Italian Journal of Pediatrics

RESEARCH Open Access

# Clinical features and risk factors of adenovirus-related plastic bronchitis in children

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## Abstract

**Background** To analyze the clinical characteristics of children with adenoviral pneumonia, identify independent risk factors for early prediction of plastic bronchiolitis (PB), and develop a predictive nomogram.

**Methods** This retrospective study analyzed the clinical data of children diagnosed with adenoviral pneumonia. Patients were categorized into PB and non-PB groups. General characteristics, clinical symptoms, laboratory findings, and imaging results were compared between the two groups. Multivariate logistic regression was used to identify significant risk factors, and a nomogram model was constructed.

**Results** Among the 164 patients, 139 were in the non-PB group and 25 were in the PB group. Multivariate logistic regression identified diminished breath sounds, D-dimer (D-D) levels, and Lactic dehydrogenase (LDH) levels as significant risk factors for PB. The nomogram developed from these factors had an area under the receiver operating characteristic curve (AUC) of 0.904 (95% confidence interval: 0.847–0.960). The Hosmer-Lemeshow test showed good calibration ($p = 0.515$, $X^2 = 7.207$).

**Conclusions** Diminished breath sounds, D-D levels, and LDH levels are independent risk factors for PB in children with adenoviral pneumonia. The developed nomogram demonstrates high predictive accuracy and good calibration, providing a valuable tool for early prediction and clinical decision-making. Future studies should validate this nomogram in larger and diverse populations.

**Keywords** Adenoviral pneumonia, Plastic bronchitis, Fiberoptic bronchoscopy, Risk factors, Nomogram model

## Introduction

Human adenovirus (HAdV) is an enveloped, double-stranded DNA virus that is a significant pathogen responsible for respiratory infections. It is widely distributed across the globe, with 3.5-11% of community-acquired pneumonia cases in children being attributed to HAdV infection [1]. Among community-acquired pneumonia in children, pneumonia caused by HAdV is recognized as one of the more serious types. HAdV infection is also the primary pathogen responsible for severe viral pneumonia in children, particularly those under 5 years of age. The clinical manifestations in some infected children can be extremely severe, predisposing them to a wide range of

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systemic diseases and serious intrapulmonary and extrapulmonary complications, which can sometimes even be life-threatening [2–4]. Plastic bronchiolitis (PB) is a severe intrapulmonary complication characterized by the formation of rubbery tubular structures within the airways, leading to partial or complete obstruction and resulting in impaired lung ventilation. In severe cases, this condition can be life-threatening. If left untreated, patients may suffer from sequelae such as pulmonary atelectasis and occlusive bronchitis. Therefore, early diagnosis is crucial for improving prognosis. However, early diagnosis remains a significant clinical challenge due to the atypical symptoms and imaging manifestations, which can easily lead to misdiagnosis or missed diagnosis [5, 6]. PB can be classified into two types based on the pathology of bronchial tubular patterns: Type I is related to respiratory diseases and is mainly caused by infections, while Type II is primarily associated with congenital heart disease. In recent years, reports of PB caused by HAdV have been gradually increasing and need to be taken seriously by clinicians [7–9].

In this study, we retrospectively analyzed children who met the diagnostic criteria for adenovirus pneumonia and underwent bronchoscopy. Patients were screened for comorbid PB, and we compared clinical symptoms, laboratory findings, and imaging results between patients with and without PB. The study identified risk factors for comorbid PB in children with adenoviral pneumonia and developed a nomogram to enable early recognition of PB. This early detection aims to facilitate timely removal of PB via bronchoscopy, thereby promoting recovery and reducing the incidence of poor prognosis in children with adenoviral pneumonia.

## Methods

### Study participants

This study included children with adenovirus pneumonia who were admitted to Tianjin Children's Medical Center between January 2018 and April 2024.

The inclusion criteria were:

1) age > 1 month and < 18 years.
2) evidence of HAdV infection based on HAdV positivity on multiplex polymerase chain reaction (PCR) performed using nasopharyngeal swab, sputum, and BAL fluid samples.
3) Children who meet the diagnostic criteria for ADV pneumonia as detailed in the 2019 version of the Diagnostic and Treatment Guidelines for ADV Pneumonia in Children, jointly issued by the National Health Commission of the People's Republic of China and the State Administration of Traditional Chinese Medicine [10].
4) Bronchoscopy indications in the Chinese Pediatric Bendable Bronchoscopy Guidelines (2018 edition) were met, there were no contraindications, and bronchoscopy was performed and treated with parental consent [11].
5) The diagnostic criteria for PB include the expectoration or bronchoscopic removal of a cast shaped like a bronchial branch, confirmed by pathological examination.

The exclusion criteria were:

1) Incomplete medical records.
2) Comorbidities, including severe congenital heart disease, neuromuscular diseases, inherited metabolic disorders, severe immunodeficiency, and myelosuppression associated with hematologic malignancies.
3) Presence of confirmed mixed infections with other viruses, bacteria, fungi, or other pathogens.
4) PB due to other diseases such as asthma, lymphatic circulation disorders, chronic heart disease, cystic fibrosis, bronchiectasis.

### Data collection

Patients were divided into PB ($n = 25$) and non-PB groups ($n = 139$) according to their bronchoscopy results. The clinical data of children with Data collection were collected and mainly included the following: (1) general information: name, sex, age, days of hospitalization, season of incidence, history of eczema, and history of wheezing; (2) clinical manifestations: fever, cough, shortness of breath, sound of wet babble, decreased breath sounds, hypoxemia; (3) laboratory tests: C-reactive protein (CRP), white blood cell count (WBC), lymphocyte percentage (Lym), neutrophil percentage (N), Neutrophil Percentage (NEUT%), Neutrophil-to-Lymphocyte Ratio (NLR), Lymphocyte Percentage (LYMP%), Platelet-to-Lymphocyte Ratio (PLR), hemoglobin (Hb), blood platelets (PLTs), D-dimer (D-D), Ferritin (SF), Interleukin IL-6 (IL-6), Glucose (GLU), alanine transaminase (ALT), aspartate aminotransferase (AST), lactic dehydrogenase (LDH), creatine kinase (CK), creatine kinase MB (CK-MB), albumin (ALB), globulin (G), procalcitonin (PCT), erythrocyte sedimentation rate (ESR), immunoglobulin G (IgG), immunoglobulin M (IgM), immunoglobulin A (IgA), complement 3 (C3), complement 4 (C4); (4) imaging examination results: pleural effusion, atelectasis, pleural thickening, pulmonary emphysema, consolidation of lung.

### Statistical analysis

Statistical analyses were performed using R software (V.4.3.3, R Foundation for Statistical Computing, Vienna,

Austria) [12]. Categorical variables were analyzed using the $\chi 2$ test or Fisher's exact test. Normally distributed data were presented as the mean ± standard deviation (SD) and compared using the independent samples t-test. Skewed distribution data were expressed as the median (interquartile range), and the Mann–Whitney U rank-sum test was used for comparison between groups. To identify independent risk factors for the development of PB in children with adenoviral pneumonia, univariate analysis was initially performed. Variables with a *p*-value of $<0.05$ in the univariate analysis were subsequently included in a multivariate logistic regression. A stepwise selection method was applied to identify the final set of independent risk factors. Based on the results of the multivariate analysis, a nomogram was developed to estimate the individual risk of PB. To evaluate the model's stability and generalizability, an internal validation procedure was conducted. The predictive performance of the model was quantitatively assessed through the calculation of the area under the receiver operating characteristic (ROC) curve (AUC), a widely accepted metric for evaluating model discrimination capability. Model calibration was systematically examined using the Hosmer-Lemeshow goodness-of-fit test, complemented by the construction of calibration plots. These analytical approaches, in conjunction with ROC curve analysis, were employed to comprehensively assess the model's predictive accuracy and robustness. A two-tailed *p*-value of $<0.05$ was considered statistically significant.

## Results

Between January 2018 and April 2024, a total of 1715 children diagnosed with adenovirus pneumonia were hospitalized in the Department of Pediatric Respiratory Medicine at Tianjin Children's Hospital, Tianjin University, China. Among these, 406 children met the indications for bronchoscopy and underwent bronchoscopy with the consent of their guardians [11]. After excluding 290 children with co-infections from other pathogens, 164 children with adenovirus pneumonia were included in this study. This cohort comprised 25 children in the PB group and 139 in the non-PB group (Fig. 1).

In the PB group, there were 25 cases, comprising 13 males and 12 females, with ages ranging from 2 to 7 years and a median age of 3 years. The non-PB group included 139 cases, with 74 males and 65 females, aged 2 to 7 years, and a median age of 4 years. There was no statistically significant difference ($P>0.05$) when comparing the proportions of age, sex, height, weight, hospitalization time, duration of fever, fever peaks, extrapulmonary complications, cough, wet rales, hypoxemia, shortness of breath, dyspnea, history of eczema, history of wheezing, pulmonary atelectasis, pleural thickening, emphysema, and pulmonary solids. However, the proportions of children with decreased breath sounds and pleural effusions in the PB group were higher than those in the non-PB group, with the difference being statistically significant ($P<0.05$), as shown in Table 1.

In the comparison of various laboratory test indices, the levels of D-D, ESR, LDH, AST, and PCT were significantly higher in the PB group than in the non-PB group, with all differences being statistically significant ($P<0.05$). However, there were no statistically significant differences between the two groups for the following indices: CRP, WBC, HGB, PLT, N, NEUT%, NLR, Lym, LYMP%, PLR, SF, C3, C4, IgG, IgA, IgM, IgE, ALB, G, ALT, GLU, CK, CKMB, La, and IL-6 (all $P>0.05$). For detailed results, please refer to Table 2.

A multifactorial logistic analysis was performed to determine the independent risk factors affecting the occurrence of PB in children with adenovirus

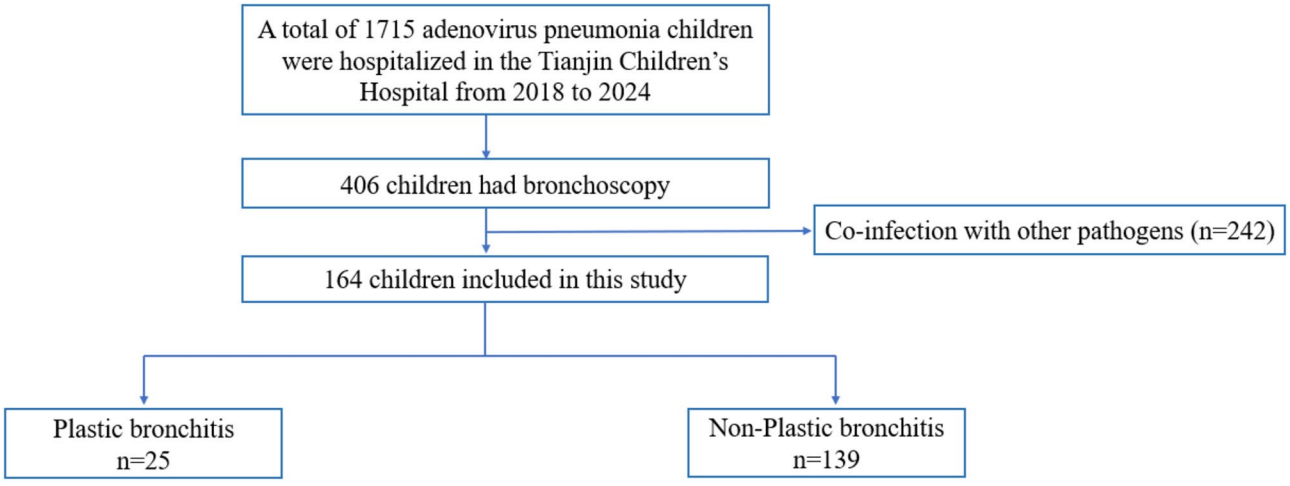


**Fig. 1** Flowchart of patient enrollment and selection process

**Table 1** Demographic characteristics、clinical features and radiological findings of non-PB and PB children with adenovirus pneumonia at admission

| Variable | Non-PB group, $n=139$ | PB group, $n=25$ | *P* |
|---|---|---|---|
| gender, girl, n(%) | 65 (47%) | 12 (48%) | 1.00 |
| age, years[b] | 4.0 (2.0 to 7.0) | 3.0 (2.0 to 7.0) | 0.79 |
| extrapulmonary complications, n(%) | 67 (49%) | 15 (60%) | 0.40 |
| seasons, spring, n(%) | 22 (16%) | 1 (4%) | 0.19 |
| summer, n(%) | 16 (12%) | 1 (4%) | |
| autumn, n(%) | 25 (18%) | 7 (28%) | |
| winter, n(%) | 75 (54%) | 16 (64%) | |
| expectoration, n(%) | 63 (46%) | 14 (56%) | 0.46 |
| history of eczema, n(%) | 45 (33%) | 7 (28%) | 0.83 |
| history of wheezing, n(%) | 13 (9%) | 1 (4%) | 0.62 |
| decreased breath sounds, n(%) | 19 (14%) | 16 (64%) | < 0.001 |
| sound of wet babble, n(%) | 129 (94%) | 24 (96%) | 0.98 |
| hypoxemia, n(%) | 14 (10%) | 2 (8%) | 1.00 |
| shortness of breath, n(%) | 18 (13%) | 4 (16%) | 0.94 |
| dyspnea, n(%) | 11 (8%) | 3 (12%) | 0.78 |
| pleural effusion, n(%) | 19 (14%) | 9 (36%) | 0.02 |
| atelectasis, n(%) | 20 (15%) | 6 (24%) | 0.37 |
| pleural thickening, n(%) | 42 (30%) | 5 (20%) | 0.41 |
| pulmonary emphysema, n(%) | 1 (1%) | 1 (4%) | 0.70 |
| consolidation of lung, n(%) | 60 (44%) | 15 (60%) | 0.19 |
| days of hospitalization, day[b] | 7.0 (5.0 to 10.0) | 8.0 (6.0 to 10.0) | 0.17 |
| fever days[b] | 3.0 (2.0 to 5.0) | 3.0 (2.0 to 4.0) | 0.97 |
| thermal peak,℃[b] | 39.4 (38.6 to 39.8) | 39.5 (39.0 to 40.1) | 0.25 |

[a]Mean (SD); [b]Median (IQR)

**Table 2** Laboratory indices of non-PB and PB children with adenovirus pneumonia

| Variable | Non-PB group, $n=139$ | PB group, $n=25$ | *P* |
|---|---|---|---|
| CRP(mg/L)[b] | 12.8 (3.8 to 28.0) | 12.4 (6.8 to 57.2) | 0.09 |
| WBC($10^9$/L)[b] | 8.3 (6.3 to 11.0) | 7.0 (5.5 to 10.1) | 0.14 |
| Hb(g/L) [a] | 123.1 (13.3) | 121.0 (12.9) | 0.47 |
| PLT($10^9$/L)[b] | 288.0 (211.0 to 387.0) | 272.0 (204.0 to 306.0) | 0.13 |
| N($10^9$/L)[b] | 5.2 (3.2 to 7.1) | 4.5 (3.5 to 6.3) | 0.66 |
| NEUT% [b] | 0.6 (0.5 to 0.7) | 0.7 (0.6 to 0.8) | 0.29 |
| NLR[b] | 2.3 (1.3 to 4.0) | 3.2 (1.6 to 3.8) | 0.53 |
| Lym($10^9$/L)[b] | 2.2 (1.5 to 3.2) | 1.7 (1.3 to 2.5) | 0.07 |
| LYMP% [b] | 0.3 (0.2 to 0.4) | 0.2 (0.2 to 0.3) | 0.71 |
| PLR[b] | 127.8 (85.0 to 190.3) | 137.8 (102.9 to 164.3) | 0.63 |
| D-D(mg/L) [b] | 0.6 (0.2 to 1.0) | 1.2 (0.8 to 2.7) | < 0.001 |
| ESR(mm/h) [b] | 30.0 (17.0 to 42.0) | 35.0 (27.0 to 55.0) | 0.01 |
| SF(ng/mL) [b] | 141.2 (93.2 to 248.4) | 245.5 (88.9 to 815.2) | 0.08 |
| C3(g/L) [a] | 1.2 (0.3) | 1.2 (0.3) | 0.50 |
| C4(g/L) [b] | 0.3 (0.2 to 0.4) | 0.4 (0.2 to 0.4) | 0.24 |
| IgG(g/L) [b] | 8.4 (6.5 to 10.8) | 8.6 (7.1 to 11.4) | 0.29 |
| IgA(g/L) [b] | 1.0 (0.6 to 1.7) | 1.1 (0.5 to 1.8) | 0.99 |
| IgM(g/L) [b] | 1.2 (0.9 to 1.8) | 1.1 (0.8 to 1.9) | 0.80 |
| IgE(IU/mL) [b] | 100.0 (31.1 to 272.8) | 79.8 (34.7 to 244.4) | 0.68 |
| ALB(g/L) [a] | 41.3 (4.1) | 40.4 (5.4) | 0.10 |
| G(g/L) [b] | 24.9 (22.3 to 28.4) | 27.1 (24.1 to 30.7) | 0.12 |
| ALT(U/L) [b] | 13.0 (10.0 to 19.0) | 14.0 (11.0 to 18.0) | 0.16 |
| LDH(U/L) [b] | 400.0 (311.0 to 543.0) | 497.0 (374.0 to 662.0) | 0.01 |
| AST(U/L) [b] | 34.0 (25.0 to 45.0) | 42.0 (35.0 to 58.0) | 0.008 |
| GLU(mmol/L) [b] | 5.1 (4.5 to 5.8) | 5.2 (4.8 to 6.3) | 0.17 |
| PCT(ng/ml) [b] | 0.2 (0.1 to 0.4) | 0.4 (0.2 to 0.7) | 0.002 |
| CK(U/L) [b] | 86.0 (58.0 to 146.0) | 83.0 (62.0 to 220.0) | 0.52 |
| CK-MB(U/L) [b] | 4.0 (3.9 to 8.0) | 4.0 (3.9 to 9.0) | 0.98 |
| La(mmol/L) [b] | 2.6 (2.3 to 3.5) | 2.8 (2.4 to 3.0) | 0.60 |
| IL-6(pg/ml) [b] | 31.8 (15.0 to 52.5) | 40.6 (14.7 to 73.7) | 0.28 |

Abbreviations: CRP: C-reactive protein, WBC: White blood cell, N: Peripheral neutrophils, Lym: Peripheral lymphocytes, NEUT%: Neutrophil Percentage, LYMP%: Lymphocyte Percentage, NLR: Neutrophil-to-lymphocyte ratio, E: Peripheral eosinophils, Hb: Hemoglobin, PLT: Platelet, PLR: Platelet lymphocyte rate, D-D: D-Dimer, ESR: Erythrocyte Sedimentation Rate, SF: Ferritin, C3: Complement 3, C4: Complement 4, IgG: Immunoglobulin G, IgA: Immunoglobulin A, IgM: Immunoglobulin M, IgE: Immunoglobulin E, PCT: Procalcitonin, ALB: Albumin, LDH: Lactic dehydrogenase, CK: Creatine kinase, CKMB: Creatine kinase isomer-MB, IL-6: Interleukin IL-6

[a]Mean (SD)

[b]Median (IQR)

pneumonia, using the indicators that were statistically different in the above univariate analysis as independent variables and the occurrence of PB as the dependent variable. The results indicated that decreased breath sounds, higher levels of D-D, and LDH, were independent risk factors for the development of PB in children with adenovirus pneumonia (Table 3).

A nomogram assessing the risk of PB was developed using the three risk factors identified from the logistic regression analysis (Fig. 2a). The AUC of the nomogram was 0.904. This nomogram assigns a weighted score to each independent risk factor, with the highest possible score being 100 points and the predicted incidence of PB ranging from 0.01 to 0.95. A higher total score, calculated from the sum of the points assigned to each high-risk factor, corresponds to a higher risk of PB occurrence. The Hosmer–Lemeshow test was employed to validate the model, yielding a result of $p=0.515$, $X^2=7.207$, indicating that the information in the current data had been fully utilized. The AUC value demonstrated that the predictive power of the model in the primary cohort was 0.904 (95% CI 0.8–0.960) (Fig. 2b). The calibration chart indicated that the nomogram had a good fit for predicting PB incidence in RMPP patients (Fig. 2c).

## Discussion

In recent years, the number of case reports on PB has increased alongside a growing understanding of the condition and the widespread adoption of fiberoptic

**Table 3** Multivariate logistic regression for predicting PB

| Variable | OR | 95% CI | *P* |
|---|---|---|---|
| Diminished breath sounds | 17.77 | 5.92–62.11 | < 0.001 |
| pleural effusion | 0.71 | 0.14–3.06 | 0.66 |
| D-D(mg/L) | 2.12 | 1.39–3.34 | < 0.001 |
| ESR(mm/h) | 1.04 | 0.99–1.08 | 0.05 |
| LDH(U/L) | 1.01 | 1.00-1.01 | 0.003 |
| AST(U/L) | 0.95 | 0.90–1.01 | 0.06 |
| PCT(ng/ml) | 1.45 | 0.86–1.89 | 0.11 |

Abbreviations: D-D: D-Dimer, ESR: erythrocyte sedimentation Rate, LDH: Lactic dehydrogenase, AST: aspartate transaminase, PCT: procalcitonin, OR Odd ratio, CI Confidence interval

bronchoscopy techniques [13, 14]. In China, PB is primarily observed in respiratory infectious diseases and is particularly common among preschool and school-age children. While Mycoplasma pneumoniae remains the most frequent pathogen causing PB, there has been a notable rise in reports of PB linked to HAdV infections in recent years [7–9]. Therefore, the objective of our study was to evaluate the clinical characteristics of children with adenovirus pneumonia and identify the risk factors associated with the development of concurrent PB. In this study, logistic regression analysis identified diminished breath sounds, D-D level, and LDH level as independent risk factors for PB in children with adenovirus pneumonia. Based on these factors, a predictive nomogram was developed for clinical use.

Currently, there are limited studies examining the risk factors for the occurrence of PB in children with adenovirus pneumonia. Several previous studies have identified diminished breath sounds as a significant independent risk factor for PB in these children [8, 15, 16]. Additionally, other studies have indicated that elevated levels of inflammatory markers and LDH, as well as coagulation abnormalities, are prevalent in the PB group compared to the non-PB group [16–18]. The above findings are highly consistent with our results.

Human airway epithelial cells provide an ideal environment for the growth and replication of HAdV [19]. HAdV can directly invade these cells and, when released in large quantities, can induce a storm of inflammatory factors, ultimately leading to cell lysis and death [20, 21]. In severe cases of HAdV infection, there may be shedding of airway epithelial cells, abnormal accumulation of inflammatory cells in the airways, and mucus overproduction. These factors can lead to obstruction of the bronchial lumen and even the development of PB [7]. Obstruction of the bronchial lumen interferes with the volume and rate of gas entry into the alveoli, potentially resulting in auscultatory findings of diminished breath sounds. This obstruction can also lead to serious pulmonary complications, such as pulmonary consolidation and atelectasis [14, 22].

D-D is a degradation product of fibrin, generated through the action of fibrinolytic enzymes, and it serves as a key indicator for assessing coagulation dysfunction. In a study characterizing 43 children with PB, Huang et al. [8] found that 67.4% of the children exhibited elevated D-D levels, a phenomenon particularly common in children with HAdV infection. The potential reason for this may be that HAdV infection causes damage to the endothelial cells of blood vessels, activating the coagulation system and creating a hypercoagulable state [23, 24]. This state alters the microcirculation of lung tissues, leading to the accumulation of inflammatory factors, cells, and mucus within the airways, ultimately resulting in PB.

LDH is an enzyme found in the cytoplasm of all cells and serves as an easily detectable, nonspecific indicator of inflammation, reflecting tissue damage. LDH is released into the bloodstream when cell lysis or cell membrane damage occurs, making it a key marker for assessing the severity of infection and the state of inflammatory disease [8]. Previous studies have demonstrated a significant correlation between LDH levels and adenovirus infection in children [25]. Additionally, LDH levels can reflect the severity of pneumonia. Research by scholars such as Lai and He has shown that LDH levels are significantly elevated in children with severe adenovirus pneumonia, further validating its usefulness as an indicator of disease severity [26–28]. Several studies have already highlighted that LDH levels in children with refractory Mycoplasma pneumoniae pneumonia are significant risk factors for constructing a nomogram to predict PB [22, 29, 30]. Our study further confirms that LDH levels can also be used as an independent risk factor for predicting the occurrence of PB in children with adenovirus pneumonia. This phenomenon may be related to the localized and systemic excessive inflammatory response in the lungs of children with adenovirus pneumonia. This inflammatory response not only damages the cells but also significantly increases cell membrane permeability, leading to the release of intracellular LDH into the bloodstream and triggering elevated LDH levels. This finding not only broadens the application of LDH in different types of pneumonia but also provides a new reference for clinical practice, potentially aiding in the early diagnosis and improved therapeutic efficacy for children with adenovirus pneumonia.

Given that diminished breath sounds, LDH, and D-dimer are all clinically readily available, our predictive model offers substantial clinical value in the early detection of PB in children with adenoviral pneumonia. By identifying key risk factors, the model enables clinicians to detect high-risk patients at an early stage, facilitating timely interventions, such as bronchoscopy, which is essential for both diagnosing and managing PB and preventing severe complications, including hypoxemia and

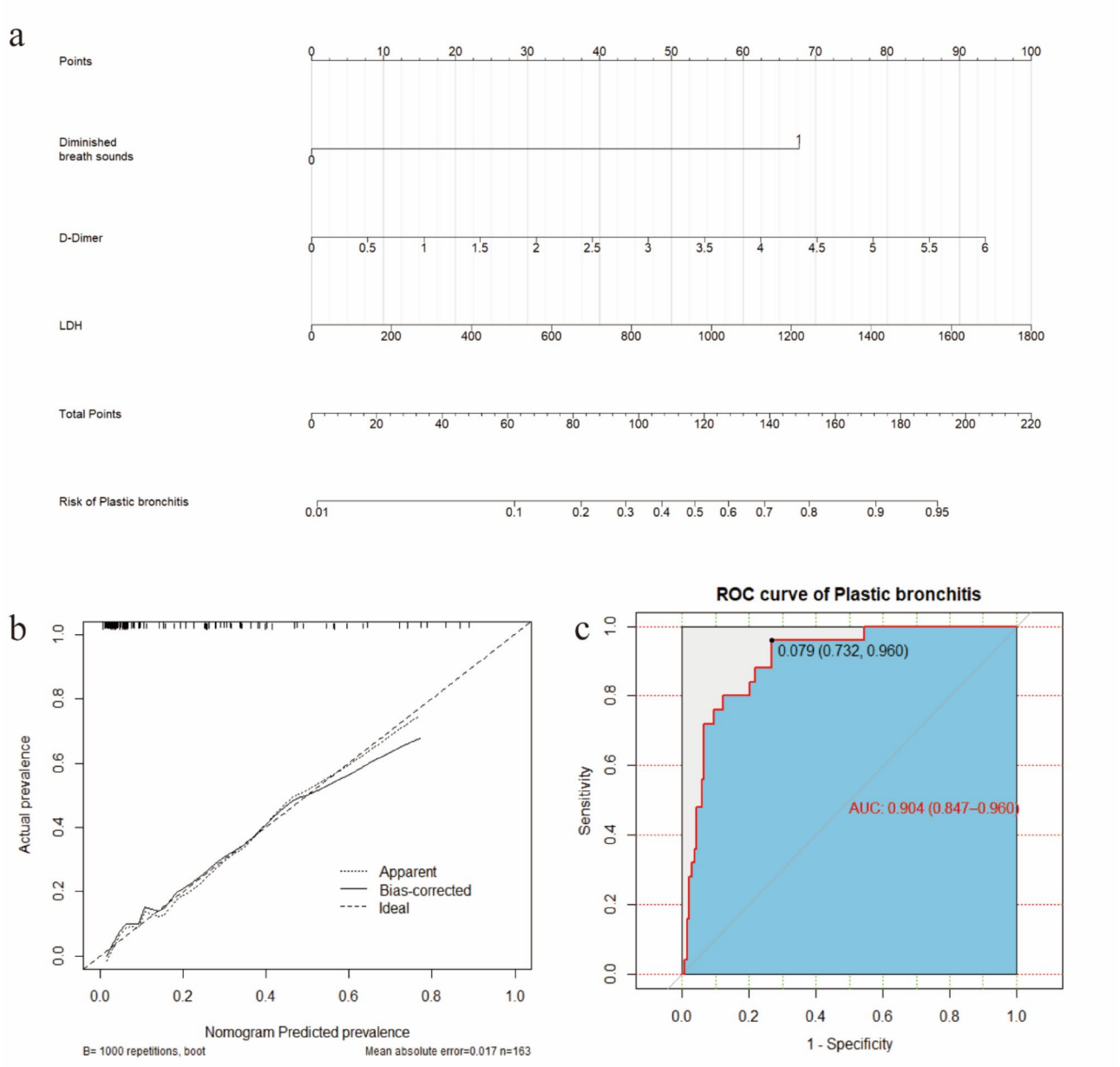


**Fig. 2** **a**: Nomogram of regression equations for calculating risk score and predicting risk of PB in adenoviral pneumonia patients. **b**: Nomogram calibration curve. Horizontal axis indicates risk of PB occurrence predicted by nomogram. Vertical axis represents actual observed risk of PB occurrence. **c**: Receiver operating characteristic (ROC) curve analysis of main cohort

respiratory failure. In clinical settings with limited access to advanced diagnostic tools, this model serves as a crucial tool for early risk stratification, assisting healthcare providers in prioritizing patients most likely to develop PB. Moreover, the nomogram provides a straightforward, practical approach to risk assessment, supporting more informed decision-making and efficient allocation of resources. Ultimately, the implementation of this model may enhance patient management by enabling early identification and intervention, thereby improving the prognosis of children with adenoviral pneumonia.

Our study has several limitations. To begin with, being a single-center study, the generalisability of our findings remains uncertain. Secondly, the number of study children included in this study was limited, although we included all children with adenoviral pneumonia with PB since the introduction of bronchoscopy in our hospital. The relatively small sample size of the PB group may have introduced bias in model training and increased the risk of overfitting. While we employed internal validation through bootstrapping to enhance model stability, external validation using independent cohorts is warranted

to further confirm its predictive performance. Finally, our study did not identify the type of HAdV, and which of these may be particularly associated with the development of PB. Future multicenter studies with larger sample sizes and external validation are needed to strengthen the robustness and applicability of our findings.

## Conclusions

In a nutshell, our study developed a nomogram incorporating three factors—diminished breath sounds, D-D levels, and LDH levels—to predict the risk of PB in children with adenovirus pneumonia. The nomogram demonstrated strong performance and may assist in clinical identification and decision-making for patients with PB resulting from adenovirus pneumonia.

**Acknowledgements**
We thank all participants and staff of this study and the physicians at the Tianjin Children's Hospital affiliated to Tianjin University.

**Author contributions**
YW, YJ and RX analyzed and interpreted the patient data. YW, YJ and RX were major contributors in writing the manuscript. XC, NZ and WL provided the acquisition of data. XW and YZ were major contributors in reviewing and editing the manuscript. All authors read and approved the final manuscript.

**Funding**
This study was financially supported by the Natural Science Foundation of Tianjin Municipality (20JCZDJC00630) and Tianjin Key Medical Discipline (Specialty) Construction Project (No. TJYXZDXK-040 A).

**Data availability**
The datasets generated and/or analysed during the current study are not publicly available due [REASON WHY DATA ARE NOT PUBLIC] but are available from the corresponding author on reasonable request.

## Declarations

**Ethics approval and consent to participate**
This study was approved by the Ethic Committee of the Tianjin Children's Hospital (KY2020-19). All procedures performed in the studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Written informed consent was obtained from individual participants and their legal guardians.

**Consent for publication**
Not applicable.

**Competing interests**
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.

Received: 15 February 2025 / Accepted: 12 April 2025
Published online: 27 April 2025

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