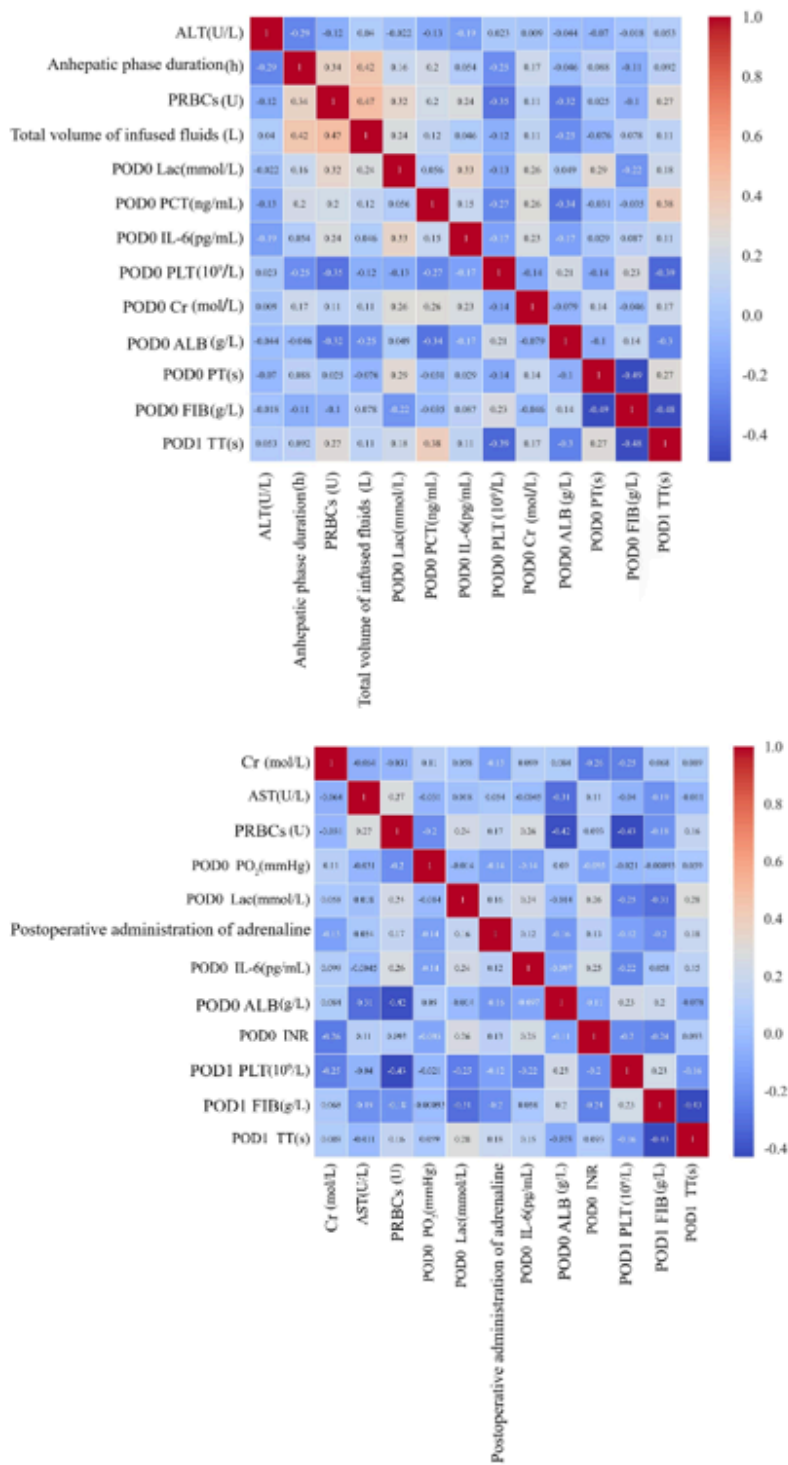


Figure 1

A: Spearman correlation analysis in OLT patients; B: Spearman correlation analysis in ELRA patients

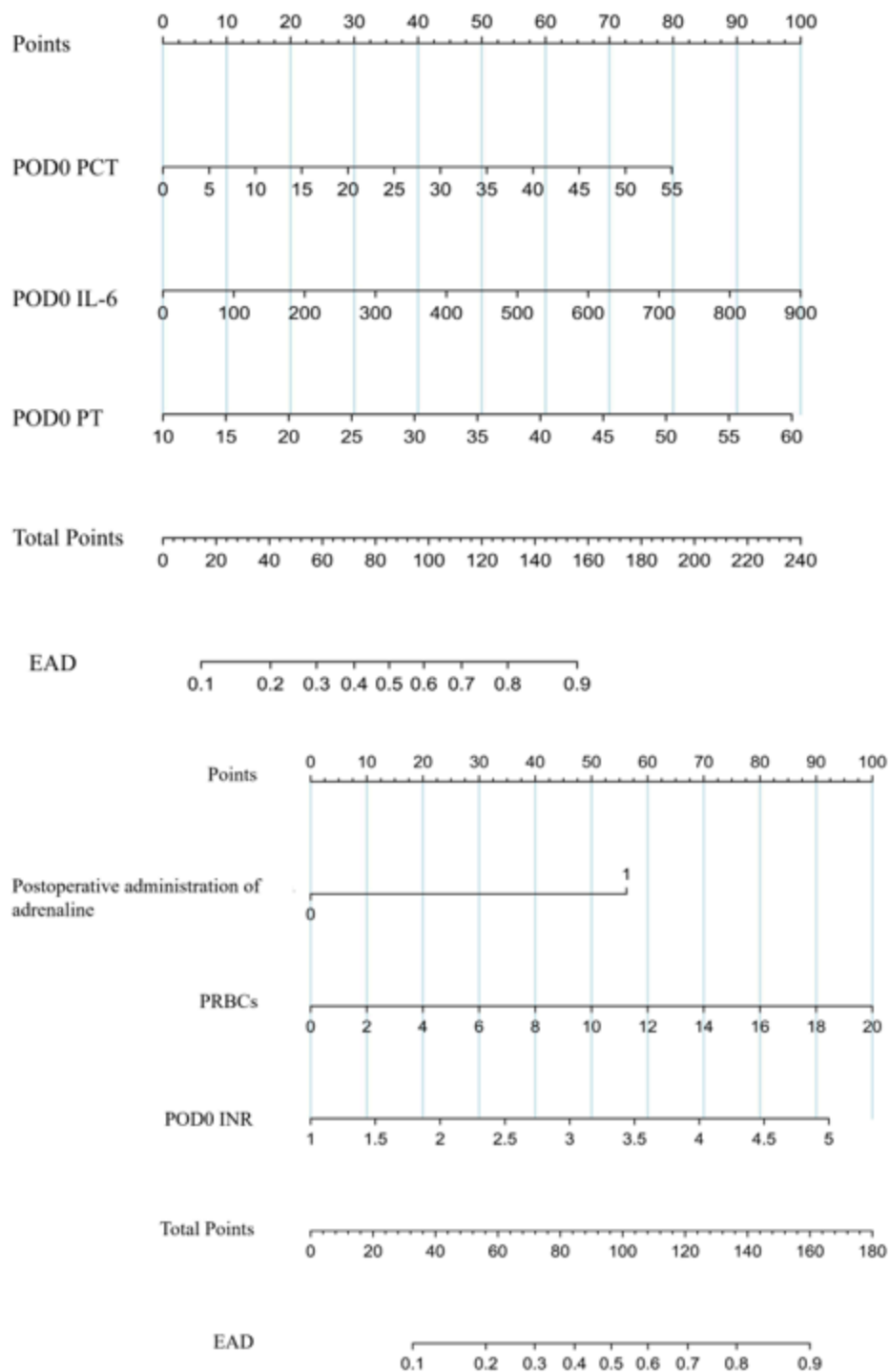


Figure 2

A: Nomogram for predicting EAD following OLT; B: Nomogram for predicting EAD following ELRA

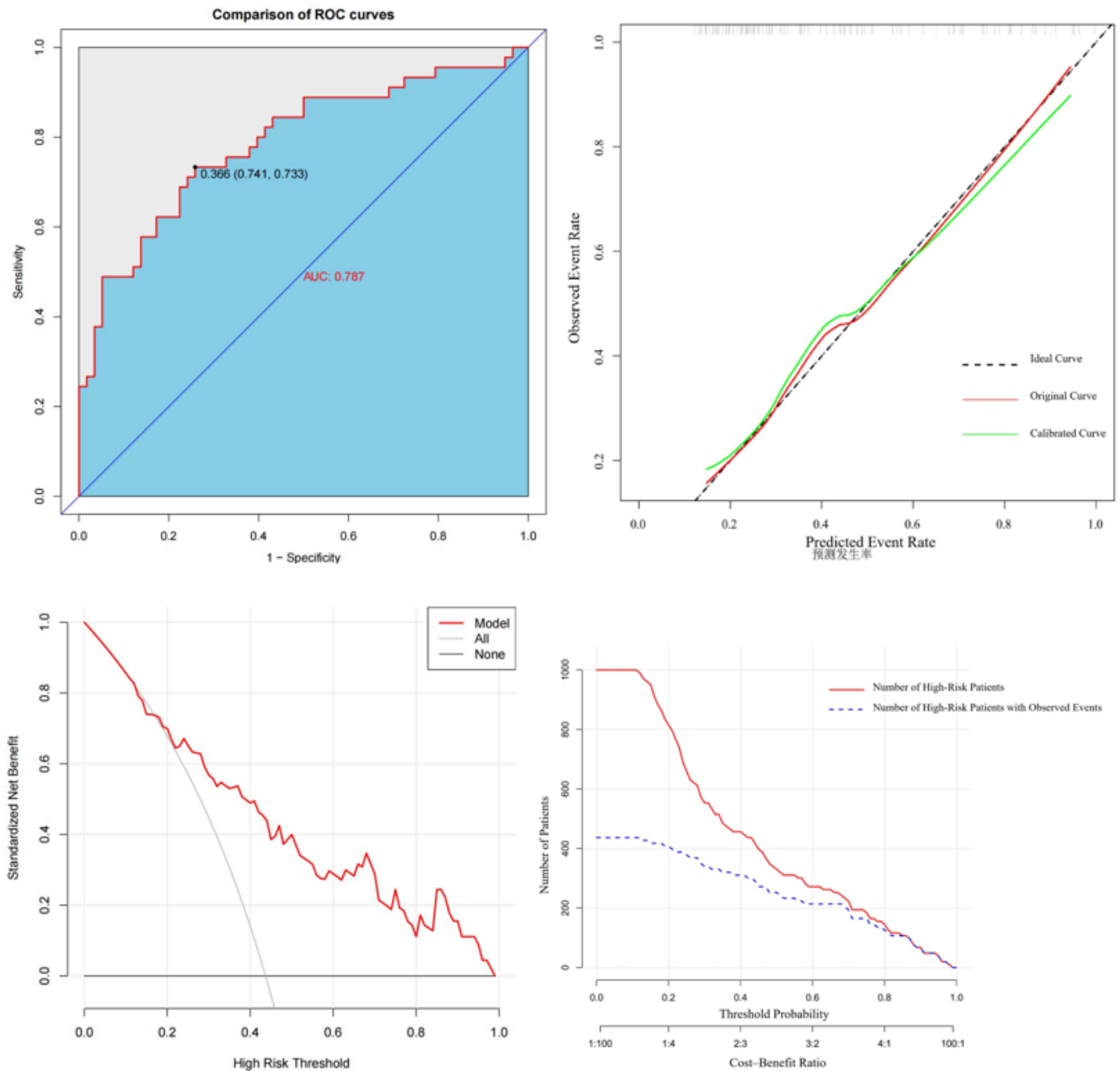


Figure 3

A: ROC curve of the EAD prediction nomogram following OLT; B: Calibration curve of the EAD prediction nomogram following OLT; C: Decision curve analysis (DCA) of the EAD prediction nomogram following OLT; D: CIC of the EAD prediction model after OLA

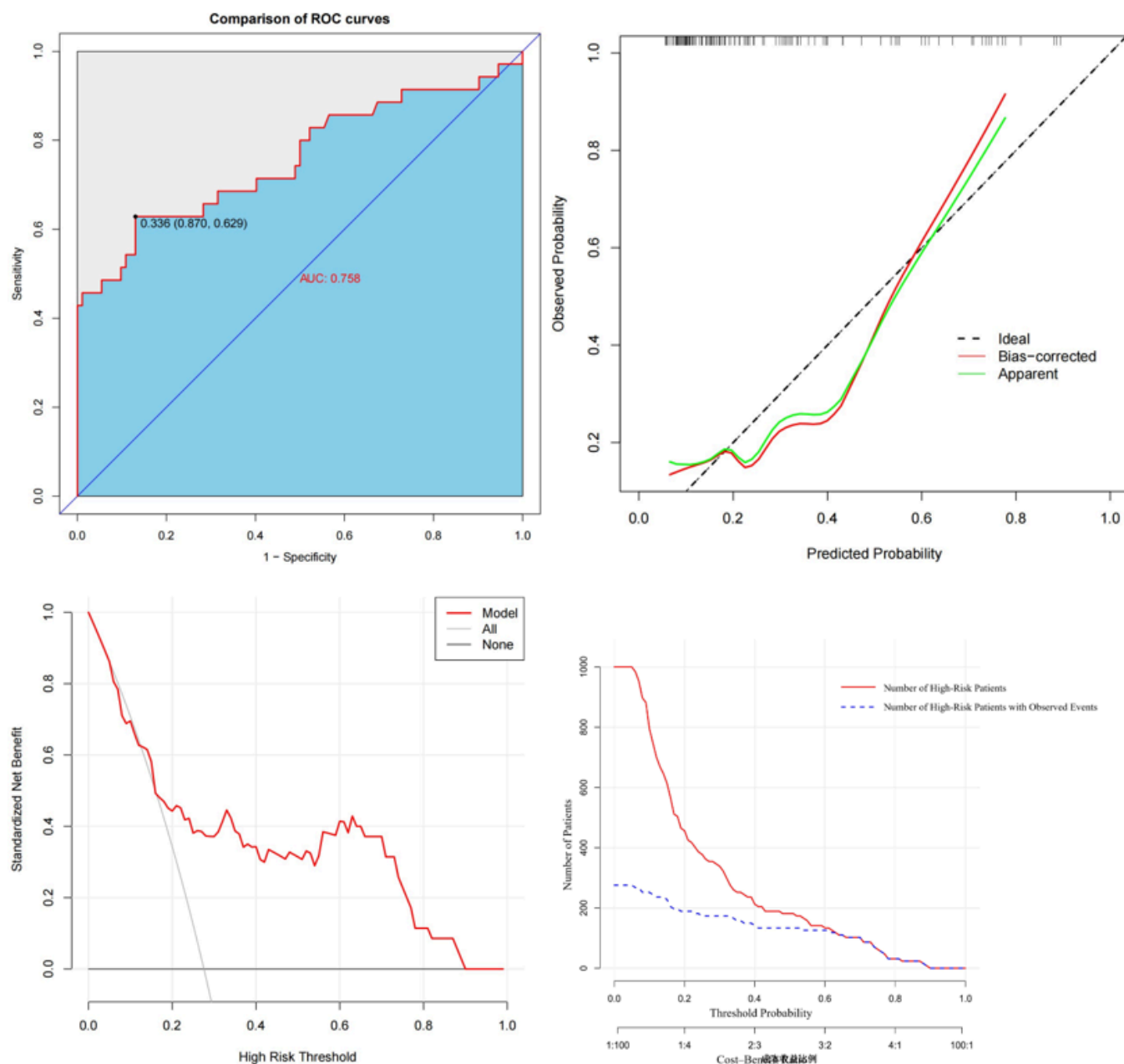


Figure 4

A: ROC curve of the EAD prediction nomogram following ELRA; B: Calibration curve of the EAD prediction nomogram following ELRA; C: Decision curve analysis (DCA) of the EAD prediction nomogram following ELRA; D: CIC of the EAD prediction model after ELRA

Supplementary Files

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