Journal of Inflammation Research

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Exploring Clinical Characteristics and Risk Factors of *Mycoplasma pneumoniae*-Induced Segmental/Lobar Pneumonia in Chongqing, China, During 2023: A Single-Center Retrospective Cohort Study of Hospitalized Children

Yaozheng Ling[1,2], Yu Zhao[1,2], Chenghao Mei[1,2], Fang Deng[1,2], Zhou Fu[1,2], Gang Geng[1,2]

[1]Department of Respiratory Medicine, Children's Hospital of Chongqing Medical University, National Clinical Research Center for Children and Adolescents' Health and Diseases, Ministry of Education Key Laboratory of Child Development and Disorders, Chongqing Key Laboratory of Child Rare Diseases in Infection and Immunity, Chongqing, People's Republic of China; [2]Intelligent Application of Big Data in Pediatrics Engineering Research Center of Chongqing Education Commission of China, Chongqing, People's Republic of China

Correspondence: Gang Geng, Department of Respiratory Medicine, Children's Hospital of Chongqing Medical University, Chongqing, People's Republic of China, Email 285544470@qq.com

Purpose: *Mycoplasma pneumoniae* (MP)-segmental/lobar pneumonia represents a growing proportion of *Mycoplasma pneumoniae* pneumonia (MPP) in children. We aimed to identify the clinical characteristics and risk factors for MP-segmental/lobar pneumonia in children to facilitate early diagnosis and intervention.

Methods: We conducted a retrospective cohort study at a tertiary pediatric center in Chongqing, China, involving 1406 children hospitalized for MPP from January to December 2023. Based on imaging findings, patients were categorized into MP-ordinary pneumonia group and MP-segmental/lobar pneumonia group. Clinical data were compared, and univariate and multivariate logistic regression analyses were performed to identify independent risk factors. The predictive performance was assessed using receiver operating characteristic (ROC) curves.

Results: The MP-segmental/lobar pneumonia group exhibited a significantly longer duration of pre-admission fever, longer hospital stays, and higher rates of bronchoscopy, respiratory failure, and pulmonary complications. (all $P < 0.05$). Logistic regression analysis identified the following independent risk factors for MP-segmental/lobar pneumonia: sex, days with fever prior to admission, C-reactive protein (CRP), D-dimer, lactate dehydrogenase (LDH), platelet–lymphocyte ratio (PLR), and platelet (PLT). ROC curve analysis identified five primary predictive indicators (days with fever prior to admission >5.5 days, D-dimer >0.539mg/L, CRP >11.33mg/L, LDH >309.5U/L, PLR >155.815) and two secondary predictive indicators (PLT >340.5×10^9/L, female sex) based on predicted efficacy. The combined use of these seven indicators further enhanced predictive performance (AUC = 0.741, 95% CI: 0.715–0.766).

Conclusion: MP-segmental/lobar pneumonia exhibits more pronounced clinical symptoms and elevated inflammatory markers. The seven clinical indicators—days with fever prior to admission, CRP, D-dimer, LDH, PLR, PLT and female sex—serve as useful predictive markers for segmental/lobar pneumonia caused by MP. These indicators aid clinicians in the early diagnosis and intervention of MPP, thereby preventing its progression to segmental/lobar pneumonia.

Keywords: *Mycoplasma pneumoniae*, segmental/lobar pneumonia, clinical features, risk factors, predictive indicators

Introduction

Mycoplasma pneumoniae (MP) is an important pathogen responsible for respiratory infections in children. It is one of the smallest known self-replicating organisms, possessing a streamlined and highly stable genome, lacking a cell wall, and exhibiting unique disease manifestations.[1] Over recent years, *Mycoplasma pneumoniae* infections has gradually increased in community-acquired pneumonia (CAP) among children, accounting for approximately 10% to 30% of all CAP and

https://doi.org/10.2147/JIR.S577148
Received: 31 October 2025
Accepted: 6 December 2025
Published: 8 January 2026

upper respiratory tract (URT) infection cases.[2] MP is not only a common pathogen causing CAP in school-aged and preschool children, but is also frequently observed in infants and young children.[3] Although most cases of *Mycoplasma pneumoniae* pneumonia (MPP) are mild and self-limiting, a minority of pediatric patients may develop severe and life-threatening pneumonia accompanied by extrapulmonary manifestations affecting virtually all organ systems.[4,5]

Segmental/lobar pneumonia is a type of CAP in children. It is an acute inflammatory lung disease caused by pathogenic microbial infection, primarily affecting more than one segment of lung tissue and potentially progressing to lobar involvement after fusion.[6] In recent years, the incidence of segmental/lobar pneumonia in children caused by MP infection has been gradually increasing.[7] Typical clinical symptoms include cough, fever, shortness of breath, among others. When the disease further progresses, it may be accompanied by atelectasis, pulmonary necrosis, pulmonary abscess, and in severe cases, complications such as acute respiratory distress syndrome, multiple organ dysfunction syndrome, or respiratory failure.[8] Based on the current spatio-temporal-regional epidemiology of MP globally, MP infection poses varying degrees of healthcare burden on medical systems in all countries.[9] For example, in China during 2023, MP infection rates were high in many regions. Furthermore, in this epidemic, the incidence of acute, severe, and critical cases was significantly elevated.[10]

MPP is not accompanied by segmental/lobar lesions in the early stages. Timely and effective treatment of MPP before it progresses to segmental/lobar pneumonia can prevent its progression to refractory pneumonia, necrotizing pneumonia, and other acute and critical conditions, thereby minimizing both pulmonary and extrapulmonary complications. Previous studies have reported that in MP-segmental/lobar pneumonia, pediatric patients exhibit significantly longer fever duration, hospital stays, and higher rates of pleural effusion and co-infections compared to those with MP-ordinary pneumonia.[7,11] Additionally, inflammatory markers such as C-reactive protein, white blood cell, erythrocyte sedimentation rate, platelet, etc., were also significantly elevated. Therefore, early identification of risk factors for MPP progression to segmental/lobar pneumonia is important for the timely control of disease progression, reduction of serious pulmonary complications, and improvement of prognosis. In this study, we retrospectively analyzed the clinical characteristics of children with segmental/lobar pneumonia due to MP infection, identified the risk factors for progression of MPP to MP-segmental/lobar pneumonia, and provided evidence for early intervention and treatment of MPP for clinical pediatricians.

Methods

Study Design and Population

This retrospective study included children diagnosed with MPP who were admitted to the Children's Hospital of Chongqing Medical University between January and December 2023. These children were screened according to the inclusion and exclusion criteria. They were categorized into an MP-ordinary pneumonia group and an MP-segmental /lobar pneumonia group based on their imaging manifestations. This study was conducted in accordance with the ethical principles of the Declaration of Helsinki, in compliance with the RECORD (REporting of studies Conducted using Observational Routinely collected health Data) guidelines and was approved by the Institutional Review Board (IRB) of the Children's Hospital of Chongqing Medical University (No. 2024–440).

Inclusion Criteria: ① All included children met the diagnostic criteria for MPP as stipulated in the Chinese Guidelines for the Diagnosis and Treatment of *Mycoplasma Pneumoniae* Pneumonia in Chinese Children (2023 Edition): clinical presentation of fever and cough, which may be accompanied by headache, rhinorrhea, or sore throat, with imaging demonstrating inflammatory involvement of the bronchi, bronchioles, alveoli, or pulmonary interstitium, and combining either or both of the following: (i) a single serum MP antibody titer ≥1:160 (via passive agglutination method) or a ≥4-fold increase in MP antibody titer in paired sera; (ii) positive MP-DNA or MP-RNA testing. ② Meeting the diagnostic criteria for segmental/lobar pneumonia: presence of clinical symptoms of respiratory infection (eg, fever and/or cough) with chest imaging revealing homogeneous consolidative shadows in one or more pulmonary segments or lobes.[6,8] ③ 29 days ≤ age ≤ 14 years. ④ Availability of complete clinical data.

Exclusion Criteria: ① A history of underlying respiratory diseases, such as pulmonary tumors, bronchial asthma, bronchial foreign bodies, hospital-acquired pneumonia, pulmonary tuberculosis, or recurrent lower respiratory tract infections. ② Patients in the recovery phase, defined as a disease duration of over 4 weeks, accompanied by a stable

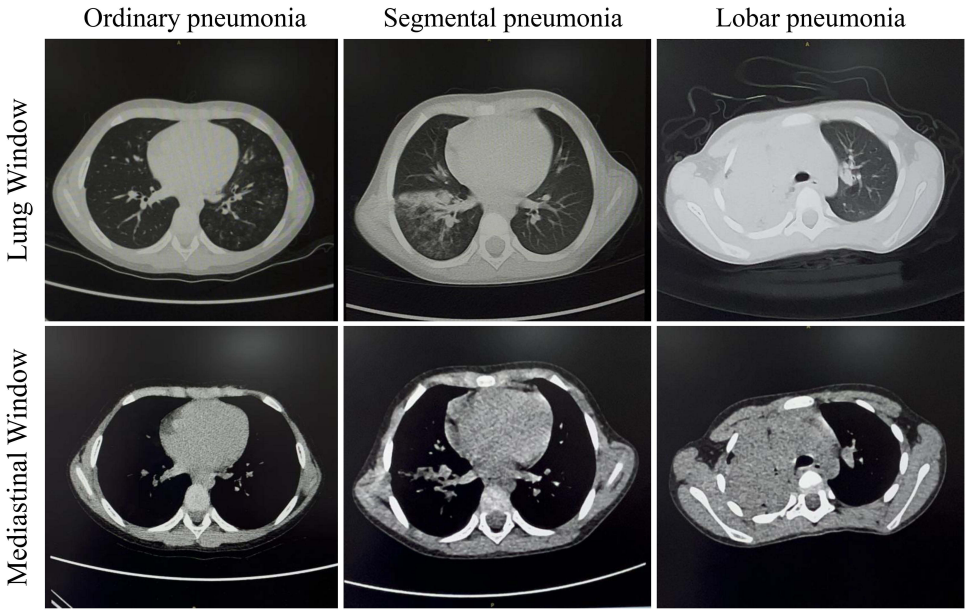

Figure 1 Characteristics of pulmonary imaging of MP-ordinary pneumonia, MP-segmental pneumonia, and MP-lobar pneumonia.
Abbreviation: MP, *Mycoplasma pneumoniae*.

temperature at least 1 week, or imaging showing the lesions with clear improvement. ③ Individuals exist congenital developmental abnormalities, including cardiovascular system (eg, atrial septal defect, ventricular septal defect, patent ductus arteriosus) and respiratory system (eg, tracheoesophageal fistula, airway malformations). ④ Individuals with congenital immunodeficiency, genetic mutations, hematologic malignancies and solid tumors. ⑤ A recent history of major trauma, emergency surgery, or being in the postoperative period. ⑥ Extensive missing clinical data or transfer to another hospital during the course of treatment.

Grouping Criteria: Patients were classified into two groups based on pulmonary imaging findings: the ordinary pneumonia group and the segmental/lobar pneumonia group. As shown in Figure 1, CT images of ordinary pneumonia typically present scattered linear or patchy opacities without consolidation of an entire segment or lobe. Segmental pneumonia is characterized by inflammatory lesions confined to one or more pulmonary segments. Progression to lobar pneumonia involves consolidation of an entire lobe, manifesting as a homogeneous high-density shadow on imaging.

Data Collection

This study systematically collected clinical information (sex, age, days of illness prior to admission, days of cough prior to admission, days of fever prior to admission, length of hospital stays, costs of hospitalization, bronchoscopy rate, and the incidence of respiratory failure and pulmonary complications, etc) from pediatric patients following admission. This included basic information, pre-admission medical history, and laboratory test results [white blood cell count (WBC), C-reactive protein (CRP), serum amyloid A (SAA), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), platelet (PLT), procalcitonin (PCT), fibrinogen (FIB), D-dimer, lactate dehydrogenase (LDH), alanine aminotransferase (ALT), aspartate aminotransferase (AST), total protein (TP), prealbumin (PAB), albumin (ALB), globulin (GLB), and albumin-to-globulin ratio (A/G), etc.] obtained within 24 hours after admission, as well as results of pulmonary imaging examinations (pre-admission outpatient examination or within 48 hours after admission), bronchoscopy findings, comprehensive complication assessment results, and pathogen testing results.

Microbial cultures were performed on blood, bronchoalveolar lavage fluid (BALF), and sputum specimens. Serologic testing for MP was conducted using the passive agglutination method. Molecular analyses included testing for MP antibiotic resistance genes (A2063G/A2064G) via fluorescent probe polymerase chain reaction and detection of respiratory pathogens in BALF samples using a targeted amplification assay. Additionally, nasopharyngeal swabs were analyzed

https://doi.org/10.2147/JIR.S577148

for seven common respiratory pathogens by immunofluorescence assay. All pathogen detection and laboratory analyses, including measurements of WBC, CRP, D-dimer, LDH and etc., were performed by the Department of Clinical Laboratory at the Children's Hospital of Chongqing Medical University, a facility operating in compliance with the ISO-15189 quality standard.

Statistical Analysis

Statistical analysis was conducted using SPSS 26.0 software. Quantitative data from both groups that met the criteria of normal distribution and homogeneity of variance were expressed as mean±standard deviation, with intergroup comparisons made using the independent samples *t*-test. Non-normally distributed data were described using median and interquartile range (IQR), and compared between groups using the Mann–Whitney *U*-test. Categorical variables were expressed as n (%), with intergroup comparisons performed using the Chi-square test or Fisher's exact test. Subsequently, univariate logistic regression analysis and multivariate logistic regression analysis were employed to identify independent risk factors for segmental/lobar pneumonia associated with *Mycoplasma pneumoniae* infection. Receiver operating characteristic (ROC) curves were generated using R software (version 4.3.1) and the RStudio programming environment to evaluate the predictive performance of each risk factor. $P < 0.05$ was considered statistically significant.

Results

Comparison of Clinical Characteristics Between MP-Ordinary Pneumonia and MP-Segmental/Lobar Pneumonia

A retrospective study was conducted on 1956 pediatric patients diagnosed with MPP at the Children's Hospital of Chongqing Medical University between January and December 2023. After screening based on the inclusion and exclusion criteria (Figure 2), 1406 children with MPP were enrolled in the study. Based on imaging findings,

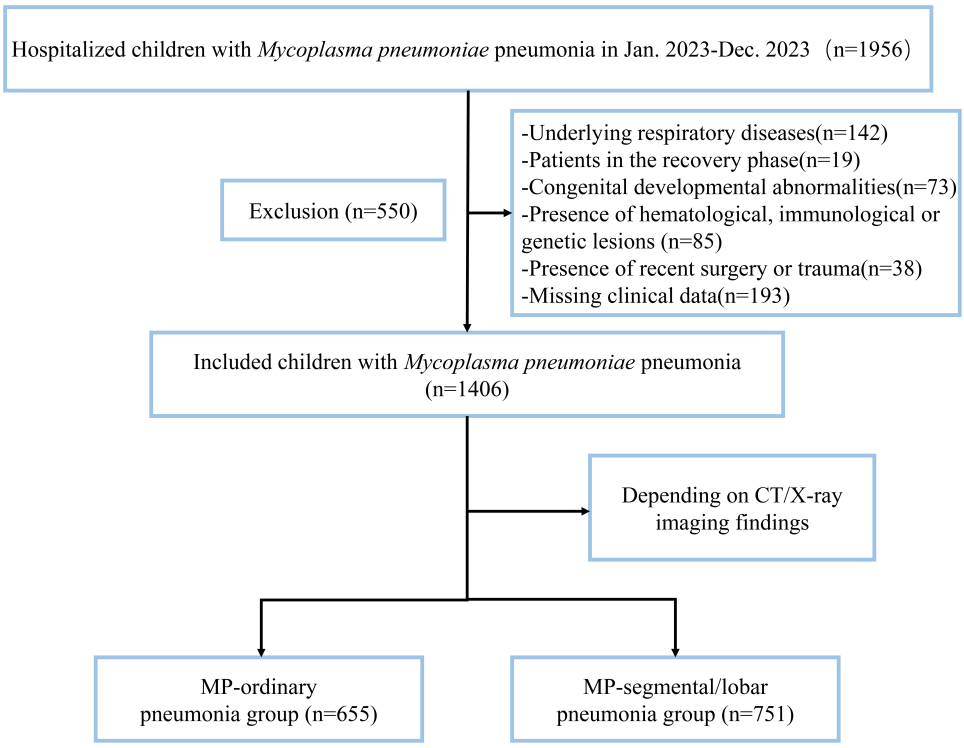

Figure 2 Flowchart of the study.
Abbreviations: MP, *Mycoplasma pneumoniae*; Jan., January; Dec., December; CT, Computed Tomography.

Table 1 Differences in Clinical Features Between MP-Ordinary Pneumonia and MP-Segmental/Lobar Pneumonia

Clinical information	Total n=1406	Group		
		MP-Ordinary Pneumonia n=655	MP-Segmental/Lobar Pneumonia n=751	*P*
Age(years)	5.86±2.55	5.74±2.62	5.97±2.49	0.094
Sex [n (%)]				<0.001
Male	693(49.3%)	363(55.4%)	330(43.9%)	
Female	713(50.7%)	292(44.6%)	421(56.1%)	
Days of illness prior to admission (days)	8(6, 10)	8(6. 10)	8(7, 10)	0.535
Days with fever prior to admission(days)	6(4, 8)	6(4, 8)	7(5, 8)	<0.001
Days with cough prior to admission(days)	7(5, 10)	7(5, 10)	7(5, 9)	0.045
Length of hospital stays (days)	6(5, 8)	6(4, 7)	7(5, 8)	<0.001
Costs of hospitalization (yuan)	8441.68(6514.125, 10890.44)	7815.8(5857.43, 9567.17)	10010.08(8036.65, 12287.76)	<0.001
Flexible Fiberoptic Bronchoscope Ratio	640(45.5%)	159(24.3%)	481(64.0%)	<0.001
Combined infection rate				0.479
N	1236(88.7%)	580(89.8%)	656(87.7%)	
Combined bacterial infection	113(8.1%)	48(7.4%)	65(8.7%)	
Combined viral infection	40(2.9%)	17(2.6%)	23(3.1%)	
Mixed infection (bacteria + virus)	5(0.4%)	1(0.2%)	4(0.5%)	
Combined respiratory failure rate	142(10.1%)	43(6.6%)	99(13.2%)	<0.001
Complications [n (%)]				<0.001
N	1059(75.3%)	577(88.1%)	482(64.2%)	
Pleural effusion	127(9%)	22(3.4%)	105(14%)	
Atelectasis	144(10.2%)	47(7.2%)	97(12.9%)	
Necrotizing pneumonia	26(1.8%)	3(0.5%)	23(3.1%)	
Plastic bronchitis	5(0.4%)	0	5(0.7%)	
Pleural effusion + Atelectasis	45(3.2%)	6(0.9%)	39(5.2%)	

Abbreviations: m, Male; f, Female; N, negative, MP, *Mycoplasma pneumoniae*.

patients were categorized into the MP-ordinary pneumonia group (n=655) and the MP-segmental/lobar pneumonia group (n=751). The MP-segmental/lobar pneumonia group had a significantly higher proportion of females (421, 56.1%) than the MP-ordinary pneumonia group (292, 44.6%) (P<0.001). Additionally, the MP-segmental/lobar pneumonia group exhibited significant increases compared to the MP-ordinary pneumonia group in the following variables: fever duration prior to admission, hospitalization costs, length of stay, bronchoscopy rate, and the incidence of respiratory failure and pulmonary complications (all P<0.001). There were no statistically significant differences between the two groups in terms of age (P=0.094), days of illness prior to admission (P=0.535), and combined infections rate (P=0.479) (Table 1).

Laboratory Findings of MP-Ordinary Pneumonia and MP-Segmental/Lobar Pneumonia

The MP-segmental/lobar pneumonia group demonstrated significantly higher levels of PLT, NLR, PLR, CRP, PCT, FIB, D-dimer, LDH, and ALT compared to the MP-ordinary pneumonia group (P< 0.05). Conversely, the MP-segmental/lobar pneumonia group had significantly lower levels of TP, PAB, ALB, and A/G compared to the MP-ordinary pneumonia group (P< 0.05). No significant differences were observed between the two groups for the following laboratory parameters: WBC (P=0.176), SAA (P=0.092), GLB, (P=0.989), AST, (P=0.183), AST/ALT ratio (P=0.443), *Mycoplasma pneumoniae* A2063G/A2064G drug resistance gene ratio (P=0.239), and bronchoalveolar lavage fluid (BALF) cell classification ratios-(neutrophils (P=0.66), lymphocytes (P=0.401), macrophages (P=0.394)), as well as BALF viscosity (P=0.424) (Table 2).

https://doi.org/10.2147/JIR.S577148

Table 2 Differences in Laboratory Findings Between MP-Ordinary Pneumonia and MP-Segmental/Lobar Pneumonia

Laboratory Tests	Total n=1406	Group		
		MP-Ordinary Pneumonia n=655	MP-Segmental/Lobar Pneumonia n=751	*P*
WBC (×10^9/L)	7.69±2.76	7.58±2.69	7.78±2.81	0.176
PLT(×10^9/L)	371.95±119.91	361.69±116.95	380.90±121.81	0.003
NLR	1.78(1.15, 2.61)	1.89(1.37, 2.62)	1.98(1.41, 2.93)	<0.001
PLR	151.40(118.45, 195.28)	151.28(116.96, 192.27)	168.5(131.54, 216.4)	<0.001
CRP (mg/L)	10(5.32, 17.36)	9.37(5.39, 14.56)	10.76(5.55, 19.6)	<0.001
PCT (ng/mL)	0.09(0.05, 0.16)	0.08(0.05, 0.13)	0.1(0.06, 0.17)	<0.001
SAA (mg/L)	60.28(32.91, 131.9)	57.14(32, 119.45)	74.6(35.29, 139.17)	0.092
FIB (g/L)	4.28±0.94	4.10±0.90	4.44±0.95	<0.001
D-dimer (mg/L)	0.54(0.36, 0.95)	0.43(0.29, 0.7)	0.67(0.43, 1.14)	<0.001
LDH (U/L)	282(248, 327)	275(244, 315)	291(249, 337)	<0.001
TP (g/L)	68.25±5.1	68.93±5.00	67.66±5.12	<0.001
PAB (mg/L)	110(93, 140)	110(93, 130)	105(92, 130)	<0.001
ALB (g/L)	41.81±3.48	42.50±3.09	41.20±3.68	<0.001
GLB (g/L)	26.47±3.91	26.48±4.0	26.47±3.83	0.989
A/G	1.61±0.27	1.64±0.28	1.59±0.27	<0.001
AST (U/L)	32(27, 38)	31(27, 36)	38(27, 38)	0.183
ALT (U/L)	15(12, 20)	14(11, 17)	15(11, 20)	0.008
AST/ALT	2.23±1.02	2.26±0.85	2.21±1.15	0.443
A2063G/A2064G Drug Resistance Gene Ratio				0.239
Negative	230(29.4%)	104(31.7%)	126(27.8%)	
Positive	551(70.6%)	224(68.3%)	327(72.2%)	
BALF-Neutrophils ratio (%)	66(49.75, 78)	65(49, 77)	66(50, 78)	0.66
BALF-Lymphocyte ratio (%)	15(9, 23)	16(9, 23)	15(9, 23)	0.401
BALF-Macrophage ratio (%)	11(5, 20)	10(4, 20)	11(5, 20)	0.394
BALF-viscosity				0.424
Non-mucous	255(43.2%)	73(48.3%)	182(41.5%)	
Small amount of mucus	158(26.8%)	39(25.8%)	119(27.1%)	
Flocculent mucus	97(16.4%)	23(15.2%)	74(16.9%)	
Viscous mucus	80(13.6%)	16(10.6)	64(14.6%)	

Abbreviations: MP, *Mycoplasma pneumoniae*; WBC, white blood cell; CRP, C-reactive protein; SAA, serum amyloid A; NLR, neutrophil-to-lymphocyte ratio; TP, total protein; PLT, platelet; PCT, procalcitonin; FIB, fibrinogen; LDH, lactate dehydrogenase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; PLR, platelet-to-lymphocyte ratio; PAB, prealbumin; ALB, albumin; GLB, globulin; A/G, albumin-to-globulin ratio; BALF, bronchoalveolar lavage fluid.

Comparison of Pathogen Detection results for MP-Ordinary Pneumonia and MP-Segmental/Lobar Pneumonia

The viral nasopharyngeal swab nucleic acid testing targeted seven common respiratory viruses (Figure 3A and B). In the MP-segment/lobar pneumonia group (719 tested), 652 cases were negative. Among positive results, respiratory syncytial virus (RSV) (14 cases) was most prevalent. In the MP-ordinary pneumonia group (617 tested), 599 cases were negative. Among positive results, RSV (6 cases) and parainfluenza virus 3 (PIV-3) (5 cases) were the most prevalent.

In the BALF pathogen nucleic acid detection results (Figure 3C), among the 95 cases in MPP-segmental/lobar pneumonia group, mixed infections (11 cases), *Streptococcus pneumoniae* (11 cases), and *Methicillin-resistance Staphylococcus aureus* (6 cases) were most prevalent. In MP-ordinary pneumonia group, 48 samples were tested, with *Streptococcus pneumoniae* (9 cases) and mixed infections (5 cases) being the most common.

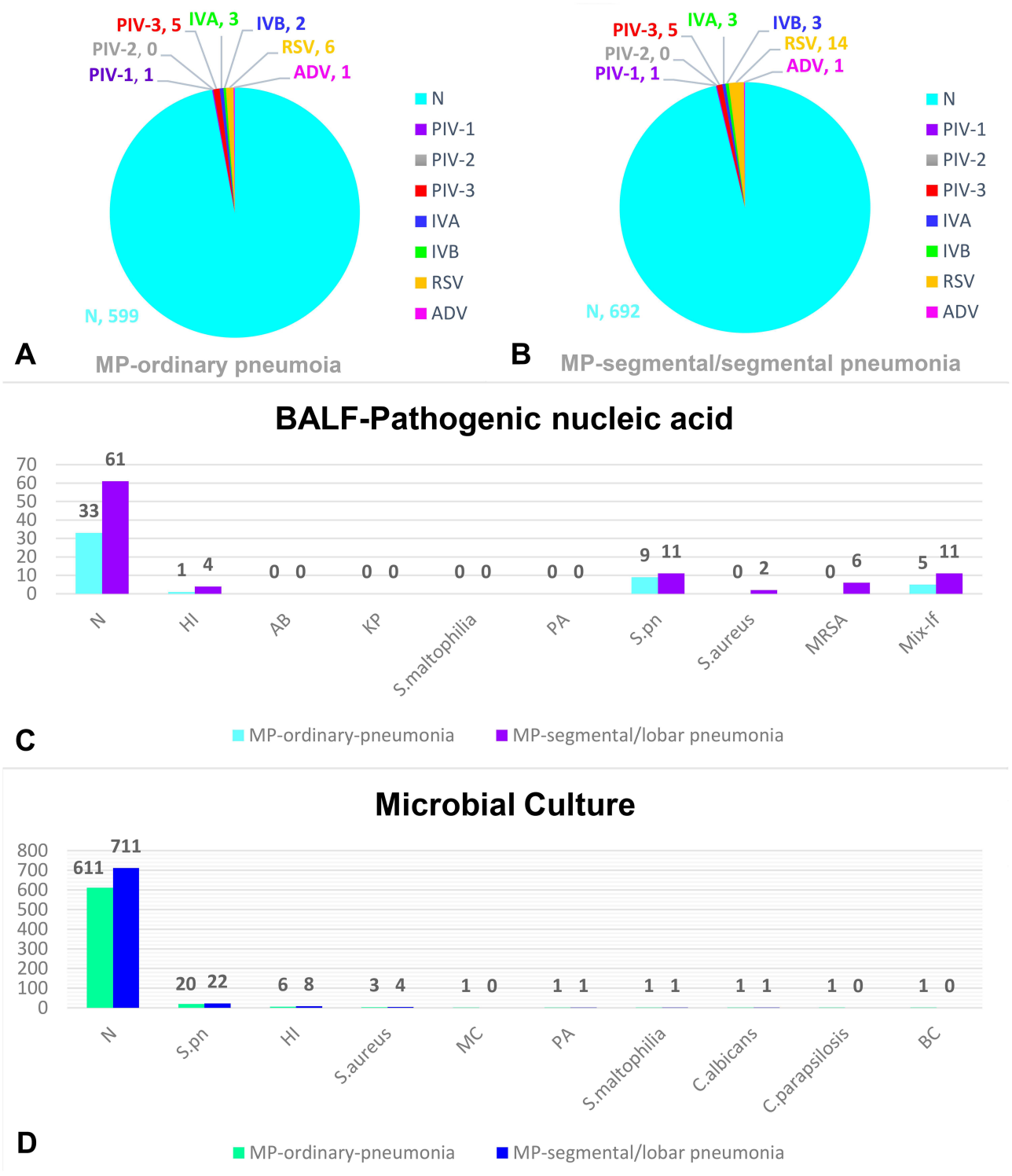

Figure 3 Comparison of respiratory virus nucleic acid detection results (**A** and **B**), BALF pathogen results (**C**), and microbial culture results (**D**) between the MP-ordinary pneumonia group and the MP-segmental/lobar pneumonia group.

Notes: In Figure 3A and B, each color represented a different type of virus; in Figure 3C and D, colors represented different groups.

Abbreviations: MP, *Mycoplasma pneumoniae*; BALF, bronchoalveolar lavage fluid; N, Negative; PIV-1, parainfluenza virus 1; PIV-2, parainfluenza virus 2; PIV-3, parainfluenza virus 3; IVA, influenza A virus; IVB, influenza B virus; RSV, respiratory syncytial virus; ADV, adenovirus; HI, *Haemophilus influenzae*; AB, *Acinetobacter baumannii*; KP, *Klebsiella pneumoniae*; PA, *Pseudomonas aeruginosa*; MC, *Moraxella catarrhalis*; BC, *Burkholderia cepacia*; MRSA, *Methicillin-resistance Staphylococcus aureus*; Mix-if, Mixed infection; *S. maltophilia, Stenotrophomonas maltophilia; S.pn, Streptococcus pneumoniae; S.aureus, Staphylococcus aureus; C.albicans, Candida albicans; C.parapsilosis, Candida parapsilosis*.

https://doi.org/10.2147/JIR.S577148

Microbiological culture testing was performed on 748 specimens from the MP-segmental/lobar pneumonia group, with *Streptococcus pneumoniae* (22 cases), *Haemophilus influenzae* (8 cases), and *Staphylococcus aureus* (4 cases) being the most common pathogens. Among the 646 microbiological culture tests conducted on specimens from the MP-ordinary pneumonia group, *Streptococcus pneumoniae* (20 cases), *Haemophilus influenzae* (6 cases), and *Staphylococcus aureus* (3 cases) were also the most frequently identified pathogens (Figure 3D).

Logistic Regression and Receiver Operating Characteristic (ROC) Curve

Based on the statistical results in Tables 1 and 2, variables with statistical significance were selected. Further selection of variables with predictive value was conducted in conjunction with clinical practice. These were included in univariate logistic regression analysis, revealing the following factors associated with increased risk of MP-segmental/lobar pneumonia: sex (female), pre-admission fever and cough duration, high inflammation markers (CRP, NLR, PLR, PLT, PCT, FIB, D-dimer, LDH, ALT), low levels of TP, PAB, ALB, and A/G ratio.

To adjust for potential confounding factors, a multivariate logistic regression analysis was performed on these 16 variables. The results identified the following independent risk factors for the progression of MPP to segmental/lobar pneumonia: sex-female (OR=0.714, 95% CI: 0.534–0.956, P=0.023), duration of fever prior to admission (OR=1.098, 95% CI: 1.044–1.154, P<0.001), CRP (OR=1.025, 95% CI: 1.010–1.040, P=0.001), D-dimer (OR=1.44, 95% CI: 1.134–1.829, P<0.001), LDH (OR=1.003, 95% CI: 1.000–1.006, P=0.025), PLR (OR=1.004, 95% CI: 1.001–1.007, P =0.020), and PLT (OR=1.002, 95% CI: 1.000–1.003, P=0.022) (Table 3).

To further evaluate the predictive efficacy of these variables for MP-segmental/lobar pneumonia, receiver operating characteristic (ROC) curves were generated. The ROC analysis indicated that days with fever prior to admission, CRP, LDH, D-dimer, and PLR demonstrated good predictive performance for MP-segmental/lobar pneumonia (Figure 4). The following thresholds were associated with an increased risk of MP-segmental/lobar

Table 3 Results of Univariate and Multivariate Logistic Regression Analyses

Variables	Univariate Logistic Regression			Multivariate Logistic Regression		
	OR	95% CI	P	OR	95% CI	P
Sex*	0.631	0.511–0.779	<0.001	0.714	0.534–0.956	0.023
Days with fever prior to admission(days)	1.177	1.136 –1.219	<0.001	1.098	1.044–1.154	<0.001
Days with cough prior to admission(days)	0.966	0.943–0.990	0.005			
CRP	1.031	1.019–1.042	<0.001	1.025	1.010–1.040	0.001
NLR	1.303	1.192–1.425	<0.001			
PLR	1.007	1.005–1.009	<0.001	1.004	1.001–1.007	0.02
PLT	1.001	1.000–1.002	0.003	1.002	1.000–1.003	0.022
PCT	6.293	2.905–13.631	<0.001			
FIB	1.486	1.319 –1.674	<0.001			
D-dimer	2.348	1.879 –2.933	<0.001	1.440	1.134–1.829	<0.001
LDH	1.005	1.004–1.007	<0.001	1.003	1.000–1.006	0.025
TP	0.951	0.931–0.972	<0.001			
PAB	0.993	0.991–0.995	<0.001			
ALB	0.893	0.865–0.923	<0.001			
A/G	0.486	0.330–0.716	<0.001			
ALT(U/L)	1.011	1.004–1.019	0.004			

Notes: sex* indicated that female was at risk compared to male.

Abbreviations: OR, Odds Ratio; CI, Confidence interval; CRP, C-reactive protein; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; PLT, platelet; PCT, procalcitonin; FIB, fibrinogen; LDH, lactate dehydrogenase; TP, total protein; PAB, prealbumin; ALB, albumin; A/G, albumin-to-globulin ratio; ALT, alanine aminotransferase.

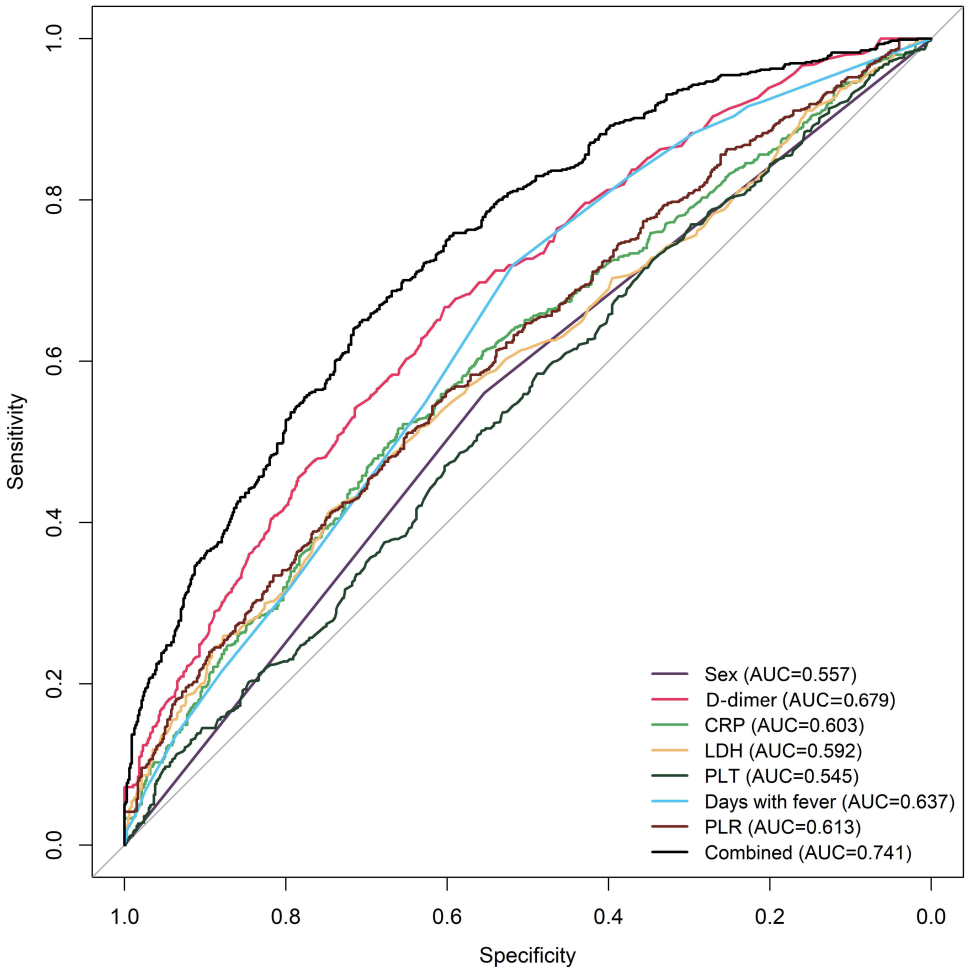

Figure 4 Receiver operating characteristic curves of single variable and combined variables risk factors for predicting MP-segmental/lobar pneumonia.
Notes: Sex indicated that female was at risk compared to male; combined variables referred to the union of these seven variables.
Abbreviations: CRP, C-reactive protein; PLR, platelet-to-lymphocyte ratio; PLT, platelet; LDH, lactate dehydrogenase.

pneumonia: days with fever prior to admission >5.5days, D-dimer >0.539mg/L, CRP >11.33mg/L, LDH >309.5U/L, PLT >340.5×10^9/L, PLR >155.815, and sex-female. Furthermore, the combination of these seven indicators showed improved predictive performance (AUC=0.741, 95% CI: 0.715–0.766, $P < 0.001$) (Table 4).

Table 4 Predictive Efficacy of Seven Variables for MP-Segmental/Lobar Pneumonia

Variable	AUC	Cut-off Value	Youden Index	Sensitivity	Specificity	95% CI	P
D-dimer	0.679	0.539	0.263	0.667	0.603	0.652–0.707	<0.001
Days with fever prior to admission	0.637	5.5	0.238	0.719	0.519	0.608–0.666	<0.001
PLR	0.613	155.815	0.163	0.545	0.618	0.583–0.641	<0.001
CRP	0.603	11.33	0.172	0.522	0.655	0.572–0.631	<0.001
LDH	0.592	309.5	0.16	0.411	0.748	0.562–0.621	<0.001
PLT	0.545	340.5	0.073	0.585	0.489	0.514–0.575	0.004
Sex*	0.557	-	0.115	0.561	0.554	0.531–0.583	<0.001
Combined	0.741	-	0.353	0.648	0.708	0.715–0.766	<0.001

Notes: sex* indicated that female was at risk compared to male.
Abbreviations: MP, *Mycoplasma pneumoniae*; AUC, Area Under Curve; CI, Confidence interval; LDH, lactate dehydrogenase; PLT, platelet; PLR, platelet-to-lymphocyte ratio; CRP, C-reactive protein.

https://doi.org/10.2147/JIR.S577148

Discussion

MP is a cell wall-less organism. Due to the absence of a cell wall, it exhibits inherent resistance to antibiotics that interfere with cell wall synthesis, such as β-lactams.[12] The direct invasion and adhesion of MP to host cells cause cellular damage, while released bacterial components, metabolites, and toxins can induce cytotoxicity, inflammatory responses, oxidative stress, apoptosis, and immunopathological damage in the host.[13,14] Given its distinct seasonal and regional epidemiological patterns, the proportion of MPP among childhood community-acquired pneumonia cases is increasing, resulting in a corresponding rise in MP-associated segmental/lobar pneumonia.[6,8] In this study, by comparing the clinical characteristics of MP-ordinary pneumonia and MP-segmental/lobar pneumonia, we found that MP-segmental/lobar pneumonia exhibited higher levels of inflammatory markers (CRP, PCT, NLR, PLR, etc)., bronchoscopy utilization rates, rates of pulmonary complications, incidence of respiratory failure, length of hospital stay, and hospitalization costs compared to MP-ordinary pneumonia. These results are consistent with our previous findings,[8] confirming that MP-segmental/lobar pneumonia represents a more severe clinical phenotype, posing a significant threat to children's health and imposing a substantial economic burden on families.

Current research predominantly focuses on predicting risk factors for severe complications (eg, necrotizing pneumonia, lung abscess, respiratory failure) and sequelae (eg, atelectasis, obstructive bronchitis) resulting from MP infection.[15–18] An often-overlooked consideration is that the progression of MPP to severe conditions such as necrotizing pneumonia, lung abscess, or atelectasis is necessarily preceded by stages of segmental inflammation and lobar consolidation. Without effective intervention or delayed intervention at this stage at this stage, pulmonary inflammation can progress to persistent consolidation, lung tissue necrosis, and abscess formation.[8] Therefore, identifying the independent risk factors for the progression of MPP to segmental/lobar pneumonia is clinically crucial, as it enables early intervention, prevents the advancement to segmental/lobar lesions, and thereby reduces the incidence of severe complications and sequelae.

In this study, a C-reactive protein level >11.33 mg/L was identified as an independent risk factor for MP-segmental /lobar pneumonia. CRP is a pentameric acute-phase protein, valued for its stability and precision as a non-specific marker of inflammation and tissue injury.[19] Following inflammation or tissue damage, cytokine signaling (eg, IL-6, IL-1, tumor necrosis factor) stimulates hepatocytes to rapidly produce CRP, elevating its concentration up to 100–1000 times the baseline level. In MPP, CRP levels have been demonstrated to correlate significantly with the severity of the inflammatory response.[20,21] Another independent risk factor identified was the duration of fever prior to hospitalization, which is intrinsically linked to the underlying inflammatory process, as fever is a primary clinical manifestation of such inflammatory response. The pathogenesis of MP infection is complex, involving direct invasion and cellular immune responses. MP produces the community-acquired respiratory distress syndrome toxin (CARDS), which can activate the NOD-like receptor thermal protein domain associated protein 3 (NLRP3) inflammasome, subsequently triggering macrophage secretion of IL-1β and IL-18. This process impairs respiratory barrier integrity and causes cellular damage. Furthermore, MP activates T cells, promoting the high expression of cytokines and inflammatory mediators that exacerbate systemic inflammation. Collectively, these inflammatory pathways account for the observed elevations in CRP and the persistence of fever.[22,23]

Lactate dehydrogenase is an oxidoreductase and a clinically established marker of inflammation which catalyzes the interconversion of pyruvate and lactate, as well as that of nicotinamide adenine dinucleotide (NADH) and NAD+. This cytoplasmic enzyme is present in all major organ systems, including the brain, kidneys, liver, heart, and lungs.[24] When organs are damaged by inflammation or other insults, particularly when lung tissue is affected by infection, LDH is released into the bloodstream due to increased cellular membrane permeability or rupture, leading to elevated serum LDH levels.[25,26] Consequently, LDH levels serve as a surrogate marker of tissue inflammatory injury.[27] In this study, a comparison between the two groups revealed that LDH levels were significantly higher in children with MP-segmental /lobar pneumonia than in those with ordinary MPP. Subsequent multivariate logistic regression analysis and ROC curve analysis confirmed that an LDH level >309.5U/L is an independent risk factor for MP-segmental/lobar pneumonia, aligning with previous reports.[16,24]

D-dimer is a specific degradation product formed when fibrin monomers, cross-linked by activator XIII, are hydrolyzed by fibrinolytic enzymes. It is a specific marker of the fibrinolytic system and a useful indicator for monitoring

inflammation and severe infections.[28,29] D-dimer levels in infectious diseases reflect the impact of inflammatory responses on coagulation function. Numerous studies have confirmed a close association between D-dimer levels and the severity of pneumonia.[29–31] In our study, D-dimer levels were significantly elevated in children with MP-segmental /lobar pneumonia compared to those with MP-ordinary pneumonia, indicating a marked inflammatory response and concomitant activation of the fibrinolytic system. Subsequent regression and ROC curve analyses confirmed D-dimer's predictive efficacy for MP-segmental/lobar pneumonia (AUC=0.679, 95% CI=0.652–0.707).

According to literature reports,[32,33] blood hypercoagulability is prevalent in children with MPP. The mechanism may involve direct damage to vascular endothelial cells from excessive and persistent inflammatory cytokines following MP infection. The subsequent exposure of subendothelial collagen activates the coagulation system, leading to a hypercoagulable state and the formation of microthrombi, which impairs pulmonary ventilation, resulting in carbon dioxide retention. Altered pulmonary microcirculation causes the retention of inflammatory mediators and necrotic cells, increased mucus exudation, and the formation of mucus plugs or casts, all of which impede the resolution of pulmonary inflammation, ultimately resulting in segmental and lobar lesions.[34] Platelets possess various surface receptors that regulate their interactions with endothelial cells during inflammation. As key effector cells in the disease-immunity continuum, they play crucial roles in responding to microbial invasion, antigen stimulation, and signal transduction.[35,36] Research has confirmed that in lung injury and systemic inflammation, activated platelets interact with endothelial cells in alveolar capillaries and larger pulmonary vessels, other platelets, and leukocytes in the pulmonary circulation, thereby initiating or amplifying pulmonary dysfunction and inflammatory injury.[37] The Platelet-to-Lymphocyte Ratio is a composite inflammatory marker that reflects the balance between platelet and lymphocyte levels, serving as an indicator of the systemic inflammatory response; it is widely used to assess inflammatory severity in pneumonia.[38,39] In this study, we similarly confirmed that PLR and PLT are independent risk factors for MP-segmental/lobar pneumonia. Although PLT (AUC=0.545) demonstrated relatively low predictive efficacy for MP-segmental/lobar pneumonia in this study, its clinically easy accessibility makes it a viable supplementary indicator for predicting MP-segmental/lobar pneumonia.

Additionally, our study identified sex as a risk factor for MP-segmental/lobar pneumonia, with female patients having a higher risk than males. Recent reports indicate a significant increase in MP-positive cases among 6- to 7-year-old children in China. This trend suggests that the relaxation of non-pharmaceutical interventions (NPIs) after the COVID-19 pandemic in 2023 and the earlier easing of the two-child policy in 2016 may be contributing factors to the severe outbreak of respiratory infections. These reports also note that female patients have a higher risk of MP infection than males.[40] Our finding is consistent with other studies reporting a higher susceptibility to MPP in female patients.[41–43] Female are more likely to test positive for *Mycoplasma pneumoniae* antibodies, possibly due to differences in activity and social behavior; female children may participate more frequently in indoor group activities, which could promote the transmission of MP in a closed environments.[44] However, in this study, gender also demonstrated limited predictive efficacy for MP-segmental/lobar pneumonia (AUC=0.557). Therefore, similar to PLT, it can serve as an auxiliary clinical indicator for the predictive analysis of MP-segmental/lobar pneumonia.

This study is based on a large sample dataset, which provides more robust and generalizable findings than small-sample studies. This study has several limitations. First, its single-center, retrospective design limits the generalizability of the findings, which requires validation through future multicenter, prospective studies. Second, compared with other literature where the predictive efficacy of the indicators reaches more than 0.8, the standalone predictive performance of these seven indicators in this study was not particularly good. Despite this, the combination of these indicators in a predictive model demonstrated good performance (AUC = 0.741, 95% CI: 0.715–0.766), bolstered by the large sample size from which they were derived.

Conclusion

A clinical feature analysis was conducted on data from children hospitalized with MPP at a tertiary pediatric center in Chongqing, China, between January and December 2023. The analysis revealed that MP-segmental/lobar pneumonia is associated with more severe clinical symptoms and significantly elevated inflammatory markers compared to MP-ordinary pneumonia. And we identified five major independent risk factors for the progression of MPP to segmental/lobar pneumonia: D-dimer >0.539 mg/L, CRP >11.33 mg/L, LDH >309.5 U/L, PLR >155.815, and fever duration prior

to admission >5.5 days. Two secondary risk factors were also identified: PLT >340.5×10^9/L and female sex. The predictive model incorporating these seven indicators demonstrated good performance, with an area under the curve (AUC) of 0.741. These indicators may serve as a valuable assessment tool for clinicians to identify children with MPP at high risk of progressing to segmental/lobar pneumonia, thereby facilitating timely intervention and improving outcomes.

Data Sharing Statement

The data sets used and/or analyzed during the current study are available from the corresponding author Gang Geng upon reasonable request.

Ethics Approval and Informed Consent

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki, in compliance with the RECORD (REporting of studies Conducted using Observational Routinely collected health Data) guidelines and was approved by the Institutional Review Board (IRB) of the Children's Hospital of Chongqing Medical University (No. 2024–440). The IRB waived the requirement for informed consent because this study used only de-identified retrospective data from the hospital's electronic medical record and did not impose additional risks or interventions on patients.

Acknowledgments

We are thankful to all the staff of the Clinical Big Data Center of Children's Hospital of Chongqing Medical University for their assistance in retrieving our data.

Author Contributions

Yaozheng Ling: Conceptualization, Data curation, Formal analysis, Methodology, Software, Visualization, Writing – original draft, Writing – review & editing; Yu Zhao: Data curation, Investigation, Resource, Visualization, Writing – review & editing; Chenghao Mei: Methodology, Software, Validation, Visualization, Writing – review & editing; Fang Deng: Methodology, Resources, Supervision, Writing – review & editing; Zhou Fu: Resources, Supervision, Writing – review & editing; Gang Geng: Conceptualization, Project administration, Funding acquisition; Resources Writing – review & editing. All authors made a significant contribution to the work reported; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This research was supported by the Chongqing Natural Science Foundation General Program, CSTB2023NSCQ-MSX0495.

Disclosure

The authors declare no conflicts of interest in this work. The author(s) declare that DeepSeek-V3.2 was used in order to improve language and readability of this paper. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

References

1. Meyer Sauteur PM, Beeton ML, Pereyre S. Mycoplasma pneumoniae: delayed re-emergence after COVID-19 pandemic restrictions. *Lancet Microbe*. 2024;5(2):e100–e101. doi:10.1016/s2666-5247(23)00344-0
2. Yoon IA, Hong KB, Lee HJ, et al. Radiologic findings as a determinant and no effect of macrolide resistance on clinical course of mycoplasma pneumoniae pneumonia. *BMC Infect Dis*. 2017;17:402. doi:10.1186/s12879-017-2500-z
3. Zhang XB, He W, Gui YH, et al. Current mycoplasma pneumoniae epidemic among children in Shanghai: unusual pneumonia caused by usual pathogen. *World J Pediatr*. 2024;20:5–10. doi:10.1007/s12519-023-00793-9
4. Dawood A, Algharib SA, Zhao G, et al. Mycoplasmas as host pantropic and specific pathogens: clinical implications, gene transfer, virulence factors, and future perspectives. *Front Cell Infect Microbiol*. 2022;12(855731). doi:10.3389/fcimb.2022.855731
5. Chen YC, Hsu WY, Chang TH. Macrolide-resistant mycoplasma pneumoniae infections in pediatric community-acquired pneumonia. *Emerg Infect Dis*. 2020;26:1382–1391. doi:10.3201/eid2607.200017

6. Wang Y, Ma L, Li Y, Li Y, Zheng Y, Zhang X. Epidemiology and clinical characteristics of pathogens positive in hospitalized children with segmental/lobar pattern pneumonia. *BMC Infect Dis*. 2020;20:205. doi:10.1186/s12879-020-4938-7
7. Gao J, Yue B, Li H, Chen R, Wu C, Xiao M. Epidemiology and clinical features of segmental/lobar pattern mycoplasma pneumoniae pneumonia: a ten-year retrospective clinical study. *Exp Ther Med*. 2015;10:2337–2344. doi:10.3892/etm.2015.2818
8. Ling Y, Meng F, Li J, et al. Bronchoscopy improves short-term imaging improvement in segmental/lobar pneumonia: a single-center retrospective cohort study. *Respir Med*. 2025;242:108088. doi:10.1016/j.rmed.2025.108088
9. Yamazaki T, Kenri T. Epidemiology of mycoplasma pneumoniae infections in japan and therapeutic strategies for macrolide-resistant M. pneumoniae. *Front Microbiol*. 2016;7:693. doi:10.3389/fmicb.2016.00693
10. Yan C, Xue GH, Zhao HQ, Feng YL, Cui JH, Yuan J. Current status of mycoplasma pneumoniae infection in China. *World J Pediatr*. 2024;20:1–4. doi:10.1007/s12519-023-00783-x
11. Youn YS, Lee KY, Hwang JY, et al. Difference of clinical features in childhood mycoplasma pneumoniae pneumonia. *BMC Pediatr*. 2010;10(48). doi:10.1186/1471-2431-10-48
12. Xie P, Zhang Y, Qin Y, et al. Macrolide resistance in mycoplasma pneumoniae in adult patients. *Front Cell Infect Microbiol*. 2025;15:1496521. doi:10.3389/fcimb.2025.1496521
13. Jiang Z, Li S, Zhu C, Zhou R, Leung PHM. Mycoplasma pneumoniae infections: pathogenesis and vaccine development. *Pathogens*. 2021;11:10. doi:10.3390/pathogens10020119
14. Atkinson TP, Balish MF, Waites KB. Epidemiology, clinical manifestations, pathogenesis and laboratory detection of mycoplasma pneumoniae infections. *FEMS Microbiol Rev*. 2008;32(6):956–973. doi:10.1111/j.1574-6976.2008.00129.x
15. Zhang X, Sun R, Jia W, Li P, Song C. Clinical characteristics of lung consolidation with mycoplasma pneumoniae pneumonia and risk factors for mycoplasma pneumoniae necrotizing pneumonia in children. *Infect Dis Ther*. 2024;13:329–343. doi:10.1007/s40121-023-00914-x
16. Luo Y, Wang Y. Risk prediction model for necrotizing pneumonia in children with mycoplasma pneumoniae pneumonia. *J Inflamm Res*. 2023;16:2079–2087. doi:10.2147/jir.S413161
17. Li X, Zhao L, Cui X, et al. Development and validation of a nomogram for predicting necrotizing pneumonia in children with refractory mycoplasma pneumoniae pneumonia. *Ital J Pediatr*. 2025;51:150. doi:10.1186/s13052-025-02006-7
18. Masters IB, Isles AF, Grimwood K. Necrotizing pneumonia: an emerging problem in children? *Pneumonia*. 2017;9:11. doi:10.1186/s41479-017-0035-0
19. Huang W, Xu X, Zhao W, Cheng Q. Refractory mycoplasma pneumonia in children: a systematic review and meta-analysis of laboratory features and predictors. *J Immunol Res*. 2022;2022:9227838. doi:10.1155/2022/9227838
20. Lee YC, Chang CH, Lee WJ, et al. Altered chemokine profile in refractory mycoplasma pneumoniae pneumonia infected children. *J Microbiol Immunol Infect*. 2021;54:673–679. doi:10.1016/j.jmii.2020.03.030
21. Jian X, Li M, Wang X. The predictive value of MUC5AC levels in the sputum of children with mycoplasma pneumoniae pneumonia treated with fiberbronchoscopy. *Microbiol Spectr*. 2025;13:e0010325. doi:10.1128/spectrum.00103-25
22. Chen P, Huang Z, Chen L, et al. The relationships between LncRNA NNT-AS1, CRP, PCT and their interactions and the refractory mycoplasma pneumoniae pneumonia in children. *Sci Rep*. 2021;11:2059. doi:10.1038/s41598-021-81853-w
23. Li P, Wang W, Zhang X, Pan J, Gong L. Observational retrospective clinical study on clinical features of macrolide-resistant mycoplasma pneumoniae pneumonia in Chinese pediatric cases. *Sci Rep*. 2024;14:5632. doi:10.1038/s41598-024-55311-2
24. Wei D, Zhao Y, Zhang T, Xu Y, Guo W. The role of LDH and ferritin levels as biomarkers for corticosteroid dosage in children with refractory Mycoplasma pneumoniae pneumonia. *Respir Res*. 2024;25:266. doi:10.1186/s12931-024-02892-1
25. Gupta GS. The lactate and the lactate dehydrogenase in inflammatory diseases and major risk factors in COVID-19 patients. *Inflammation*. 2022;45:2091–2123. doi:10.1007/s10753-022-01680-7
26. Drent M, Cobben NA, Henderson RF, Wouters EF, van Dieijen-Visser M. Usefulness of lactate dehydrogenase and its isoenzymes as indicators of lung damage or inflammation. *Eur Respir J*. 1996;9:1736–1742. doi:10.1183/09031936.96.09081736
27. Choi YJ, Jeon JH, Oh JW. Critical combination of initial markers for predicting refractory mycoplasma pneumoniae pneumonia in children: a case control study. *Respir Res*. 2019;20:193. doi:10.1186/s12931-019-1152-5
28. Zhang L, Yan X, Fan Q, et al. D-dimer levels on admission to predict in-hospital mortality in patients with Covid-19. *J Thromb Haemost*. 2020;18:1324–1329. doi:10.1111/jth.14859
29. Huang X, Li D, Liu F, Zhao D, Zhu Y, Tang H. Clinical significance of D-dimer levels in refractory mycoplasma pneumoniae pneumonia. *BMC Infect Dis*. 2021;21. doi:10.1186/s12879-020-05700-5
30. Zheng Y, Hua L, Zhao Q, et al. The level of D-dimer is positively correlated with the severity of mycoplasma pneumoniae pneumonia in children. *Front Cell Infect Microbiol*. 2021;11:687391. doi:10.3389/fcimb.2021.687391
31. Jiang TT, Sun L, Wang TY, et al. The clinical significance of macrolide resistance in pediatric mycoplasma pneumoniae infection during COVID-19 pandemic. *Front Cell Infect Microbiol*. 2023;13:1181402. doi:10.3389/fcimb.2023.1181402
32. Chen J, Wang Y, Hong M, et al. Application of peripheral blood routine parameters in the diagnosis of influenza and Mycoplasma pneumoniae. *Virol J*. 2024;21:162. doi:10.1186/s12985-024-02429-4
33. Liu J, He R, Wu R, et al. Mycoplasma pneumoniae pneumonia associated thrombosis at Beijing Children's hospital. *BMC Infect Dis*. 2020;20:51. doi:10.1186/s12879-020-4774-9
34. Huang JJ, Yang XQ, Zhuo ZQ, Yuan L. Clinical characteristics of plastic bronchitis in children: a retrospective analysis of 43 cases. *Respir Res*. 2022;23:51. doi:10.1186/s12931-022-01975-1
35. Semple JW, Italiano JE Jr, Freedman J. Platelets and the immune continuum. *Nat Rev Immunol*. 2011;11:264–274. doi:10.1038/nri2956
36. Vieira-de-Abreu A, Campbell RA, Weyrich AS, Zimmerman GA. Platelets: versatile effector cells in hemostasis, inflammation, and the immune continuum. *Semin Immunopathol*. 2012;34:5–30. doi:10.1007/s00281-011-0286-4
37. Middleton EA, Weyrich AS, Zimmerman GA. Platelets in pulmonary immune responses and inflammatory lung diseases. *Physiol Rev*. 2016;96:1211–1259. doi:10.1152/physrev.00038.2015
38. Wang RH, Wen WX, Jiang ZP, et al. The clinical value of neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), platelet-to-lymphocyte ratio (PLR) and systemic inflammation response index (SIRI) for predicting the occurrence and severity of pneumonia in patients with intracerebral hemorrhage. *Front Immunol*. 2023;14:1115031. doi:10.3389/fimmu.2023.1115031

39. Wang C, Jiang X, Wu D, Ge M, Deng L. GNRI, PLR and stroke-associated pneumonia: from association to development of a web-based dynamic nomogram. *Clin Interv Aging*. 2023;18:1893–1904. doi:10.2147/cia.S433388
40. Sun Y, Li P, Jin R, et al. Characterizing the epidemiology of mycoplasma pneumoniae infections in China in 2022-2024: a nationwide cross-sectional study of over 1.6 million cases. *Emerg Microbes Infect*. 2025;14:2482703. doi:10.1080/22221751.2025.2482703
41. Nantanda R, Bwanga F, Najjingo I, Ndeezi G, Tumwine JK. Prevalence, risk factors and outcome of mycoplasma pneumoniae infection among children in Uganda: a prospective study. *Paediatr Int Child Health*. 2021;41:188–198. doi:10.1080/20469047.2021.1980698
42. Mao J, Niu Z, Liu M, et al. Comparison of the epidemiological characteristics of mycoplasma pneumoniae infections among children during two epidemics in Wuhan from 2018 to 2024. *BMC Pediatr*. 2025;25:71. doi:10.1186/s12887-025-05435-9
43. Ai L, Liu B, Fang L, Zhou C, Gong F. Comparison of mycoplasma pneumoniae infection in children admitted with community acquired pneumonia before and during the COVID-19 pandemic: a retrospective study at a tertiary hospital of southwest China. *Eur J Clin Microbiol Infect Dis*. 2024;43:1213–1220. doi:10.1007/s10096-024-04824-9
44. Yan L, Dong H, Zhang H, Du Z, Lai M, Zhang L. Positive rates of Mycoplasma pneumoniae antibodies in children before, during and after COVID-19 outbreak: an observational study in Chengdu, China from 2017 to 2024. *Front Microbiol*. 2025;16:1649615. doi:10.3389/fmicb.2025.1649615