

ORIGINAL ARTICLE

A Nomogram Model to Predict the Risk of Concurrent Liver Injury in Pediatric Patients with Infectious Mononucleosis

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SUMMARY

Background: Infectious mononucleosis (IM), an acute self-limiting disease predominantly caused by Epstein-Barr virus (EBV), is common in children. Liver injury is one of its most frequent complications. Early identification and prediction of the risk of liver injury in children with IM are crucial for timely intervention and improved prognosis. This study aimed to develop and validate a nomogram model for predicting the risk of concurrent liver injury in pediatric patients with IM.

Methods: This retrospective study enrolled 202 pediatric patients with IM who were diagnosed and treated at our hospital between January 2023 and December 2024. Based on serum alanine aminotransferase (ALT) levels, patients were divided into a liver injury group (ALT > 50 U/L, n = 116) and a control group (ALT < 50 U/L, n = 86). General clinical data and laboratory parameters were collected and compared between the two groups. Multivariable logistic regression analysis was employed to identify independent risk factors for concurrent liver injury in pediatric patients with IM. Subsequently, a nomogram prediction model was constructed and verified based on these factors.

Results: Among the 202 pediatric patients with IM, the incidence of liver injury was 57.42%. The incidence of hepatosplenomegaly was significantly higher in the liver injury group compared to the control group ($p < 0.05$). Statistically significant differences were observed between the two groups regarding neutrophil percentage (NEU), lymphocyte percentage (LYM), platelet count (PLT), platelet distribution width (PDW), uric acid (UA), beta2-microglobulin (β 2-MG), atypical lymphocytes, and interleukin-6 (IL-6) ($p < 0.05$). Multivariable logistic regression analysis revealed that PDW, UA, β 2-MG, atypical lymphocytes, and IL-6 were independent risk factors for concurrent liver injury in pediatric patients with IM. The nomogram model constructed based on these independent risk factors exhibited great discrimination and calibration, with a concordance index (C-index) of 0.942 (95% CI: 0.877 - 1.007) and an area under the receiver operating characteristic curve (AUC) of 0.960. Decision curve analysis (DCA) showed the model provided substantial net clinical benefit across threshold probabilities ranging from 4% to 100%.

Conclusions: The nomogram model constructed in this study can effectively predict the risk of concurrent liver injury in pediatric patients with IM.

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KEYWORDS

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INTRODUCTION

Infectious mononucleosis (IM), an acute, self-limiting lymphoproliferative disease caused by primary infection with EBV, has a high incidence in children and adolescents [1]. The main clinical manifestations include fever, pharyngitis, cervical lymphadenopathy, hepatosplenomegaly, palpebral edema, and rash [2]. Although most children with IM have a great prognosis, some may develop various complications [3]. Among these, liver injury is the most common complication in children with IM, primarily manifesting as abnormal liver function indicators, such as elevated serum ALT, which directly reflects damage to hepatocytes [4]. A few severe cases may progress to chronic hepatitis or even liver failure, posing significant challenges for clinical management [5]. Therefore, early identification and prediction of the risk of concurrent liver injury in children with IM are of great importance for guiding clinical decision-making, facilitating timely intervention, and improving patient prognosis.

However, current risk assessment for concurrent liver injury in patients with IM primarily relies on clinical experience and partial laboratory parameters, lacking a systematic, objective, and readily, clinically applicable early prediction tool [6]. Notably, predictive models specifically for pediatric patients remain scarce. This study aimed to retrospectively analyze the clinical data and laboratory findings of pediatric patients with IM, identify independent risk factors associated with concurrent liver injury using multivariable logistic regression analysis, and subsequently develop a risk nomogram prediction model based on these factors. This model is intended to provide a scientific basis for early identification and intervention in high-risk pediatric patients.

MATERIALS AND METHODS

Patients

This retrospective study enrolled 239 pediatric patients diagnosed and treated for IM at Dazhou Integrated Traditional Chinese Medicine & Western Medicine Hospital between January 2023 and December 2024. The study was approved by the Ethics Committee of our hospital. All subjects met the diagnostic criteria for IM, based on clinical symptoms, laboratory findings, and imaging examinations [7]. After excluding 37 cases due to chronic liver disease, viral hepatitis, sepsis, severe bacterial infection, immune dysfunction, use of hepatotoxic drugs, or incomplete data, a total of 202 pediatric patients were included in the final analysis. Liver injury was defined as a serum ALT > 50 U/L. Patients were subsequently divided into the control group (n = 86) and the liver injury group (n = 116) based on the presence or absence of liver injury. General clinical data, including gender, age, duration of hospitalization, duration of symptoms, clinical manifestations, and laboratory pa-

rameters, were collected for all enrolled patients.

Measurement of laboratory parameters

Complete blood cell analysis was performed using the CAL-7000 automated hematology analyzer (Mindray, China), which recorded white blood cell count (WBC), NEU, LYM, monocyte percentage (MONO), eosinophil percentage (EOS), PLT, mean platelet volume (MPV), PDW, and C-reactive protein (CRP) results. The atypical lymphocytes percentage was determined by preparing blood smears, which were then differentially counted under a microscope by an experienced physician. The final result was recorded as the average of three separate counts. Serum biochemical parameters, including liver function tests, were measured using an XPT automated biochemical analyzer (Siemens, Germany). Serum IL-6 levels were measured using the CARIS 2000 automated chemiluminescence analyzer (WAN-TAI Biopharm, China).

Prediction model development and validation

Univariate analysis was performed to compare the differences in clinical characteristics and laboratory parameters between the liver injury group and the control group. Variables that demonstrated statistically significant differences in the univariate analysis were selected as candidate variables and were subsequently included in a multivariate logistic regression analysis to identify independent risk factors for liver injury. Based on these independent risk factors, a predictive nomogram model was then constructed. The discriminative ability of this model was evaluated by AUC. Its predictive accuracy was assessed using a calibration curve, and the clinical net benefit was evaluated by DCA.

Statistical analysis

Statistical analyses and plotting were performed using R4.5.0 software for Windows. For categorical variables, frequencies and percentages were used, and the chi-squared test was applied to compare between two groups. The Kolmogorov-Smirnov test was used to assess the normality of continuous variables. Data that met normal distribution were presented as mean \pm standard deviation, and an independent samples *t*-test was used for comparing two groups; data that did not meet normal distribution were presented as medians (interquartile ranges, IQRs), and the Mann-Whitney U test was used for comparing two groups. Multivariate logistic regression analysis was used to identify independent risk factors for IM with liver injury. A risk nomogram model was constructed using the R4.5.0 software and the “rms” package. Internal validation was performed using the “caret” package and the bootstrap resampling method. The C-index was calculated, and calibration curves, ROC curves, and DCA were plotted. A *p*-value of < 0.05 was considered statistically significant.

RESULTS

Patients' characteristics

A total of 202 pediatric patients with IM were included in the study. Among them, 116 were assigned to the liver injury group and 86 to the control group. The incidence of liver injury was 57.42%. No statistically significant differences ($p > 0.05$) were observed between the two groups regarding gender, age, hospital stay, duration of symptoms, fever, pharyngitis, cervical lymph node enlargement, eyelid edema, and rash. However, the incidences of hepatomegaly and splenomegaly were significantly higher in the liver injury group compared to the control group ($p < 0.05$), which is consistent with the clinical manifestations of liver injury (Table 1).

Comparison of liver function indicators

The liver injury-related parameters were all significantly higher in the liver injury group compared to the control group ($p < 0.05$). However, parameters of hepatic synthetic function, namely total protein (TP), albumin (Alb), and prealbumin (PA), showed no significant differences compared to the control group ($p > 0.05$) (Table 2). This indicates that in the early stage of liver injury in pediatric patients with IM, hepatic synthetic function was not yet impaired, suggesting that timely intervention may lead to a better prognosis.

Comparison of conventional laboratory parameters

The analysis of results showed no statistically significant differences ($p > 0.05$) between the liver injury group and the control group in terms of WBC, MONO, EOS, MPV, CRP, creatine kinase (CK), and creatine kinase-MB (CK-MB). In the liver injury group, LYM, PDW, atypical lymphocytes, UA, β 2-MG, and IL-6 were all significantly higher than in the control group. Conversely, NEU and PLT were significantly lower in the liver injury group compared to the control group ($p < 0.05$) (Table 3).

Independent risk factors for concurrent liver injury in pediatric patients with IM

To identify independent risk factors for concurrent liver injury in pediatric patients with IM (dependent variable; coded: Yes = 1, No = 0), factors that were statistically significant in the univariate analysis were entered into a multivariate logistic regression analysis as independent variables. These variables included: hepatomegaly (coded: Yes = 1, No = 0), splenomegaly (coded: Yes = 1, No = 0), NEU (actual measured value), LYM (actual measured value), PLT (actual measured value), PDW (actual measured value), UA (actual measured value), β 2-MG (actual measured value), IL-6 (actual measured value), and atypical lymphocytes (actual measured value). The results indicated that PDW, atypical lymphocytes, UA, β 2-MG, and IL-6 were independent risk factors for concurrent liver injury in pediatric patients with IM (Table 4).

Construction and validation of the nomogram model

Based on the identified independent risk factors for concurrent liver injury in pediatric patients with IM, a risk nomogram model was constructed (Figure 1). The calibration curve results showed that the predicted values were in strong agreement with the actual observed values, with a C-index of 0.942 (95% CI: 0.877 - 1.007) (Figure 2A). The receiver operating characteristic (ROC) curve for internal validation of the nomogram model showed an AUC of 0.960 (Figure 2B). DCA indicated that when the threshold probability was within the range of 4% to 100%, using this nomogram to predict the risk of concurrent liver injury in pediatric patients with IM provided a high net benefit (Figure 2C).

DISCUSSION

Infectious mononucleosis, as a systemic disease caused by EBV infection, has a high incidence in children. The liver is one of the common target organs for EBV infection, and approximately 50% - 90% of IM patients may experience varying degrees of liver injury, manifesting as elevated serum transaminases [8]. Although concurrent liver injury in most IM patients is self-limiting, a few severe cases may progress to chronic liver disease or fulminant hepatic failure, posing a serious threat to the children's health [4]. Therefore, the early identification of risk factors for concurrent liver injury in children with IM and the establishment of effective predictive models are crucial for guiding clinical early intervention. This study, through a retrospective analysis of the clinical data and laboratory results of 202 children with IM, successfully identified independent risk factors associated with concurrent liver injury and constructed a nomogram model with good predictive performance. Through multivariate logistic regression analysis, this study identified PDW, UA, β 2-MG, atypical lymphocyte percentage, and IL-6 as independent risk factors for concurrent liver injury in children with IM. PDW is an indicator reflecting platelet volume heterogeneity, and its elevation is usually associated with platelet activation, inflammatory response, and oxidative stress [9]. In various liver diseases, such as viral hepatitis, cirrhosis, and liver cancer, changes in PDW levels have been reported to be correlated with disease severity or prognosis [10-12]. Platelet activation and inflammatory response may play a role in the pathogenesis of concurrent liver injury in IM patients, but their specific mechanisms still require further research to elucidate [13]. UA has pro-inflammatory and pro-oxidative stress effects and is involved in the pathophysiological processes of various diseases, including liver diseases [14]. Hyperuricemia has been confirmed to be associated with non-alcoholic fatty liver disease, the progression of viral hepatitis, and liver fibrosis [15,16]. Cell damage and metabolic disorders caused by EBV infection may lead to increased uric acid production or decreased excretion, thereby elevating serum UA [17]. Monitoring and con-

Table 1. Comparison of clinical characteristics of participants.

Variables	Liver injury group (n = 116)	Control group (n = 86)	p-value
Age	4 (5.8)	4 (3.7)	0.395
Gender, n (%)			
Male	51 (44)	35 (40)	0.566
Female	65 (56)	22 (60)	
Hospital stays, days	6 (5.8)	6 (5.7)	0.091
Duration of symptoms, days	5 (4.6)	4 (4.5)	0.121
Fever, n (%)			
Yes	90 (78)	68 (79)	0.864
No	26 (22)	18 (21)	
Pharyngitis, n (%)			
Yes	94 (81)	72 (84)	0.711
No	22 (19)	14 (16)	
Cervical lymph node enlargement, n (%)			
Yes	90 (78)	68 (79)	0.864
No	26 (22)	18 (21)	
Hepatomegaly, n (%)			
Yes	28 (24)	10 (12)	0.029 *
No	88 (76)	76 (88)	
Splenomegaly, n (%)			
Yes	68 (59)	28 (33)	< 0.001 *
No	48 (41)	58 (67)	
Eyelid edema, n (%)			
Yes	32 (28)	22 (26)	0.872
No	84 (72)	64 (74)	
Rash, n (%)			
Yes	12 (10)	8 (9)	0.806
No	104 (90)	78 (91)	

* p < 0.05 indicates a statistically significant difference.

Table 2. Comparison of liver function indicators.

Variables	Liver injury group (n = 116)	Control group (n = 86)	p-value
ALT	173.24 ± 125.55	26.09 ± 11.01	< 0.001 *
AST	132.03 ± 97.47	39.21 ± 11.90	< 0.001 *
ALP	244.19 ± 120.96	183.40 ± 56.31	0.003 *
GGT	82.09 ± 75.79	15.61 ± 8.07	< 0.001 *
TBIL	11.36 ± 9.52	6.60 ± 2.38	0.002 *
DBIL	5.37 ± 1.19	2.41 ± 1.24	0.01 *
LDH	515.19 ± 115.61	404.81 ± 146.25	< 0.001 *
TP	71.03 ± 4.22	69.84 ± 6.56	0.277
ALB	40.91 ± 2.32	41.60 ± 3.08	0.208
PA	119.28 ± 34.42	118.19 ± 29.83	0.869

ALT alanine aminotransferase, AST aspartate aminotransferase, ALP alkaline phosphatase, GGT gamma-glutamyl transferase, TBIL total bilirubin, DBIL direct bilirubin, LDH lactate dehydrogenase, TP total protein, ALB albumin, PA prealbumin. * p < 0.05 indicates a statistically significant difference.

Table 3. Comparison of conventional laboratory parameters.

Variables	Liver injury group (n = 116)	Control group (n = 86)	p-value
WBC	15.15 ± 5.73	15.24 ± 6.31	0.941
NEU	24.20 (17.98, 31.63)	32.70 (23.70, 42.90)	0.002 *
LYM	66.45 (57.65, 72.45)	59.20 (49.70, 67.60)	0.009 *
MONO	7.61 ± 5.30	7.56 ± 4.75	0.962
EOS	0.2 (0.1, 0.4)	0.3 (0.2, 0.7)	0.091
PLT	170 (149, 215)	230 (184, 264)	0.001 *
MPV	10.35 ± 1.37	10.18 ± 1.71	0.573
PDW	15.70 (14.28, 16.33)	13.60 (11.40, 15.70)	< 0.001 *
CRP	8.03 (4.50, 12.59)	8.83 (4.53, 16.72)	0.271
Atypical lymphocytes	12 (9, 16)	8 (5, 11)	< 0.001 *
CK	75.74 ± 46.25	77.02 ± 54.13	0.933
CK-MB	31.52 ± 18.49	28.91 ± 13.52	0.440
UA	380 (340, 440)	284 (244, 332)	< 0.001 *
β2-MG	3.90 ± 1.06	2.89 ± 0.57	< 0.001 *
IL-6	17.39 (6.75, 23.72)	9.24 (4.67, 19.63)	0.012 *

WBC white blood cell count, NEU neutrophil percentage, LYM lymphocyte percentage, MONO monocyte percentage, EOS eosinophil percentage, PLT platelet count, MPV mean platelet volume, PDW platelet distribution width, CRP C-reactive protein, CK creatine kinase, CK-MB creatine kinase-MB, UA uric acid, β2-MG beta2-microglobulin, IL-6 interleukin-6. * p < 0.05 indicates a statistically significant difference.

Table 4. Independent risk factors for concurrent liver injury in pediatric patients with IM.

Variables	β	Standard error	OR (95% CI)	p-value
PDW	0.733	0.299	2.081 (1.271 - 4.203)	0.015 *
UA	0.029	0.009	1.029 (1.015 - 1.051)	< 0.001 *
β2-MG	2.571	0.787	5.572 (3.405 - 8.224)	0.001 *
Atypical lymphocytes	0.262	0.113	1.299 (1.071 - 1.799)	0.021 *
IL-6	0.132	0.063	1.141 (1.026 - 1.323)	0.038 *

PDW platelet distribution width, UA uric acid, β2-MG beta2-microglobulin, IL-6 interleukin-6. * p < 0.05 indicates a statistically significant difference.

trolling UA levels in children with IM may have potential significance in preventing liver injury. β2-MG is a low-molecular-weight protein found on the surface of all nucleated cells and is primarily produced by lymphocytes [18]. Elevated serum levels of β2-MG typically reflect accelerated cell turnover, decreased glomerular filtration rate, or activation of the immune system [19]. In viral diseases such as EBV infection, the production of β2-MG increases significantly due to lymphocyte activation and proliferation [20]. The results of this study showed that β2-MG is a strong independent predictor of liver injury in children with IM (OR = 5.572). This is consistent with the pathophysiological mechanism whereby EBV infection pri-

marily invades the lymphatic system and elicits a strong immune response, suggesting that β2-MG can serve as a sensitive indicator reflecting the intensity of systemic inflammatory response and the degree of liver involvement in children with IM [18,20]. The presence of atypical lymphocytes is a characteristic hematological change in IM, primarily composed of CD8⁺ T lymphocytes activated after EBV infection [21]. These activated T lymphocytes play a crucial role in clearing virus-infected cells, but their excessive immune response can also cause hepatocellular injury [22]. IL-6 is a pleiotropic cytokine that plays a central role in inflammatory response, immune regulation, and the acute phase response [23]. Viral infections can significantly induce

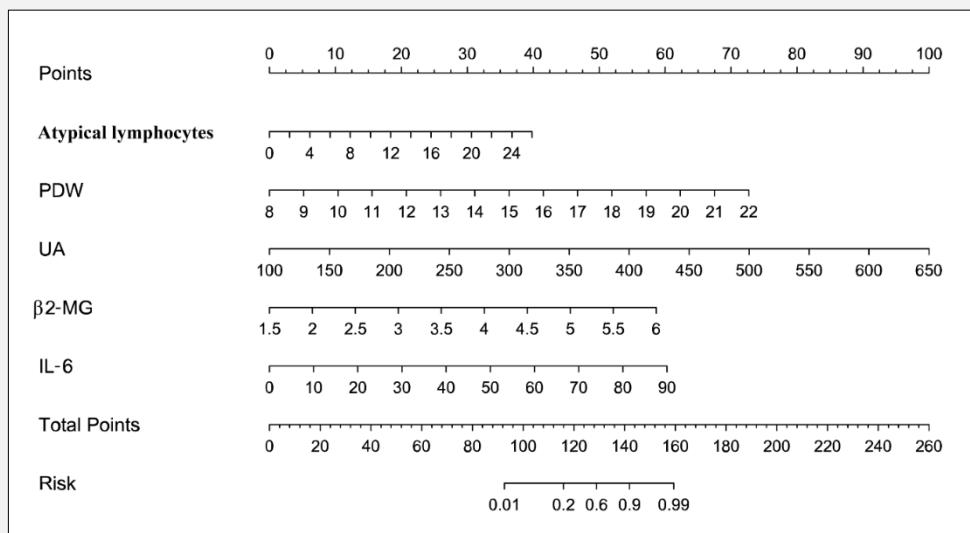


Figure 1. Nomogram for prediction of concurrent liver injury in pediatric patients with IM.

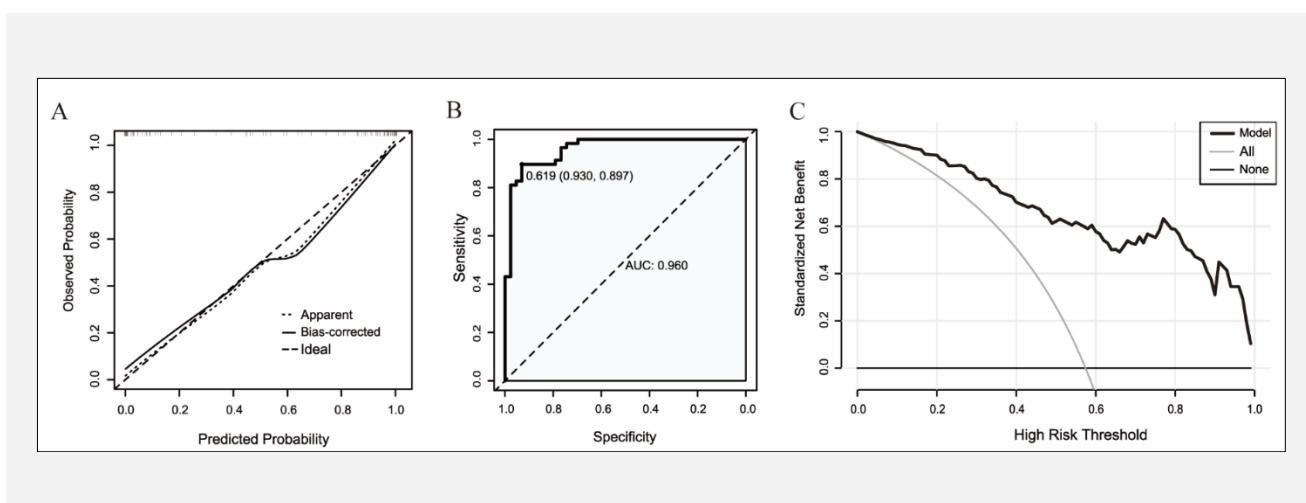


Figure 2. Internal validation of the nomogram model: calibration curve, ROC curve, and decision curve.

A) Calibration curve, B) ROC curve, C) decision curve.

the production of IL-6. On the one hand, IL-6 can protect the liver by inducing the production of acute phase proteins; on the other hand, excessive or persistent production of IL-6 may exacerbate liver injury through mechanisms such as promoting inflammatory cell infiltration and inducing hepatocyte apoptosis [24]. Based on the five aforementioned independent risk factors, we developed a nomogram prediction model. A nomogram, as an intuitive and user-friendly clinical decision-making tool, can transform complex regression

equations into a graphical scoring system, thereby enabling clinicians to individually assess a patient's risk of disease onset. This nomogram model demonstrated good predictive performance and discrimination ability, and it also showed high net benefit across a wide range of threshold probabilities. By integrating multiple objective indicators, this model enables more accurate and earlier identification of high-risk pediatric patients, thus providing a scientific basis for clinical decision-making.

CONCLUSION

This study retrospectively analyzed the clinical data of 202 children with IM and successfully identified PDW, UA, β 2-MG, reactive lymphocyte percentage, and IL-6 as independent risk factors for predicting concurrent liver injury in pediatric IM patients. The nomogram model constructed based on these factors demonstrated good predictive efficacy and clinical utility. This model provides a scientific basis for the early identification of liver injury risk in children with IM and for guiding targeted clinical interventions in a timely manner, holding significant clinical importance for the improvement of patient prognosis.

Declaration of Interest:

None of the authors have any commercial or other association that might pose a conflict of interest.

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