BMJ Health & Care Informatics

Development of an explainable prediction model for portal vein system thrombosis post-splenectomy in patients with cirrhosis

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To cite: Qu D, Dai D, Li G, *et al*. Development of an explainable prediction model for portal vein system thrombosis post-splenectomy in patients with cirrhosis. *BMJ Health Care Inform* 2025;**32**:e101319. doi:10.1136/bmjhci-2024-101319

► Additional supplemental material is published online only. To view, please visit the journal online (https://doi.org/10.1136/bmjhci-2024-101319).

Received 17 October 2024
Accepted 23 March 2025

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ABSTRACT

Background Portal vein system thrombosis (PVST) is a common and potentially life-threatening complication following splenectomy plus pericardial devascularisation (SPDV) in patients with cirrhosis and portal hypertension. Early prediction of PVST is critical for timely intervention. This study aimed to develop a machine learning-based prediction model for PVST occurrence within 3 months after splenectomy.

Methods 392 patients with cirrhosis who underwent splenectomy at the Second Affiliated Hospital of Xi'an Jiaotong University between 1 July 2016 and 31 December 2022 were enrolled in this study and followed up for 3 months. The predictive model integrated 37 candidate predictors based on accessible clinical data, including demographic characteristics, disease features, imaging results, laboratory values, perioperative details and postoperative prophylactic therapies, and finally, eight predictors were selected for model construction. The five machine learning algorithms (logistic regression, Gaussian Naive Bayes, decision tree, random forest and AdaBoost) were employed to train the predictive models for assessing risks of PVST, which were validated using five fold cross-validation. Model discrimination and calibration were estimated using receiver operating characteristic curves(ROC), accuracy, sensitivity, specificity, positive predictive value, negative predictive value and Brier scores. The outcome of the predictive model was interpreted using SHapley Additive exPlanations (SHAP), which provided insights into the factors influencing PVST risk prediction.

Results During the 3-month follow-up, a total of 144 (36.73%) patients developed PVST. The AdaBoost model demonstrated the highest discriminative ability, with a mean area under the receiver operating characteristic curve (AUROC) of 0.72 (95% CI 0.60 to 0.84). Important features for predicting PVST included albumin, platelet addition, the diameter of the portal vein, γ-glutamyl transferase, length of stay, activated partial thromboplastin time, D-dimer level and history of preoperative gastrointestinal bleeding, as revealed by SHAP analysis.

Conclusions The machine learning-based prediction models can provide an initial assessment of 3-month PVST risk after SPDV in patients with cirrhosis and portal hypertension. The AdaBoost model demonstrates moderate discriminative ability in distinguishing between high-risk and low-risk patients, with an AUROC of 0.72 (95% CI 0.60 to 0.84). By incorporating SHAP analysis, the model can offer transparent explanations for personalised risk predictions, facilitating targeted preventive

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Portal vein system thrombosis (PVST) is a common and potentially life-threatening complication in patients with cirrhosis following splenectomy, yet effective predictive tools to guide early intervention remain limited. Traditional statistical methods often fail to capture the multifactorial and complex nature of PVST, leaving a significant gap in risk assessment and personalised management.

WHAT THIS STUDY ADDS

⇒ This study introduces a machine learning-based prediction model for PVST within 3 months after splenectomy, achieving a moderate area under the receiver operating characteristic curve (AUROC) of 0.72 (95% CI 0.60 to 0.84). By identifying key predictors such as albumin, portal vein diameter and D-dimer levels, and integrating SHapley Additive exPlanations (SHAP), the model provides interpretable insights into personalised risk predictions. This enhances clinical decision-making and facilitates targeted preventive interventions.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ The findings highlight the potential of machine learning to improve risk stratification and personalised care for PVST. The incorporation of SHAP analysis ensures transparency, fostering clinician trust and model adoption. Future research should focus on multicentre validation and the integration of additional data, such as imaging and novel biomarkers, to further refine prediction accuracy and generalisability.

interventions and reducing excessive interventions across the entire patient population.

INTRODUCTION

In China, splenectomy plus pericardial devascularisation (SPDV) has emerged as an effective and feasible surgical intervention for liver cirrhosis with portal hypertension (PHT).[1 2] However, the occurrence of portal vein system thrombosis (PVST) following SPDV represents a common and potentially life-threatening postoperative complication. PVST referred to the blockage or stenosis of the portal vein, splenic vein and superior mesenteric vein (SMV), or intrahepatic portal vein branches by a thrombus. Current data indicate that the incidence of PVST after SPDV in patients with cirrhosis ranges from 21.6% to 44.2%.[3 4] PVST is associated with increased mortality risk in individuals with cirrhosis, with an OR of 1.6 for death.[5] Additionally, PVST can worsen PHT, increasing the risk of variceal bleeding. If the thrombosis extends to the SMV, it can lead to intestinal infarction.[6 7] The occurrence of PVST also complicates future liver transplantation.[8–10] While most PVST cases arise in the early postoperative phase, some studies have documented thrombotic events occurring as late as 4 months after splenectomy. In our study, we found that during a 6-month follow-up, the incidence of PVST was 38.52% (151/392), with 95.36% (144/151) occurring within the first 3 months. Due to its complex risk factors and subtle clinical manifestations, PVST can be misdiagnosed or recognised late, highlighting the need for early prediction.

Early prediction of PVST is critical for optimising clinical management and improving patient outcomes. Current tools for predicting PVST, such as nomograms developed using traditional statistical methods, have shown limited accuracy.[4 11 12] These tools often rely on linear relationships and may not fully capture the complex, non-linear interactions among risk factors. Machine learning (ML) approaches, on the other hand, have garnered considerable attention in recent years due to their superior predictive capabilities in diverse conditions, such as cardiovascular diseases and cancer.[13–16] The strength of ML lies in its ability to identify complex non-linear relationships among predictors, yielding more reliable predictions.[17] However, there is limited research exploring the use of ML algorithms to predict PVST after SPDV. Therefore, developing an ML-based prediction model holds significant potential to enhance the accuracy of PVST risk assessment in patients with cirrhosis and PHT.

This study aimed to develop explainable models using distinct ML algorithms to accurately predict PVST risk within the first 3 months after SPDV in individuals with cirrhosis and PHT. By incorporating SHapley Additive exPlanations (SHAP), the models also provide transparent explanations for personalised risk predictions, facilitating targeted preventive interventions and reducing unnecessary medical treatments across the patient population. The findings of this study may contribute to improving the early diagnosis and management of PVST, ultimately enhancing clinical outcomes for patients undergoing SPDV.

METHODS

Study design and population

This is a clinical study with a 3-month follow-up according to the incidence of the PVST in our study. We enrolled 392 patients with cirrhosis who had SPDV at the Second Affiliated Hospital of Xi'an Jiaotong University from 1 July 2016 to 31 December 2022. The inclusion criteria were patients: (1) with PHT and cirrhosis; (2) who underwent SPDV; and (3) who did not have PVST detected by preoperative portal vein colour ultrasound or abdominal CT. The exclusion criteria were defined as follows: (1) patients who died within 1 week after SPDV; (2) presence of thrombosis in other body parts; (3) individuals with malignant tumours and severe systemic diseases (eg, cardiovascular and cerebrovascular diseases, respiratory diseases); and (4) other conditions including blood disorders, trauma or splenic cysts. The participants of the study were followed up for 3 months from the surgery and underwent portal vein colour ultrasound or abdominal CT examination at 7 days, 1 month and 3 months after SPDV. This study adhered to the Transparent Reporting of a Multivariable Prediction Model for Individual Prognosis or Diagnosis (TRIPOD) reporting guideline[18] for prognostic studies. TRIPOD is a predictive model report specification including six parts: title and abstract, introduction, method, result, discussion and other information, with a total of 22 checklist items (https://www.bmj.com/content/350/bmj.g7594.long). The TRIPOD statement plays a crucial role in enhancing the transparency and reproducibility of prediction models. Informed consent was waived from participants. Details of patient selection and study design are displayed in figure 1.

Outcome

The primary outcome of this study was the occurrence of PVST at 3 months after splenectomy, defined as the obstruction or narrowing of the portal vein, splenic vein and SMV, or intrahepatic portal vein branches due to a thrombus.[19] Postoperative PVST diagnosis relied on portal vein colour ultrasound or abdominal CT scans.

Predictor variables

We collected data on 37 potential candidate variables from available clinical data, including demographic details, disease characteristics, imaging and laboratory tests, perioperative data and postoperative prophylactic anticoagulant and anti-platelet (PLT) therapy. We first excluded any variables with more than 25% missing values, which included operation time, diameter of the splenic vein, intraoperative red blood cell transfusion, intraoperative plasma transfusion, intraoperative blood loss, mean PLT volume and PLT distribution width,

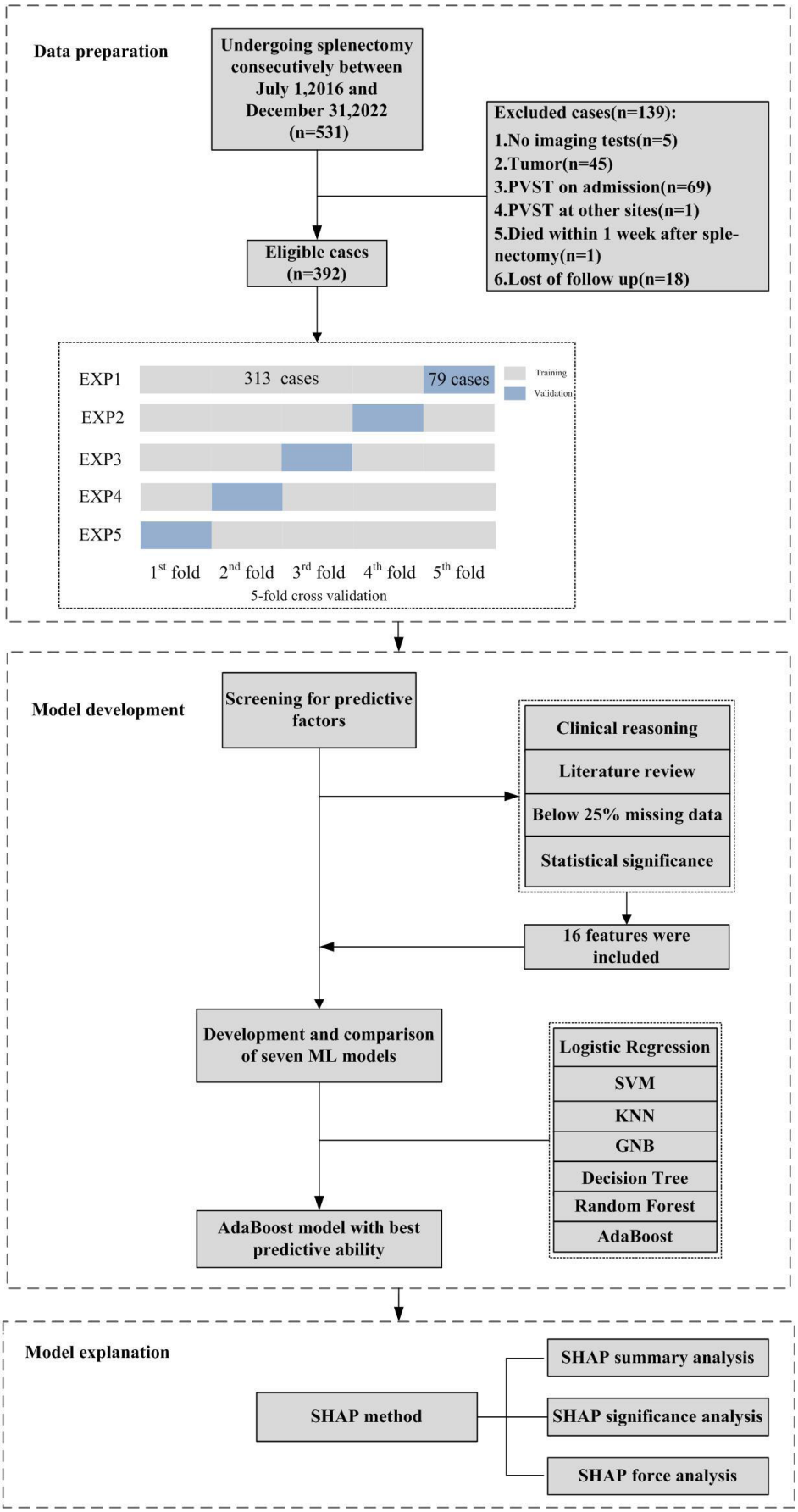

Figure 1 Flow chart of patient selection and study design. GNB, Gaussian Naive Bayes; ML, machine learning; PVST, portal vein system thrombosis; SHAP, SHapley Additive exPlanations.

totalling seven characteristics. The proportions of missing data per variable are detailed in online supplemental table S1. Next, we performed feature selection using the least absolute shrinkage and selection operator (LASSO), which is among the most widely used feature selection techniques. This algorithm uses LassoCV, a fivefold cross-validation approach, to automatically eliminate factors with zero coefficient (Python V.0.22.1, sklearn). Finally, combining SHAP values, we ultimately selected eight key features: length of stay (LOS), gastrointestinal haemorrhage, diameter of the portal vein (DPV), PLT addition, albumin (ALB), γ-glutamyl transferase (GGT), activated partial thromboplastin time (APTT) and D-dimer levels (online supplemental table S2).

Development and validation of the models

To establish derivation and validation cohorts, we employed a five fold cross-validation approach. The study population was randomly divided into five subsets with comparable event rates. For the derivation cohort, four subsets (80%) were merged, while the remaining subset (20%) was set aside as the validation set. This iterative process was executed five times for each outcome, ensuring that each subset functioned as the validation set, thus accommodating patient variability. We use the grid search method to optimise the hyperparameters and determine the optimal hyperparameter combination through cross-validation. For the predictors with missing data, we applied the missForest method.[20]

Five representative ML algorithms were chosen to predict the occurrence of PVST during a 3-month follow-up. Gaussian Naive Bayes, decision tree,[21] and logistic regression,[22] are inherently interpretable models, whereas random forest and AdaBoost[23] are classified as 'black box' models due to their opaque relationships between input features and predicted outcomes.

The predictive performance was evaluated through assessments of discrimination and calibration. Discrimination of the model was quantified by the area under the receiver operating characteristic curve (AUC), accuracy, sensitivity, specificity, positive predictive value and negative predictive value. AUC values closer to 1 indicate higher model accuracy. The accuracy of probability of the best performing model was assessed using the Brier score, which is defined as the mean squared difference between the observed and the predicted outcome. Brier scores range from 0 to 1.00, with 0 representing the best possible calibration. Calibration plots were employed to compare predicted probabilities with actual incidences.

Model explanation

To elucidate the impact and contribution of each feature variable to the final model, we used the SHAP method. SHAP is a technique that ranks the significance of input features and clarifies the model's predictive outcomes, serving to address the 'black-box' dilemma. The importance of each feature was assessed by computing the mean absolute value of its SHAP score. It has been shown that using the SHAP method is useful to interpret ML models built based on real hospital data and can uncover the underlying relationships between features and outcome.[24]

Statistical analysis

All statistical analyses were conducted using Python V.3.8.0 and IBM SPSS Statistics software V.25.0. Continuous variables were described using the mean±SD for normally distributed data and median (M) with the IQR (P25, P75) for skewed distributions. Continuous variables were compared using the Student's t-test or non-parametric rank-sum test (Kruskal-Wallis test) as appropriate. Categorical variables were expressed as frequencies or proportions and compared using the χ^2 test or Fisher's exact test. The significance level was set at $p<0.05$ (two sided).

RESULTS

Population characteristics

This study included a population of 531 patients who underwent SPDV. Among them, 139 patients were excluded due to thrombosis at baseline, lack of imaging, loss to follow-up or death. Finally, a total of 392 patients were included in this study. The data were randomly divided into five subsets: a training set with 313 cases and a validation set with 79 participants. The clinical baseline characteristics are shown in table 1. The average age was 50±10.57, and 53.57% of participants were males. During the 3-month follow-up, 144 patients experienced thrombosis, resulting in an incidence rate of 36.73%.

Feature selection

For feature selection, we selected features from the dataset using the LASSO method (online supplemental figures S1 and S2). This process identified 11 features with non-zero coefficients in the final model. Integration with SHAP analysis further refined the feature set, resulting in the selection of eight key predictors (LOS, gastrointestinal haemorrhage, DPV, PLT addition, ALB, GGT, APTT, LOS) to construct the new predictive model.

ML for outcome prediction

The results from the five ML models are summarised in table 2. Of these methods, the AdaBoost model exhibited the highest predictive performance (AUC=0.72, 95% CI 0.60 to 0.84), better than other ML models. The discrimination capabilities of the various models were illustrated through receiver operating characteristic curves in figure 2. The mean Brier score of the AdaBoost model was 0.233. The calibration curve of the AdaBoost model was close to the ideal curve (online supplemental figure S3), demonstrating that the AdaBoost model fitted well internally.

Model interpretation

The SHAP method was used to illustrate how these variables affect the occurrence of PVST in the model. As shown in SHAP summary plots (figure 3A,B), the contributions of the feature to the model were evaluated using the

Table 1 Baseline characteristics between PVST and non-PVST patients

Characteristics	PVST (n=144)	Non-PVST (n=248)	P value
Age (years)	49.5 (41–55.75)	52 (45–60.75)	0.101
Gender, n (%)			0.023
Male	88 (61.1)	122 (49.2)	
Female	56 (38.9)	126 (50.8)	
LOS (days)	28 (23–35)	23 (20–35.5)	0.001
Aetiology, n (%)			0.234
HBV	85 (59)	140 (56.4)	
HCV	21 (14.6)	24 (9.7)	
Others	38 (26.4)	80 (33.9)	
Spleen length (cm)	25 (20–30)	20 (18–25)	0.990
Spleen width (cm)	18 (13–25)	15 (14–16)	0.709
Spleen thickness (cm)	10 (8–15)	107 (5–12)	0.441
Oesophagogastric fundal varices, n (%)			0.001
Yes	126 (87.5)	182 (73.4)	
No	18 (12.5)	66 (26.6)	
Therapeutic endoscopy, n (%)			0.010
Yes	29 (20.1)	27 (10.9)	
No	115 (79.9)	221 (89.1)	
Gastrointestinal haemorrhage, n (%)			0.000
Yes	91 (63.2)	104 (41.9)	
No	53 (36.8)	144 (58.1)	
Child-Pugh class, n (%)			0.333
A	26 (19.1)	55 (23.9)	
B	106 (77.9)	172 (74.8)	
C	4 (3)	3 (1.3)	
Diabetes, n (%)			0.074
Yes	8 (5.6)	27 (10.9)	
No	136 (94.4)	221 (89.1)	
Operations, n (%)			0.010
Open	119 (82.6)	172 (69.4)	
Laparotomy	21 (14.6)	69 (27.8)	
Conversion to open	4 (2.8)	7 (2.8)	
Ascites, n (%)			0.292
Yes	32 (22.2)	67 (27)	
No	112 (77.8)	181 (73)	
Anticoagulation therapy, n (%)			0.560
Yes	73 (48.3)	131 (54.4)	
No	78 (51.7)	110 (45.6)	
Antiplatelet therapy, n (%)			0.781
Yes	67 (53.5)	119 (48)	
No	77 (46.5)	129 (52)	
Diameter of portal vein (mm)	16 (14–18)	15 (13–17)	0.000
White cell count (10^9/L)	2.13 (1.56–2.53)	2.06 (1.48–2.87)	0.403
Red cell count (10^{12}/L)	3.51 (3.09–3.89)	3.46 (2.91, 3.80)	0.158
PLT (10^9/L)	46 (34–56)	39 (27–55.5)	0.504

Continued

Table 1 Continued

Characteristics	PVST (n=144)	Non-PVST (n=248)	P value
PLT addition (10^9/L)	391 (212–611)	266 (205–542)	0.003
TBIL (μmol/L)	21 (16.01–28.8)	22.10 (16.48–30.88)	0.255
DBIL (μmol/L)	7.23 (5.10, 9.99)	7.88 (5.62,10.53)	0.514
IBIL (μmol/L)	12.89 (9.38,18.57)	14.48 (10.05–20.20)	0.230
ALT (IU/L)	17 (13–24)	25 (17–31)	0.287
AST (IU/L)	24 (19–32)	28 (23–37.29)	0.536
ALB (g/L)	39.70 (33.6–41.90)	37 (33.4–40.10)	0.009
GGT	24 (16–37)	27 (18–51)	0.035
APTT (s)	36 (30.60, 40.40)	35.40 (31.45, 39.80)	0.007
INR	1.19 (1.10–1.33)	1.22 (1.13–1.31)	0.306
D-dimer (ng/mL)	660 (367.50–1157.50)	710 (330–1480)	0.053

Continuous values were presented as median (IQR). Categorical values were presented as number (%).
ALB, serum albumin; ALT, alanine transaminase; APTT, activated partial thromboplastin time; AST, aspartate transaminase; DBIL, direct bilirubin; GGT, γ-glutamyl transferase; HBV, hepatitis B virus; HCV, hepatitis C virus; IBIL, indirect bilirubin; INR, international normalised ratio; LOS, length of stay; PLT, platelet; PLT addition, the difference in PLT before and after splenectomy; PVST, portal vein system thrombosis; TBIL, total bilirubin.

average SHAP values and exhibited in descending order. Additionally, the SHAP dependence plot (figure 3C) can show an individual-level breakdown of how these model features result in changes in individual risk prediction scores of the patient by identifying which feature values affect the final risk prediction score by shifting it higher or lower.

DISCUSSION

In this clinical study, we employed five distinct ML algorithms to predict the risk of PVST during the 3-month follow-up after splenectomy in patients with cirrhosis. Although some early predictive models have been developed using traditional statistical methods, the performance of ML approaches remained unclear. Our findings indicate that the ML-based prediction models can provide an initial risk assessment. Notably, the AdaBoost model demonstrates moderate discriminative ability in distinguishing between high-risk and low-risk patients, with an AUROC of 0.72 (95% CI 0.60 to 0.84). This highlights the potential of ML methods in clinical prediction.

We identified eight key clinical features as significant predictors of PVST risk, including ALB, PLT addition, DPV, GGT, LOS, APTT, D-dimer level and a history of preoperative gastrointestinal bleeding. While previous studies have explored PVST risk in patients with cirrhosis, few have used ML to construct predictive models.[4] ML is particularly powerful for analysing complex, extensive data and can adeptly manage high variability and complicated intervariable relationships. Given the intricate nature of our clinical data, employing ML proved to be essential.

A key objective of the study was to develop an interpretable predictive model, and the trade-off between explainability and model performance was inevitable. By combining AdaBoost with SHAP, the study managed to strike a balance between interpretability and performance. While the AUROC of 0.72 indicates certain

Table 2 Performance evaluation of the five prediction models

	AUC (95% CI)	Accuracy	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Logistic regression	0.615 (0.484 to 0.746)	0.564	0.621	0.531	0.437	0.714
Random forest	0.680 (0.552 to 0.809)	0.673	0.255	0.919	0.682	0.678
AdaBoost	**0.720 (0.599 to 0.841)**	**0.679**	**0.641**	**0.70**	**0.558**	**0.771**
Decision tree	0.608 (0.489 to 0.728)	0.597	0.531	0.636	0.466	0.698
GNB	0.681 (0.556 to 0.806)	0.643	0.662	0.632	0.513	0.762

The AdBoost Model performance is the best.
AUC, area under the receiver operating characteristic curve; GNB, Gaussian Naive Bayes.

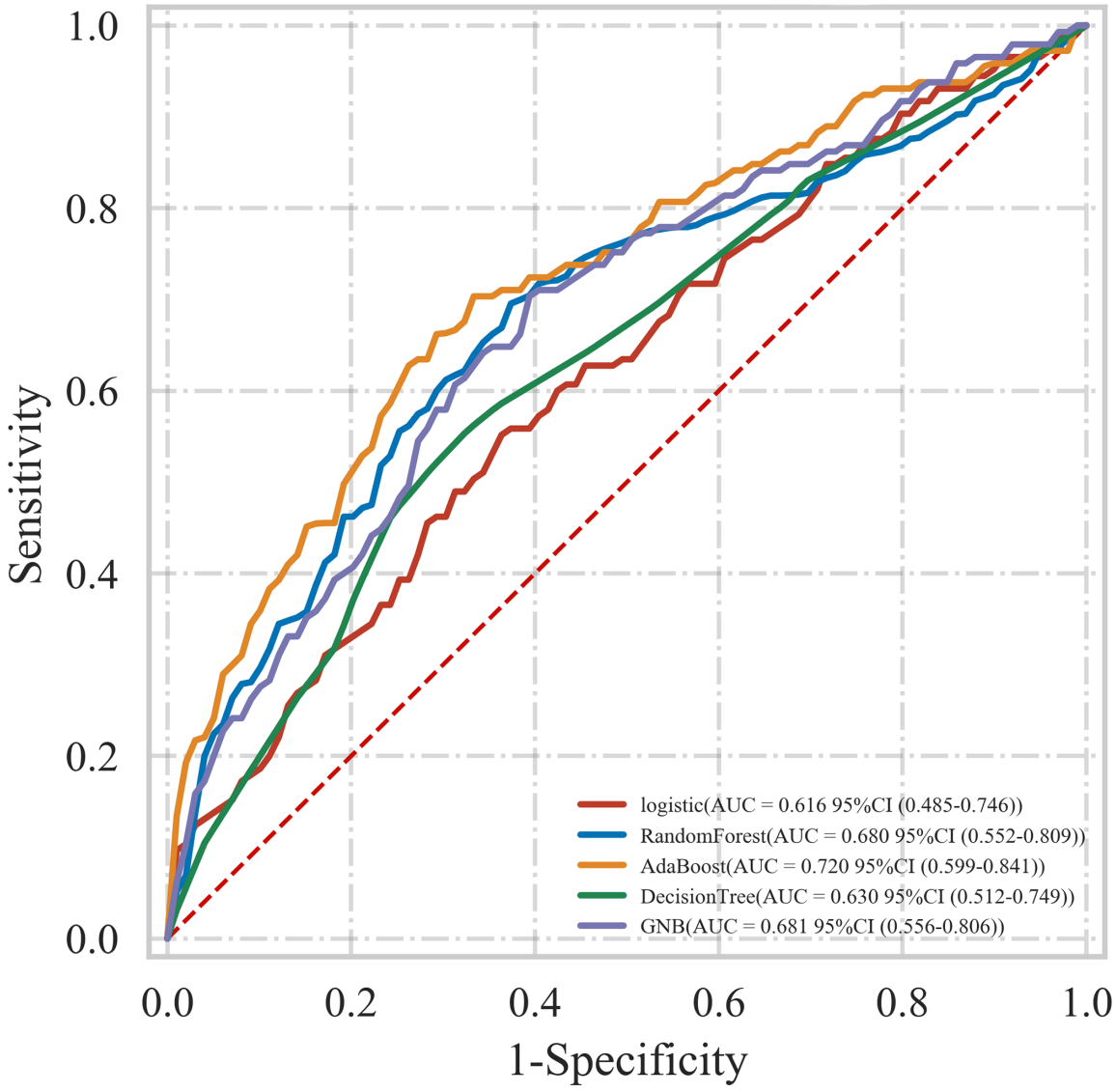

Figure 2 Receiver operating characteristic. AUC, area under the receiver operating characteristic curve; GNB, Gaussian Naive Bayes.

limitations, an AUROC range of 0.7–0.8 still holds clinical significance in some practical applications, particularly in scenarios where a balance between interpretability and performance is required. SHAP analysis provided valuable insights into the importance of each feature in the model, offering explanations for the predictions and enhancing their clinical relevance.

The SHAP scores indicated noteworthy insight into the importance of each feature in the AdaBoost model. PVST pathogenesis is multifactorial, primarily driven by Virchow's triad—hypercoagulability, endothelial injury, and hemodynamic stasis—which collectively contribute to thrombus formation in the portal venous system. multifactorial, influenced by Virchow's triad—hypercoagulability, endothelial injury and reduced blood flow.[19 25] We found the DPV to be a significant predictor. A wider portal vein may decrease blood flow velocity, create turbulent flow conditions that damage the venous endothelium and facilitate thrombus formation, supporting findings from prior studies. Additionally, our results highlighted elevated D-dimer levels as a risk factor for postoperative thrombosis. Previous studies[26] have established a correlation between increased D-dimer levels and heightened coagulation activity, indicating a propensity for thrombosis. Our findings align with these established links, confirming the role of D-dimer in predicting postoperative outcomes. Notably, we found a correlation between a history of preoperative gastrointestinal bleeding and postoperative thrombotic outcomes, supporting existing literature.[19] This underscores the importance of comprehensive preoperative assessments in identifying at-risk patients. Furthermore, additional indicators of PVST risk included factors such as length of hospital stay, ALB levels, APTT and variations in PLT counts. These eight

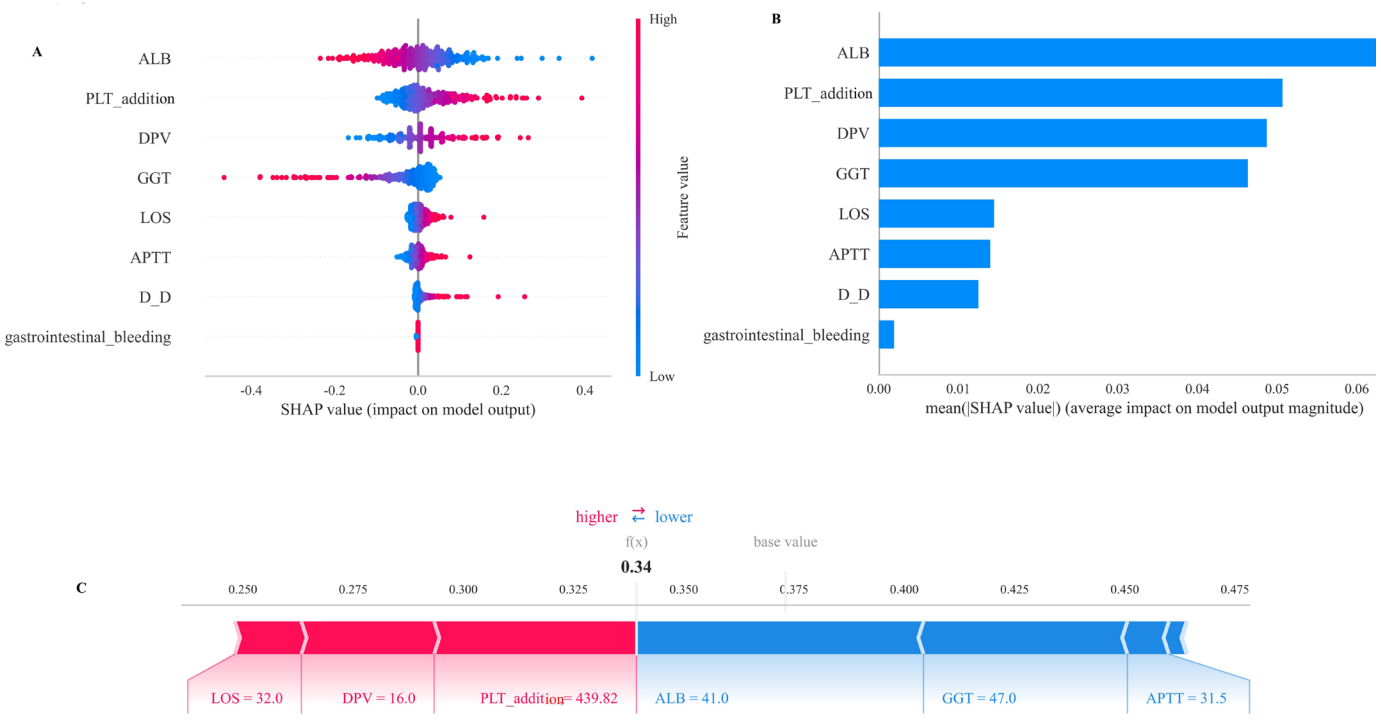

Figure 3 SHapley Additive ExPlanations (SHAP) for the AdaBoost model. (A) SHAP summary dot plot. (B) SHAP summary bar plot. All samples and features are illustrated, with each row representing a feature and x-axis representing the SHAP value. Within each row, each dot represents a patient. The colours of the dots represent the feature values: red for larger values and blue for lower values. (C) SHAP individualised predictions. ALB, albumin; APTT, activated partial thromboplastin time; DPV, diameter of the portal vein; GGT, γ-glutamyl transferase; LOS, length of stay; PLT, platelet.

clinical parameters together form a robust foundation for constructing predictive models aimed at anticipating PVST occurrence in patients with cirrhosis following splenectomy.

In summary, our study highlights the utility of ML in predicting PVST risk in a complex clinical landscape and identifies critical clinical features that contribute to the prediction. The findings suggest that integrating these parameters into clinical practice can enhance risk stratification and potentially guide personalised patient management after splenectomy.

Our model was designed with both model developers and clinicians in mind. Through interpretability tools such as SHAP values and feature importance, developers can gain a better understanding of the model's internal mechanisms, allowing for optimisation of its performance. Additionally, explainability is equally crucial for clinicians. Clinicians need to comprehend the model's predictions and the rationale behind them to trust and adopt its recommendations. We achieve this by providing clear interpretability results, which help clinicians understand the model's prediction process, thereby enhancing their trust in the model. We employed SHAP values as the primary method for explainability. SHAP values quantify the contribution of each feature to the model's predictions, enabling clinicians to identify which features have the greatest influence on the results. We presented feature contribution plots that illustrate the positive and negative impacts of each feature on the prediction results, along with concise textual explanations to help clinicians intuitively grasp the model's reasoning. Explainability holds significant value in clinical practice. For example, by identifying high-impact features (such as ALB/DPV), clinicians can conduct more accurate risk stratification and develop personalised treatment plans. For high-risk patients, doctors can implement more proactive monitoring and intervention strategies, which may improve patient outcomes. Explainability helps clinicians build trust in the model. If doctors can understand why the model makes a particular prediction, they are more likely to integrate it into their clinical practice. For instance, through SHAP value plots, doctors can visually see the contribution of a specific feature to the prediction, thereby bolstering their confidence in the model. Despite using explainability tools like SHAP values, interpreting complex models remains challenging. For example, deep learning models may require more advanced explanation strategies to uncover their internal logic.

Several limitations should be considered. First, potential biases, such as selection bias, information bias and missing data biases, may impact the reliability of the findings. Second, the reliance on a study population from a single-centre clinical study could limit the generalisability of the results. While the data from a single centre may not fully capture the diversity of broader clinical settings or patient populations, this limitation does not diminish the value of the model developed and validated in this study. The model demonstrates practical utility for patients

within the study centre and lays a solid foundation for future multicentre research. Third, the models lack external validation. However, employing iterated cross-validation enhances result reliability and reduces the risk of overfitting, preventing an excessively optimistic evaluation of model performance. Fourth, additional data, like imaging data and novel biomarkers, have the potential to improve prediction accuracy. Future research should investigate the integration of these variables to strengthen the AdaBoost models proposed in this study.

Future research should focus on advancing the application of ML in predicting PVST. This includes emphasising comprehensive data collection, such as imaging data, to enhance prediction accuracy and strengthen the AdaBoost models developed here. Further optimisation of model selection and tuning is necessary, with consideration given to dataset characteristics (eg, non-linear relationships, high-dimensional features and class imbalance) when selecting models. Ensemble models are generally robust choices, but linear models can also perform well through appropriate tuning and selection of kernel functions. During the tuning process, it is recommended to employ a broader parameter search and cross-validation to ensure the model can adapt to the dataset's characteristics. Adopting a multicentre study design will enhance the external validity of the research results. Multicentre studies will enable us to better evaluate the applicability of these results across different clinical environments and patient populations. Furthermore, researchers should consider developing prediction models for both immediate and long-term time frames, distinguishing between early and long-term predictors to better address variations in PVST occurrences.

CONCLUSIONS

This study predicted PVST with 3 months after SPDV in patients with cirrhosis with PHT via five ML models using clinical data. The AdaBoost model performed the best and identified key factors for predicting PVST. By incorporating SHAP analysis, developers can gain a better understanding of the internal mechanisms of the model, thereby optimising its performance. This also enables clinicians to receive transparent explanations for personalised risk predictions, enhancing their trust in the model and facilitating targeted preventive interventions, while reducing excessive interventions across the entire patient population. Furthermore, this research provides a fresh perspective on clinical interventions for various health issues.

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Contributors ZFL was responsible for conception of the study and overall content as guarantor. DQ and DD jointly conceived the study idea. GDL, CD, JZha, XJS and JZhu collected and collated the data. DQ analysed the data, interpreted the model results and drafted the initial manuscript. DD developed prediction models and performed model evaluations using fivefold cross-validation, and performed SHapley Additive exPlanations (SHAP) analysis to interpret the model results. RZ and LA provided guidance on clinical concepts, particularly in the interpretation of model results and the clinical relevance of predictors. All authors critically revised the manuscript and approved the final version. Artificial intelligence was used for editing the paper for clarity and for developing the reviewed use cases as part of the narrative review.

Funding This work was funded by the Shaanxi Provincial Department of Science and Technology's key research and development plan (2020GXLH-Z-002) and the Shaanxi Province Medical and Industry Integration high-end medical equipment technology research and development platform (2023GXJS-01).

Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval Ethical approval for this study was obtained from the Ethics Committee of the Second Affiliated Hospital of Xi'an Jiaotong University (2024YS459).

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement No data are available.

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