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Research article

Establishment and validation of a nomogram model for prediction of clinical outcomes in patients with amanita phalloides poisoning

Sicheng Zhang [a,1], Maiying Fan [b,c,1], Yiyuan Zhang [b], Shumei Li [a,b], Congyu Lu [b], Junhua Zhou [a,**], Lianhong Zou [b,*]

[a] The First Affiliated Hospital of Hunan Normal University (Hunan Provincial People's Hospital), Changsha, Hunan, 410005, PR China
[b] Department of Emergency Medicine, Hunan Provincial People's Hospital (The First Affiliated Hospital of Hunan Normal University), Changsha, Hunan, 410005, PR China
[c] Clinical Research Center for Emergency and Critical Care in Hunan Province, Changsha, Hunan, 410005, PR China

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ABSTRACT

Amanita phalloides poisoning, renowned for its high mortality rates, is one of the most serious food safety issue in certain regions worldwide. Assessment of prognosis and development of more efficacious therapeutic strategies are critical importance for amanita phalloides poisoning patients. The aim of the study is to establish a nomogram to predict the clinical outcome of amanita phalloides poisoning patients based on the independent risk factor for prognosis. Herein, between January 2013 and September 2023, a cohort of 149 patients diagnosed with amanita phalloides poisoning was enrolled and randomly allocated into training and validation cohorts, comprising 102 and 47 patients, respectively. Multivariate logistic regression analysis was performed to identify the independent risk factors for morality of amanita phalloides poisoning patients in training cohort. Subsequently, a nomogram model was constructed to visually display the risk prediction model. The predictive accuracy of nomogram was verified by the validation cohort. The C index, the area under the receiver operating characteristic curve (AUC), and calibration plots were used to assessed the performance of nomogram. The clinical utility was evaluated by decision curve analysis (DCA). In the present study, the results showed that hepatic encephalopathy (HE), upper gastrointestinal bleeding (UGB), AST, and PT were the independent risk factors associated with the mortality of amantia phalloides poisoning patients. We constructed a new nomogram to evaluate the probability of death induced by amantia phalloides poisoning. The AUC for the prediction accuracy of the nomogram was 0.936 for the training cohort and 0.929 for the validation cohort. The calibration curves showed that the predicted probability matched the actual likelihood. The results of the DCA suggested that the nomogram has a good potential for clinical application. In summary, we developed a new nomogram to assess the probability of mortality for amanita phalloides poisoning patients. This nomogram might facilitate clinicians in making more efficacious treatment strategies for patients with amanita phalloides poisoning.

* Corresponding author.
** Corresponding author.
E-mail addresses: zhoujunhua@hunnu.edu.cn (J. Zhou), zoulh1986@hunnu.edu.cn (L. Zou).
[1] These authors contributed to the work equally.

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1. Introduction

Mushrooms are the fruiting bodies of fungi. There are approximately 16000 known species of mushroom throughout the world [1]. More than 4000 known mushroom species are estimated in China [2], among which over 400 species are poisonous mushrooms. Accidental ingestion of wild poisonous mushroom is the common cause of mushroom poisoning. Investigation through National Poison Data System from 1999 to 2016 shows that 7428 cases of mushroom poisoning were reported annually in the United States [3]. A total of 10036 mushroom poisoning outbreaks which leaded to 38676 illness, 21697 hospitalizations and 788 deaths were reported to the Foodborne Disease Outbreak Surveillance System in China during 2010–2020 [4]. The overall mortality rate of mushroom poisoning is about 1.4 % [5].

Amanita phalloides, also known as the "death cap", is responsible for more than 90 % of deaths related to mushroom poisoning. The mortality rate after amanita phalloides poisoning ranges from 10 to 20 % [6]. The clinical symptoms of amanita phalloides poisoning go through four stages [7,8]. These stages include the asymptomatic phase, gastrointestinal reaction phase, false recovery stage, and the most severe stage characterized by hepatic function deterioration. Lethal amanita phalloides contains three main types of toxins: phallotoxins, amatoxins and virotoxins [9]. The phallotoxins mainly cause injury of the cellular membrane and cytoskeleton [10]. The damage of liver and renal is mainly caused by amatoxins. As the most potent and abundant amatoxins, the lethal dose of alpha-amanitin is reported to be 0.1 mg/kg B W. for adult after oral administration [11]. The molecular mechanism of amatoxins is non-covalently bound RNA polymerase-II, which inhibits the transcriptional activity of RNA polymerase-II, resulting in progressive reduction of mRNA and protein synthesis in cells, especially liver cells, and eventually cell death [12]. The current therapeutic strategies for amanita phalloides poisoning mainly include volume replacement therapy, toxins binding (eg. Hemodialysis, activated charcoal), antidote therapy (eg. Penicillin G, silibinin and N-acetylcysteine), treatment for liver failure (eg. Liver transplantation). Nevertheless, the prognosis of patients with amanita phalloides poisoning is still unsatisfactory. Late and incorrect diagnosis, ineffective therapeutic strategies are the most important causes of treatment failure for amanita phalloides poisoning patients. We have previously screened serum metabolic markers for the early diagnosis of amatoxin poisoning patients by metabolomics, which provides a basis for early intervention [13]. Establishing a prognostic model for predicting outcomes in patients with amanita poisoning is imperative for the development of efficacious therapeutic interventions. Regrettably, the existing literature is scant on prognostic evaluation in patients with amanita phallodies poisoning [14]. A study based on the XGBoost algorithm helped to distinguish critically ill patients with mushroom poisoning. There is a pressing requirement for the development of an accessible and efficient prognostic assessment model for patients with amantia phalloides poisoning to enhance the optimization of therapeutic strategies.

Nomogram is considered to be an effective tool for visualizing regression models used to quantify individual probability of clinical events by taking important prognostic factors into account, and has been widely used in predicting the clinical prognosis of patients with type 2 diabetes [15], low-grade endometrial stromal sarcoma [16] and gastric cancer [17]. In the present study, we screened the risk factors for mortality in amantia phalloides poisoning patients, and established a nomogram to predict the clinical outcomes of amanita phalloides poisoning patients based on the independent risk factor for prognosis. In addition, the predictive ability of nomogram and its clinical application were validated.

2. Materials and methods

2.1. Subjects

In this retrospective single center study, we enrolled 267 patients with mushroom poisoning at Hunan Provincial People's Hospital (the First Affiliated Hospital of Hunan Normal University) between January 2013 and September 2023. The study cohort comprised hospitalized individuals who received a confirmed diagnosis of amanita phalloides poisoning upon admission. The amanita phalloides poisoning was diagnosed by macroscopic identification by a qualified mushroom expert or detection of amatoxins. The exclusion criteria were as follows: (1) patients without hospitalization; (2) patients with incomplete information on clinical features and biochemical indicators; (3) patients who consumed non-Amanita species poisoning; (4) patients with primary liver disease. According to these criteria, 149 amanita phalloides poisoning patients were selected for model establishment. Patients were randomly divided into a training cohort (n = 102) and a validation cohort (n = 47) in a ratio of 7:3. The study was performed in accordance with the Declaration of Helsinki (revised in Fortaleza, Brazil, 2013) and approved by the Medical Ethics Committee of the Hunan Provincial People's Hospital (IRB Approval No.: [2024]-10).

2.2. Management of patients with amantia phallodies poisoning

The overarching objective of effective treatment for amanita phalloides poisoning is to mitigate the severity of toxin-induced pathophysiological processes by facilitating detoxification prior to the accumulation of toxins within the systemic circulation or exacerbation of their detrimental effects. In the present study, the patients with amanita phalloides poisoning were managed with the following treatment: intensive and supportive measures (resuscitation fluids); minimizing the absorption of amatoxins and phallotoxins (oral administration of activated charcoal 5 g, 6 times per day for the first 3 days); elimination of absorbed amatoxins; drug therapy (high dose intravenous acetylcysteine 120 mg/kg/day for 5–7 day; intravenous penicillin G 300,000 IU/kg/day for 5 days; oral administration silymarin 1400 mg thrice daily for 5–7 days). In addition, blood purification therapy was performed for treatment of amanita phalloides poisoning. Patients arrived within 24 h after amanita phalloides mushroom ingestion were treated with hemoperfusion. The abnormal levels of TBIL, ALT and AST were used to diagnose the liver disfunction of the patients. Plasmapheresis was

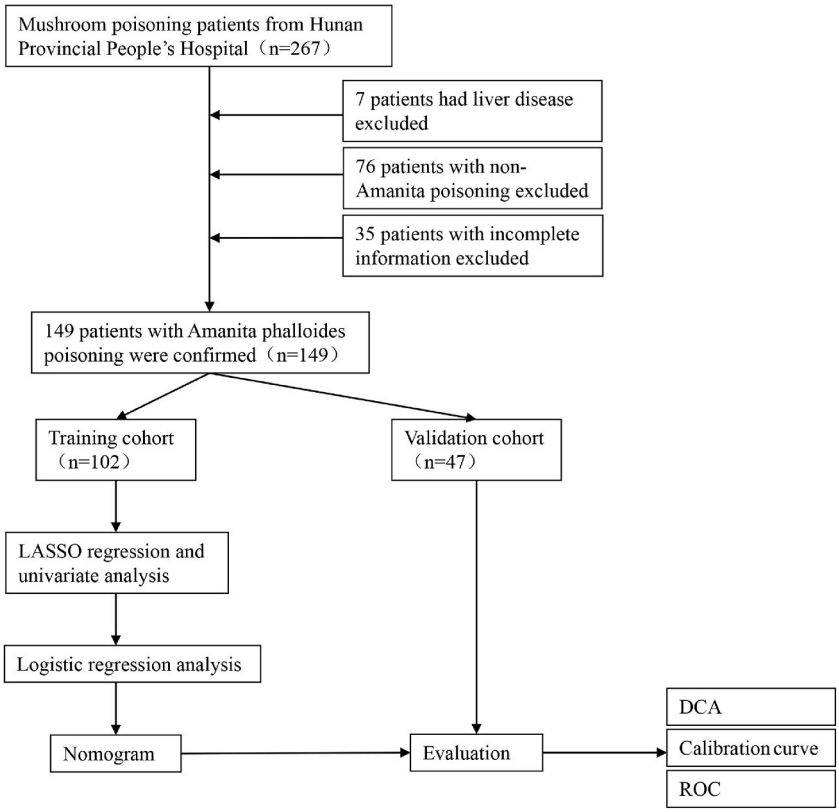

Fig. 1. Flowchart of the included and excluded participants.

carried out for patients with deteriorating liver function due to amanita phalloides poisoning.

2.3. Date collection

The demographic and clinical data were abstracted from patient's medical records. The baseline characteristics including age, gender, incubation period, history of diabetes and coronary disease, hypertension were obtained. Clinical symptoms including vomiting, UGB, dizziness and headache, Chest tightness and fatigue, fever, HE, oliguria anuria, jaundice, anemia, dyspnea were collected. Laboratory serological indexes (ALT, AST, TBIL, Cr, LDH, PT, APTT) were also obtained.

2.4. Statistical analysis

SPSS 22.0 statistical software package was used for statistical analysis. Continuous variables were expressed as means and standard deviations (SD), and categorical variables were expressed as frequencies and percentages. Statistical differences were compared using the chi-square test, corrected chi-square, or Fisher test for categorical variables. The continuous variables were compared by the independent two-sample *t*-test or Mann-Whitney *U* test, and $P < 0.05$ was considered as a significant difference. Least Absolute Shrinkage and Selection Operator (LASSO) regression was performed using the "glmnet" package to screen out relevant variables. The potential predictors subsets of risk of mortality were screened by LASSO regression and univariate analysis, and the independent risk factors by using multivariate logistic regression analysis. Nomogram models were constructed using the "rms", "Nomogram Formula" and "pROC" packages in R software to calculate the risk of mortality for each patient. The predictive ability of the nomogram was evaluated using the receiver operating characteristic (ROC) curves and calibration curves. Decision Curve Analysis (DCA) was preformed using "Decision Curve" R package to further assess the clinical utility and clinical benefit of the nomogram when it was adapted to support the clinical practice.

3. Results

3.1. Clinical characteristics of amantia phalloides poisoning patients

A total of 267 cases of mushroom poisoning were initially collected. Subsequently, individuals with incomplete data (n = 35), pre-existing liver disease (n = 7), and non-amanita phalloides poisoning (n = 76) were excluded. Consequently, a corhort of 149 patients

Table 1
Baseline characteristics in the training and validation cohorts.

Characteristics	Training Cohort (102)	Validation Cohort (47)	*P* value
Age (%)			0.884
< 65 years	77 (75.5)	36 (76.6)	
≥ 65 years	25 (24.5)	11 (23.4)	
Gender (%)			0.662
Male	46 (44.1)	23 (48.9)	
Female	56(55.9)	24 (51.1)	
Hypertension (%)			0.374
No	83 (81.4)	41 (87.2)	
Yes	19 (18.6)	6 (12.8)	
Diabetes (%)			0.302[a]
No	88 (86.3)	44 (93.6)	
Yes	14 (13.7)	3 (6.4)	
Coronary heart disease (%)			0.803[a]
No	94 (92.2)	42 (89.4)	
Yes	8 (7.8)	5 (10.6)	
Incubation period (h)	10.9 ± 8.3	8.9 ± 3.9	0.09
Vomit (%)			0.234[b]
No	1 (1)	2 (4.3)	
Yes	101 (99)	45 (95.7)	
UGB (%)			0.721
No	85 (83.3)	40 (85.1)	
Yes	17 (16.7)	7 (14.9)	
Dizziness (%)			0.932
No	68 (66.7)	31 (66)	
Yes	54 (33.3)	16 (34)	
Chest tightness and fatigue (%)			0.787
No	48 (47.1)	21 (44.7)	
Yes	54 (52.9)	26 (55.3)	
Fever (%)			0.985
No	96 (94.1)	45 (95.7)	
Yes	6 (5.9)	2 (4.3)	
HE (%)			0.486
No	89 (87.3)	39 (83)	
Yes	13 (12.7)	8 (17)	
Oliguria (%)			0.399
No	69 (67.6)	35 (74.5)	
Yes	33 (32.4)	12 (25.5)	
Jaundice (%)			0.265
No	84 (82.4)	35 (74.5)	
Yes	18 (17.6)	12 (25.5)	
Palpitation (%)			0.862
No	90 (88.2)	41 (87.2)	
Yes	12 (11.8)	6 (12.8)	
Anaemia(%)			0.127
No	83 (81.4)	33 (70.2)	
Yes	19 (18.6)	14 (29.8)	
Dyspnea (%)			0.209
No	92 (90.2)	39 (83)	
Yes	10 (9.8)	8 (17)	
Blood transfusion (%)			0.483
No	71 (69.6)	30 (63.8)	
Yes	31 (30.4)	17 (36.2)	
Hemoperfusion (%)			0.787
No	48 (47.1)	21 (44.7)	
Yes	54 (52.9)	26 (55.3)	
Hemodialysis (%)			0.293
No	58 (56.9)	31 (66)	
Yes	44 (43.1)	16 (34)	
ALT (%)			0.699
< 400 U/L	62 (60.8)	27 (57.4)	
≥ 400 U/L	40 (39.2)	20 (42.7)	
AST (%)			0.964
< 400 U/L	59 (57.8)	27 (57.4)	
≥ 400 U/L	43 (42.3)	20 (42.6)	
TBIL (μmol/L)	38.8 ± 46.1	34.6 ± 28.1	0.573
CR (μmol/L)	268.5 ± 289.6	208.3 ± 2 93.7	0.243
LDH (U/L)	1091.9 ± 869.1	1402.7 ± 1626.1	0.131

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Table 1 (*continued*)

Characteristics	Training Cohort (102)	Validation Cohort (47)	*P* value
PT (%)			0.094
< 20 s	84 (82.4)	33 (70.2)	
≥ 20 s	18 (17.6)	14 (29.8)	
APTT(sec)	46.5 ± 28.8	46.6 ± 26.6	0.980

[a] Correction chi-square.
[b] fisher test.

were enrolled in the study (Fig. 1). We randomly divided the 149 patients into a training cohort (n = 102, 70 %) and a validation cohort (n = 47, 30 %). The baseline of clinical characteristics of amantia phalloides poisoning patients were shown in Table 1. Most of patients with amantia phalloides poisoning were less than 65 years old (75.5 % and 76.6 % in training and validation cohorts respectively). The proportion of male patients with amantia phalloides poisoning in training cohort was 44.1 %, and that in validation cohort was 48.9 %. There were no significant difference in clinical characteristics observed between patients with amantia phalloides poisoning in training and validation cohorts, indicating that randomization was successful and did not introduce bias (Table 1). The results of the univariate analysis in training cohort showed that UGB, Chest tightness and fatigue, HE, jaundice, dyspnea, blood transfusion, ALT, AST, Cr, PT and APTT were significantly different between the survival group and death group (Table 2).

Next, the LASSO regression model was established to select potential predictors in the training cohort (Fig. 2). Fig. 2A displayed the change of the regression coefficient of each independent variable under different λ. λ.min = 0.0380 was selected as the optimal value (Fig. 2B). APTT, Cr, ALT, dyspnea, jaundice were excluded based on LASSO regression. As a result, variables including UGB, chest tightness and fatigue, HE, blood transfusion, AST, PT were selected for further multivariate logistic regression analysis.

3.2. Identification of independent risk factor for mortality

Following univariate analysis and lasso regression, the candidate variables were selected for inclusion in the subsequent multivariate logistic regression analysis. Based on backward stepwise elimination, HE (OR, 49.05; 95 % CI, 4.848–496.413; $P = 0.001$), UGB (OR, 16.28; 95 % CI, 2.149–123.39; $P = 0.007$), AST (OR, 6.50; 95 % CI, 1.060–39.868; $P = 0.043$), and PT (OR, 8.297; 95 % CI, 1.275–53.981; $P = 0.027$) were the independent risk factors for mortality of amantia poisoning patients (Table 3).

3.3. Establishment of a nomogram to predict the mortality risk for patients with amantia poisoning

Subsequently, a nomogram was constructed based on the outcomes of multivariate logistic regression analysis (Fig. 3). A quantitative method also was made accessible to predict the probability of death for patients with amantia poisoning. Each amantia poisoning patients was given a score for each independent risk factors for mortality. The results showed that HE and UGB corresponded to higher risk scores (100 and 76 points respectively), followed by AST (>400 U/L) and PT (>20 s) with the risk scores of 47 and 54 points respectively. The individual mortality risk was calculated according to the total scores of four risk factors. The higher total scores signified that the higher possibility of death for the patients with amantia poisoning.

3.4. Validation of the nomogram to predict the mortality risk for amantia poisoning patients

The predictive performace of nomograms was evaluated through the receiver operating characteristic (ROC) curve analysis. The results showed that the AUC was 0.936 (95 % CI = 0.863–0.999) for the training cohort (Fig. 4A) and 0.929 (95 % CI = 0.814–0.999) for the validation cohort (Fig. 4B), indicating a high level of prediction accuracy for the nomograms. Next, the calibration curve plot was used to evaluate the deviation between the predicted and observed values of nomogram. The results showed that the predicted value was excellently correlated with the observed value in both training cohort and validation cohort (Fig. 4C and D). In addition, Decision curve analysis (DCA) were used to evaluate the clinical applicability of the nomograms. The nomogram for predicting the mortality risk for amantia poisoning was more useful than the non-intervation and all-intervention methods when the threshold probabilities ranged from 0 % to 100 % in the training cohort (Fig. 4E). Decision curve analysis for the validation cohort show that using this nomogram to predict mortality risk of patients with amantia poisoning more benefit than intervening with an all-patient regimen or a no-intervention regimen when the threshold probabilities for patients and physicians are >0.3 % and <95 %, respectively (Fig. 4F). These findings indicate that nomograms exhibit exceptional predictive capability for estimating the mortality risk among patients with amantia phalloides poisoning.

4. Discussion

Amanita phalloides poisoning represents a significant global food safety concern. It is widely recognized that early prognosis prediction plays a pivotal role in formulating an appropriate therapy strategy, reducing the mortality and enhancing the treatment efficacy. Nomogram, as a dependable statistical tool, is extensively utilized for estimating the probability of clinical outcomes including mortality [18]. In the present study, we testified that HE (OR, 49.05; 95 % CI, 4.848–496.413; $P = 0.001$), UGB (OR, 16.28;

Table 2
Characteristics and univariate statistics of the dead and surviving patients with amanita phalloides poisoning in the training cohort.

characteristics	Training Cohort(n = 102)		*P* value
	Death (n = 21)	Survival (n = 81)	
Age (%)			0.104
< 65 years	13 (61.9)	64 (79)	
≥ 65 years	8 (38.1)	17 (21)	
Gender (%)			0.794
Male	10 (47.6)	36 (44.4)	
Female	11 (52.4)	45 (55.6)	
Hypertension (%)			0.999
No	17 (81)	66 (81.5)	
Yes	4 (19)	15 (18.5)	
Diabetes (%)			0.66[a]
No	17 (81)	71 (87.7)	
Yes	4 (19)	10 (12.3)	
Coronary heart disease (%)			0.437[a]
No	18 (85.7)	76 (93.8)	
Yes	3 (14.3)	5 (6.2)	
Incubation period (h)	10.9 ± 5.6	11 ± 8.8	0.983
Vomit (%)			0.206[b]
No	1 (4.8)	0 (0)	
Yes	20 (95.2)	81 (100)	
UGB (%)			<0.001
No	10 (47.6)	75 (92.6)	
Yes	11(53.4)	6 (7.4)	
Dizziness (%)			0.119
No	11 (52.4)	57 (70.4)	
Yes	10 (47.6)	24 (19.6)	
Chest tightness and fatigue (%)			0.001
No	3 (14.3)	45 (55.6)	
Yes	18 (85.7)	36 (44.4)	
Fever (%)			0.10b
No	18 (85.7)	78 (96.3)	
Yes	3 (14.3)	3 (3.7)	
HE (%)			<0.001
No	10 (47.6)	79 (97.5)	
Yes	11 (52.4)	2 (2.5)	
Oliguria (%)			0.582
No	13 (61.9)	56 (69.1)	
Yes	8 (38.1)	25 (30.9)	
Jaundice (%)			<0.001
No	11 (52.4)	73 (90.1)	
Yes	10 (48.6)	8 (9.9)	
Palpitation (%)			0.982[a]
No	18 (85.7)	72 (88.9)	
Yes	3 (14.3)	9 (11.1)	
Anaemia (%)			0.189
No	15 (71.4)	68 (84)	
Yes	6 (28.6)	13 (16)	
Dyspnea (%)			<0.001
No	14 (66.7)	78 (96.3)	
Yes	7 (33.3)	3 (3.7)	
Blood transfusion (%)			<0.001
No	8 (38.1)	63 (77.8)	
Yes	13 (61.9)	18 (22.2)	
Hemoperfusion (%)			0.157
No	7 (33.3)	41 (50.6)	
Yes	14 (66.7)	40 (49.4)	
Hemodialysis (%)			0.601
No	13 (61.9)	45 (55.6)	
Yes	8 (38.1)	36 (44.4)	
ALT (%)			0.017
< 400 U/L	8 (38.1)	54 (59.2)	
≥ 400 U/L	13 (61.9)	27 (40.8)	
AST (%)			<0.001
< 400 U/L	4 (19)	55 (67.9)	
≥ 400 U/L	17 (81)	26 (32.1)	
TBIL (μmol/L)	47.2 ± 38.4	36.6 ± 47.8	0.348
CR (μmol/L)	161.1 ± 136.3	296.3 ± 312.2	0.004
LDH (U/L)	1381 ± 1001.4	1017 ± 821.8	0.136

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Table 2 (*continued*)

characteristics	Training Cohort(n = 102)		*P* value
	Death (n = 21)	Survival (n = 81)	
PT (%)			<0.001
< 20 s	11 (52.4)	73 (90.1)	
≥ 20 s	10 (47.6)	8 (9.9)	
APTT(sec)	57.9 ± 32.8	43.5 ± 27.1	0.041

[a] Correction chi-square.
[b] fisher test.

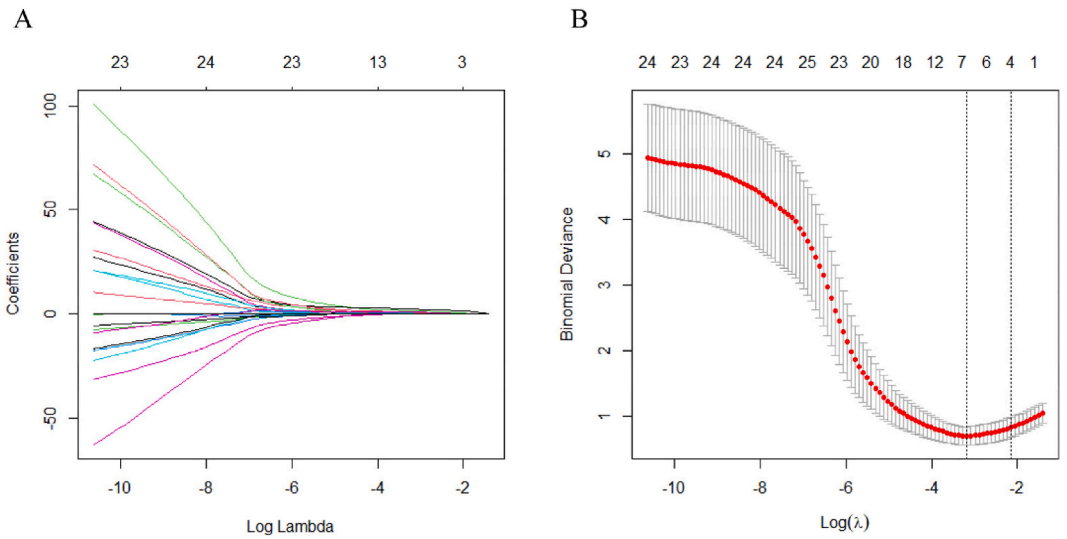

Fig. 2. Risk factor selection using LASSO regression analysis. (**A**) Lasso coefficient profiles of the features. (**B**) Log(lambda) and partial likelihood deviations are shown, and the dotted line shown at the smallest log(lambda) represents the best predictor.

Table 3
Multivariate analysis of mortality risk factors for patients with amanita phalloides poisoning in the training cohort.

variables	β	*P*	OR	95%CI
UGB	2.790	0.007	16.28	2.149–123.39
Chest tightness	1.187	0.230	3.276	0.473–22.706
HE	3.893	0.001	49.05	4.848–496.413
blood transfusion	0.513	0.534	1.670	0.332–8.408
AST	1.872	0.043	6.500	1.060–39.868
PT	2116	0.027	8.297	1.275–53.981
Constant	−5.463	0.001	0.04	

95 % CI, 2.149–123.39; $P = 0.007$), AST (OR, 6.50; 95 % CI, 1.060–39.868; $P = 0.043$), and PT (OR, 8.297; 95 % CI, 1.275–53.981; $P = 0.027$) were the independent risk factors for mortality of amantia phalloides poisoning patients. Furthermore, we established and validate a nomogram to predict the mortality risk for amantia phalloides poisoning patients.

The mortality rates among patients with amanita phalloides poisoning exhibit variability across different countries and regions, attributable to disparities in medical expertise, healthcare infrastructure and levels of health education. The mortality rate for amanita phalloides poisoning patients in the United States is about 8.3–8.8 % [19]. A study on mushroom poisoning from southern China revealed a mortality rate of 21.5 % among individuals with amantia phalloides poisoning [20]. In line with this, our study revealed that the mortality rate of patients with amanita phalloides poisoning was 19.46 %.

The previous studies revealed that the amantia poisoning patients with high level of AST, PT, APTT, TBIL, Cr and HE were more likely to have an unfavorable prognosis [21–25]. Consistent with this, our findings demonstrated that ALT (>400 U/L)、AST (>400 U/L), PT(>20 s), Cr, HE were the risk factors for mortality in patients with amantia phallodies poisoning. Additionally, our study uniquely identifies UGB, chest tightness and fatigue, jaundice and dyspnoea as factors associated with adverse clinical outcomes in individuals with amantia phallodies poisoning. The amatoxins originated from amantia phalloides are heavily enriched in the liver via the OATP1B3 transporter, reduces protein synthesis through inhibition of RNA polymerase II activity, ultimately leading to hepatocyte death [26]. ALT and AST, which were responsible for amino acid catabolism and ATP production, were the indications of liver injury.

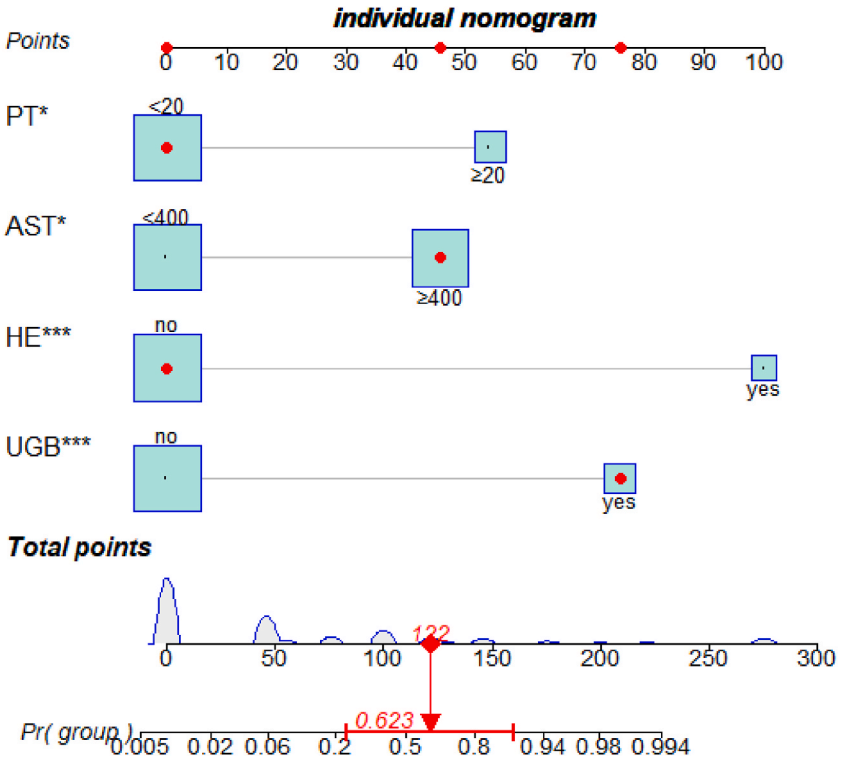

Fig. 3. A Nomogram was created to predict the probability of death for amanita phalloides poisoning patients. A total score was obtained from the values of each index, and the mortality risk of amantia poisoning patients corresponding to the total score was the predicted rate of the nomogram.

Prothrombin time (PT), a standard test for assessing exogenous coagulation factors, serves as a proxy for the activity of essential plasma coagulation factors (II, V, VII, X) [27], that are predominantly synthesised by hepatocytes. Additionally, PT is recognized as a valuable indicator of hepatic dysfunction. In current study, individuals with underlying primary hepatic conditions were excluded from the participant corhort. Therefore, abnormal levels of AST, ALT and PT may be considered as indicative markers of acute liver injury resulting from amantia phalloides poisoning. Furthermore, we also found that AST (>400 U/L) and PT (>20 s) were independent mortality risk factors for death of amantia poisoning patients and could be used as a predictor of poor prognosis, which was in agreement with Fantozzi R et al. [22]. HE is recognized as a severe complications of end-stage liver disease. Sever hepatocyte dysfunction caused by amantia phalloides poisoning can easily progress to acute liver failure which is characterized by coagulation abnormalities and HE. Normally, as a by-product of protein metabolism, ammonia is converted to urea by liver. Actue liver failure induced by amantia phalloides poisoning disordered the metabolism of ammonia [28]. The accumulation of plasma ammonia is neurotoxic and a marker for HE [29]. In line with our results, the previous studies reported that peak plasma ammonia 95.1 > μmol/L and HE were the independent risk factors for death in amanita phalloides poisoning [30]. Moreover, HE is often accompanied by gastrointestinal bleeding. In addition, the coagulation disturbances and acute stress response may contribute to the development of UGB. Acute amantia poisoning can trigger an acute stress response. Consistent with our invertigation, prior research has suggested that coagulation abnormalities linked to hepatic function are predictive of adverse clinical outcomes in patients with amantia phallodies poisoning. Therefore, UGB serves as a critically marker of severe illness in patients with amanita phalloides poisoning. Our current study identified UGB as an independent risk factor for mortality in amanita phallodies poisoning patients. Collectively, these findings indicate a correlation between hepatocyte injury and unfavorable clinical outcome in amantia phallodies poisoning.

Nomogram is a convenient and reliable tool to quantify individual risk of clinical outcome by integrating multiple important prognostic factors. It is worth noting that nomogram offer a visual representation that transform the traditional statistical models, and has been extensively utilized in prediction of incidence and prognosis across a variety of diseases [31–33]. However, there is a paucity of reports utilizing nomogram for predicting mortality in patients with amantia phallodies poisoning. In the present study, we have contructed a novel nomogram to evaluate the morality probability in patients with amanita phalloides poisoning based on the independent risk factors. ROC curves and calibration curves testified the accuracy of nomogram in both the training and validation cohorts. In addition, DCA confirmed that the constructed nomogram had a good value of clinical application. Overall, our nomogram model can serve as a valuable tool for clinicians to predict the probability of death in patients with amantia phallodies poisoning and guide early intervention to improve prognosis.

However, our study is subject to several significant limitations. Firstly, due to the inherent flaws of the retrospective study, some potential information bias is inevitable. Furthermore, variations in detection instruments and index parameters may constrain the generalisability and applicability of our nomogram. Lastly, the nomogram prediction model for mortality risk of patients with amantia

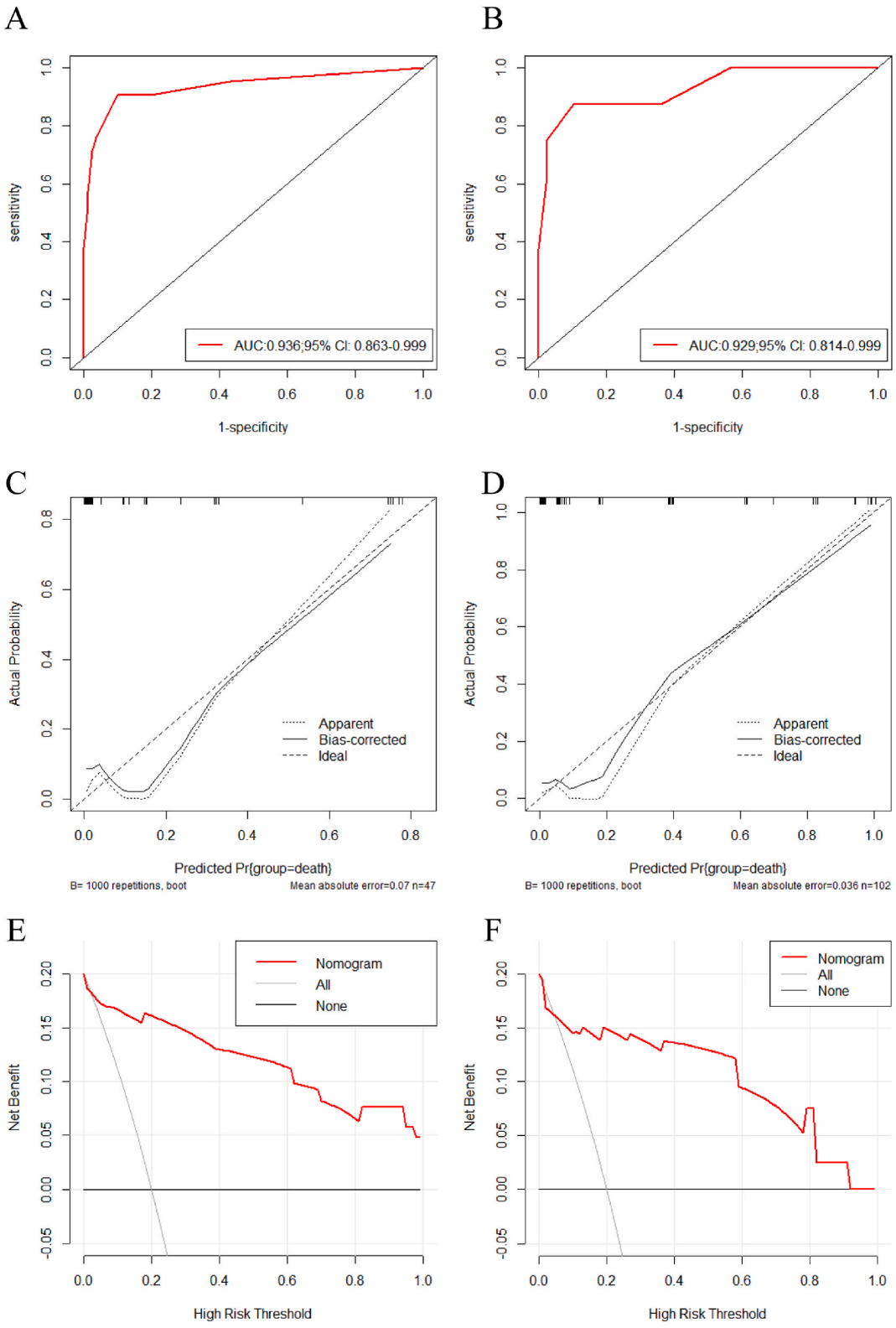

Fig. 4. Assessment of the predictive ability and clinical utility of the nomogram for predicting mortality risk of patients with amantia poisoning patients. The ROC curves was performed to detect the discriminative ability of the nomogram in the training (**A**) and validation (**B**) cohorts. The calibration curves of the nomogram for death risk prediction of amantia poisoning patients in the training (**C**) and validation (**D**) cohorts. Decision curve analysis was performed to evaluated the clinical applicability of the nomograms in the training (**E**) and validation (**F**) cohorts.

phallodies poisoning was internally validated, in the future, we should collect an external validation cohort to evaluate the nomogram model.

5. Conclusions

In conclusion, our study demonstrated that HE, UGB, AST, and PT were the independent risk factors for the mortality of amantia phallodies poisoning patients. Furthermore, we developed a novel nomogram to estimated the mortality probability for amanita phalloides poisoning patients, which might facilitate clinicians in making more efficacious treatment strategies for patients with amanita phalloides poisoning.

Ethical statement

The study was reviewed and approved by the Medical Ethics Committee of the Hunan Provincial People's Hospital (IRB Approval No.: [2024]-10).

Data availability statement

Data will be made available on request.

Additional information

No additional information is available for this paper.

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CRediT authorship contribution statement

Sicheng Zhang: Writing – review & editing, Writing – original draft, Formal analysis, Data curation. **Maiying Fan:** Writing – original draft, Resources, Investigation, Formal analysis, Data curation. **Yiyuan Zhang:** Investigation, Formal analysis. **Shumei Li:** Investigation, Data curation. **Congyu Lu:** Investigation, Data curation. **Junhua Zhou:** Writing – review & editing, Visualization, Supervision. **Lianhong Zou:** Writing – review & editing, Writing – original draft, Validation, Supervision, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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