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ORIGINAL RESEARCH

Clinical Characteristics and Early Risk Factors for Severe *Mycoplasma Pneumoniae* Pneumonia in Hospitalized Children: A Retrospective Analysis of 417 Cases

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Background: *Mycoplasma pneumoniae* pneumonia (MPP) represents a common form of community-acquired pneumonia in children, and a proportion of cases progress to severe *Mycoplasma pneumoniae* pneumonia (SMPP), which may result in serious complications and life-threatening outcomes. Effective indicators for early recognition of severe disease remain unclear.

Methods: This retrospective analysis included 417 hospitalized children diagnosed with MPP, comprising 210 children with SMPP and 207 children with general *Mycoplasma pneumoniae* pneumonia (GMPP), admitted to The Seventh Medical Centre, Chinese PLA General Hospital, from October 2023 to February 2025. Clinical information, laboratory results, and imaging findings obtained within 24 hours of admission were collected. Differences between groups were compared, and logistic regression analysis along with receiver operating characteristic (ROC) curve analysis was performed to identify risk factors and their predictive value.

Results: Children with SMPP had longer hospitalization and higher frequencies of extrapulmonary complications. Their C-reactive protein (CRP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), and D-dimer (DD) levels were markedly higher. Imaging findings more often demonstrated lobar pneumonia, atelectasis, and pleural effusion. Multivariate logistic regression analysis identified CRP, AST, LDH, and DD as independent risk factors for SMPP. ROC analysis indicated that LDH greater than 286.5 U/L, DD greater than 0.1965 mg/L, and AST greater than 25.05 U/L were strong predictors of SMPP.

Conclusion: The clinical and laboratory profiles of children with SMPP differed significantly from those with GMPP. CRP, AST, LDH, and DD function as important indicators for early identification and assessment of disease severity, supporting clinical decision making. Early monitoring of these parameters enables timely recognition of high-risk patients and may improve clinical management and prognosis.

Keywords: clinical characteristics, inflammatory factors, *Mycoplasma pneumoniae* pneumonia, predictive indicators, risk factors

Introduction

Mycoplasma pneumoniae (MP) is a major pathogen responsible for community-acquired pneumonia (CAP) in children, particularly among school-age children and adolescents, with recent reports indicating a shift toward younger age groups.[1–3] MP infection primarily affects the respiratory tract but may also involve multiple organ systems, presenting with diverse extrapulmonary manifestations that affect the skin, nervous system, blood, cardiovascular system, genitourinary system, and musculoskeletal system. Severe cases may develop complications such as pseudomembranous necrotizing laryngotracheobronchitis and myelin oligodendrocyte glycoprotein antibody-associated meningoencephalitis.[4–7] Although most patients

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with *Mycoplasma pneumoniae* pneumonia (MPP) experience significant recovery, some progress to severe *Mycoplasma pneumoniae* pneumonia (SMPP), which is characterized by worsening clinical symptoms, progressive imaging abnormalities, and increased risks of mortality and complications.[8]

Most previous studies focused on isolated biomarkers or measurements obtained later in the disease course, limiting their applicability for early clinical decision-making at hospital admission. Lactate dehydrogenase (LDH) reflects cellular injury and immune-mediated tissue damage, whereas D-dimer indicates activation of coagulation pathways and inflammation-induced hypercoagulability-both of which have been implicated in severe MPP but remain insufficiently validated as early predictors. Early identification of high-risk patients is crucial for timely escalation of treatment, including corticosteroids or immunomodulatory therapy, to prevent irreversible pulmonary damage. Therefore, to support early identification and clinical management of SMPP, we systematically collected and compared general conditions, clinical characteristics, laboratory parameters, and chest imaging findings of patients with SMPP and general *Mycoplasma pneumoniae* pneumonia (GMPP). Differences between the groups were evaluated, and clinical characteristics, risk factors, and early warning indicators associated with SMPP were examined. These findings aim to provide evidence to support early diagnosis and timely intervention for SMPP, thereby reducing the occurrence of complications and long-term sequelae and improving patient prognosis.

Materials and Methods

Study Participants

This retrospective analysis included 417 children hospitalized with MPP in The Seventh Medical Centre, Chinese PLA General Hospital, from October 2023 to February 2025. Among these patients, 210 were classified as having SMPP and 207 were classified as having GMPP. The inclusion criteria were: (1) age between 3 months and 12 years; (2) diagnosis consistent with the criteria for MPP. The exclusion criteria were: (1) failure to meet inclusion criteria or incomplete clinical data; (2) patients diagnosed with congenital heart disease, pulmonary tuberculosis, bronchial foreign bodies, bronchiectasis, asthma, or metabolic disorders; and (3) children whose condition had already entered the recovery phase at the time of admission.

At our institution, cytokine testing and routine chest CT examinations were incorporated into the standardized clinical pathway for all children hospitalized with MPP.

Methods

Diagnostic Criteria for MPP

The diagnosis of MPP followed the *Guidelines for Diagnosis and Treatment of Mycoplasma pneumoniae in Children (2023 Edition)*, which require:[9] (1) acute fever, cough, and other symptoms of respiratory tract infection; (2) lung auscultation demonstrating medium to small moist rales or imaging studies demonstrating pulmonary lesions; and (3) laboratory evidence of infection with positive MP-DNA or RNA, or positive serum MP-IgM with an antibody titer of 1:160 or greater using the particle agglutination method, or a four-fold or greater increase in paired serum antibody titers during the disease course compared with earlier levels. MP-DNA or MP-RNA was tested using throat swab samples collected at admission with a *Mycoplasma pneumoniae* nucleic acid detection kit (Fosun Diagnostics, Shanghai, China). For children with positive nucleic acid results but a disease course longer than 5 days, serological confirmation was additionally performed using the particle agglutination method in the laboratory of our hospital.

Diagnostic Criteria for SMPP

According to the 2023 edition of the *Guidelines for the Diagnosis and Treatment of Mycoplasma pneumoniae in Children*, SMPP is diagnosed when any of the following criteria are met: persistent high fever of 39 °C or greater lasting 5 days or more, or fever lasting 7 days or more without reduction in peak temperature; wheezing, tachypnea, dyspnea, chest pain, or hemoptysis indicating severe pulmonary lesions or complications; extrapulmonary organ complications not meeting critical criteria; oxygen saturation of 93% or lower at rest while breathing room air; imaging studies demonstrating high-density consolidation involving more than two-thirds of a single pulmonary lobe or multiple lobes, or unilateral or bilateral diffuse bronchiolitis with bronchitis and atelectasis; or lesion progression exceeding 50% within 24 to 48 hours.[9]

Data Collection

Clinical information was collected for both groups, including: (1) general conditions and clinical characteristics such as age, gender, length of hospital stay, season of onset, drug resistance, and clinical manifestations; (2) laboratory examinations, including tumor necrosis factor α (TNF-α), interferon γ (IFN-γ), interleukin 2 (IL-2), interleukin 4 (IL-4), interleukin 5 (IL-5), interleukin 6 (IL-6), interleukin 17 (IL-17), immunoglobulin G (IgG), immunoglobulin M (IgM), immunoglobulin A (IgA), immunoglobulin E (IgE), white blood cell count (WBC), neutrophil count (NEU), monocyte count (MON), eosinophil count (EC), platelet count (PLT), C-reactive protein (CRP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), LDH, creatine kinase-MB isoenzyme (CK-MB), cardiac troponin I (TnI), fibrinogen (FIB), activated partial thromboplastin time (APTT), and D-dimer (DD); and (3) imaging findings from chest CT examinations (performed either at presentation in the emergency department or on the day of admission)., including infectious lesions, lobar consolidation, and the extent of pleural adhesion or thickening.

Plasma samples were prospectively collected at the time of hospital admission.[10] Approximately 10 mL of peripheral blood was drawn into EDTA vacuum tubes (BD Vacutainer®) and processed within 4 hours of collection. The samples were centrifuged at 1,500 rpm (800 × g) for 10 minutes to separate plasma. The clarified plasma was then aliquoted and stored at −80°C until analysis. Cytokine levels were measured in the hospital's clinical laboratory using the EasyMagPlex Human Cytokine 12 Plex Kit (Wellgrow Biotechnology Co., Ltd., Shenzhen, China).

Serum levels of IgG, IgM, IgA, ALT, AST, LDH, and CRP were measured using an automated biochemical analyzer (iMagic-M7; Mindray, Shenzhen, China) with corresponding commercial reagent kits.[11] PLT, WBC, NEU, MON, and EC were determined using an automated hematology analyzer (XFA6000; Perlong Medical Equipment Co., China).[12] FIB and APTT were assessed using an automated coagulation analyzer (Sysmex® CS-2100i).[13] IgE, CK-MB, TnI, and DD were measured using a fully automated chemiluminescence immunoassay analyzer (CL-6000i; Mindray, Shenzhen, China).[14]

All Assays Were Performed in the Laboratory of Our Hospital

Statistical Analysis

Statistical analysis was conducted using SPSS 27.0 statistical software. Continuous variables were expressed as mean ± standard deviation ($\bar{x} \pm s$), and comparisons between groups were performed using the two-sample rank sum test. Categorical variables were reported as case numbers and percentages, with comparisons between groups conducted using the chi-square test or Fisher's exact test. Variables that demonstrated statistical significance in univariate analysis were included in multivariate binary logistic regression analysis using forward likelihood ratio stepwise regression analysis. Receiver operating characteristic (ROC) curves were generated, and the predictive performance of each independent risk factor was assessed through the calculation of the area under the ROC curve (AUC). A p value of less than 0.05 was considered statistically significant.

Results

Comparison of Basic Information and Clinical Characteristics Between SMPP and GMPP Groups

A total of 417 children were included, comprising 210 children in the SMPP group (103 males, 49.0%; 107 females, 51.0%) with a median age of onset of 6.17 years, and 207 children in the GMPP group (108 males, 52.2%; 99 females, 47.8%) with a median age of onset of 7.25 years. The length of hospital stay in the SMPP group was significantly longer than that in the GMPP group (11.57 days vs 8.66 days, $p < 0.001$). Regarding clinical manifestations, the incidence of fever (95.7% vs 87.2%, $p = 0.03$), cough (100.0% vs 95.2%, $p = 0.01$), and extrapulmonary complications (55.7% vs 0.1%, $p < 0.001$) was significantly higher in the SMPP group. No significant differences were observed between the groups in gender distribution, age composition, peak onset period, wheezing, or drug resistance (Table 1). The distribution of co-infecting pathogens in the SMPP and GMPP groups is shown in Figure 1.

Table 1 Basic Information and Clinical Characteristics of Children with SMPP and GMPP

Variable	SMPP (n=210)	GMPP (n=207)	χ2/t/Z	*P*
Sex, n (%)				
Male/Female	103/107	108/99	0.64	0.523
Age, years (median)	6.17±3.41	7.25±3.31	−3.27	0.979
Length of hospital stay, days	11.57±5.38	8.66±2.82	6.90	<0.001
Season of onset, n (%)				
Spring / summer	99(47.1%)	98(47.3%)	0.093	0.76
Autumn / winter	111(52.9%)	109(52.7%)		
Drug resistance, n (%)	193(88.9%)	190(91.8%)	0.002	0.9665
Clinical manifestations, n (%)				
Fever	201(95.7%)	182(87.2%)	8.92	0.03
Wheezing	24(11.4%)	16(7.3%)	1.65	0.2
Cough	210(100%)	197(95.2%)	10.40	0.01
Co-Infection	79 (37.6%)	71 (34.3%)	0.504	0.635
Extrapulmonary complications	117(55.7%)	2(0.1%)	153.22	<0.001

Comparison of Serum Inflammatory Cytokines and Immunoglobulin Levels Between SMPP and GMPP Groups

Compared with the GMPP group, serum levels of IFN-γ (18.87 pg/mL vs 13.26 pg/mL, $p = 0.005$), IL-4 (3.70 pg/mL vs 2.61 pg/mL, $p = 0.045$), IL-5 (2.24 pg/mL vs 1.24 pg/mL, $p = 0.015$), IL-6 (37.43 pg/mL vs 14.58 pg/mL, $p = 0.007$), and

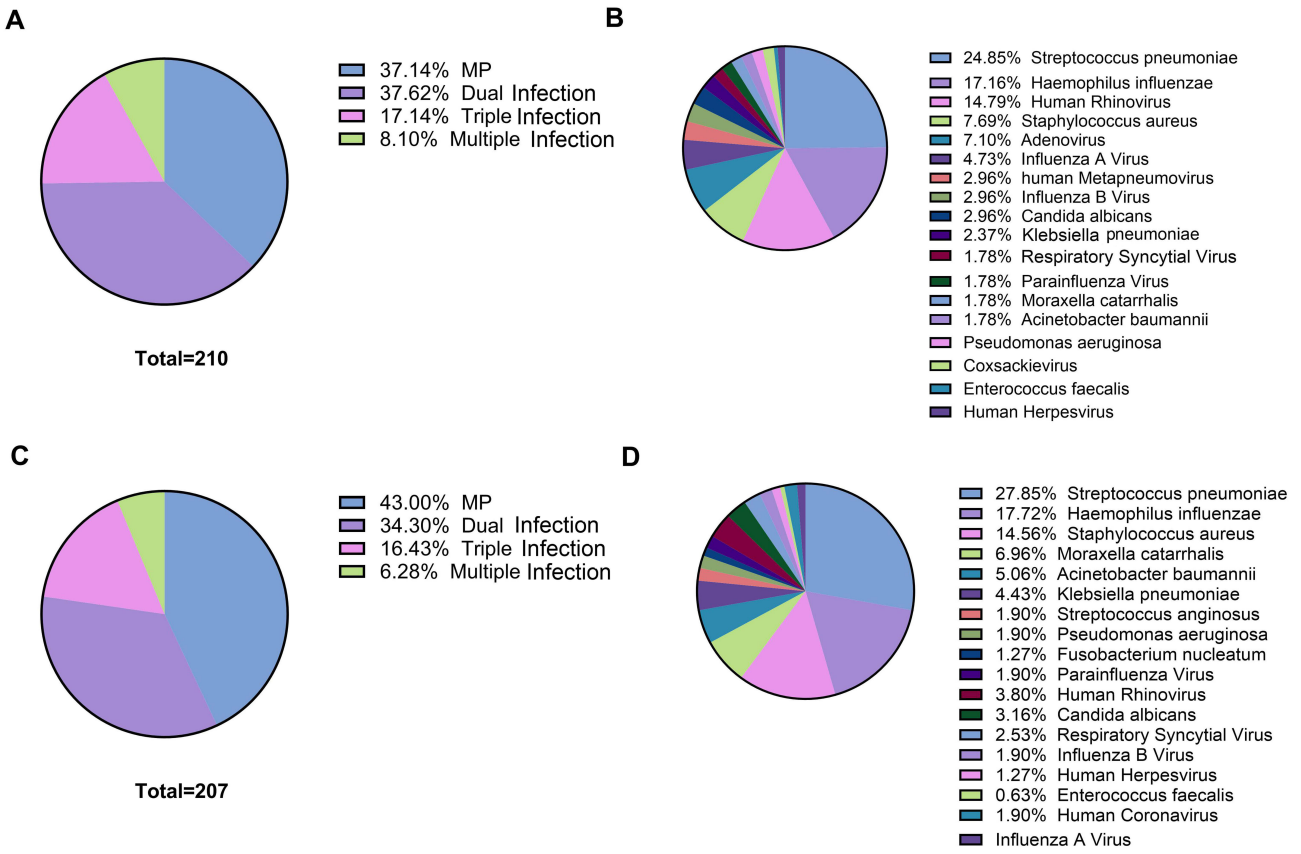

Figure 1 Distribution of co-infecting pathogens in the SMPP and GMPP groups. (**A**) Pathogen Distribution in the SMPP Group; (**B**) Distribution of Co-infecting Pathogens in the SMPP Group; (**C**) Pathogen Distribution in the GMPP Group; (**D**) Distribution of Co-infecting Pathogens in the GMPP Group.

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IgM (1.76 g/L vs 1.44 g/L, $p < 0.001$) were significantly higher in the SMPP group. Serum levels of TNF-α (2.99 pg/mL vs 4.20 pg/mL, $p < 0.001$) and IgE (194.4 g/L vs 222.6 g/L, $p = 0.032$) were significantly lower in the SMPP group. No significant differences were identified between the groups in IL-2, IL-17, IgG, or IgA levels (Table 2).

Comparison of Laboratory Test results Between SMPP and GMPP Groups

Compared with the GMPP group, the SMPP group demonstrated significantly higher levels or activities of CRP (15.38 mg/L vs 10.71 mg/L, $p = 0.026$), AST (37.48 U/L vs 22.22 U/L, $p = 0.002$), ALT (25.14 U/L vs 13.16 U/L, $p < 0.001$), LDH (376.49 U/L vs 246.27 U/L, $p < 0.001$), CK-MB (23.21 U/L vs 17.79 U/L, $p < 0.001$), TnI (2.00 μg/L vs 1.22 μg/L, $p < 0.001$), APTT (33.02 s vs 33.68 s, $p = 0.037$), and DD (0.79 mg/L vs 0.15 mg/L, $p < 0.001$). No significant differences were observed between the groups in WBC, NEU, MON, EC, PLT, or FIB levels (Table 3).

Comparison of Imaging Analysis Results Between SMPP and GMPP Groups

Abnormal imaging findings were more pronounced in the SMPP group. The incidence of infectious lesions ($p = 0.025$), lobar pneumonia ($p = 0.01$), atelectasis (7.1% vs 1.4%, $p = 0.004$), pleural effusion (23.3% vs 4.8%, $p < 0.001$), and pericardial effusion (2.4% vs 0%, $p = 0.025$) was significantly higher in the SMPP group compared with the GMPP group (Table 4).

Table 2 Serum Inflammatory Cytokine and Immunoglobulin Levels in Children with SMPP and GMP

Variable	SMPP (n=210)	GMPP (n=207)	χ2/t/Z	P
IL-2 (pg/mL)	3.69±4.52	4.20±8.25	−0.615	0.098
TNF-α (pg/mL)	2.99±2.31	4.20±8.25	2.84	<0.001
IFN-γ (pg/mL)	18.87±18.51	13.26±11.27	2.84	0.005
IL-4 (pg/mL)	3.70±5.69	2.61±2.83	1.87	0.045
IL-5 (pg/mL)	2.24±4.39	1.24±1.46	2.37	0.015
IL-6 (pg/mL)	37.43±25.41	14.58±8.45	1.78	0.007
IL-17 (pg/mL)	4.09±8.73	3.03±6.62	1.06	0.400
IgG (g/L)	9.29±2.56	9.86±2.42	−2.24	0.756
IgM (g/L)	1.76±0.86	1.44±0.59	4.25	<0.001
IgA (g/L)	1.33±0.72	1.55±0.77	−2.93	0.625
IgE (g/L)	194.40±61.58	222.6±73.28	−0.86	0.032

Table 3 Laboratory Test Results in Children with SMPP and GMPP

Variable	SMPP (n=210)	GMPP (n=207)	χ2/t/Z	P
WBC (10^9/L)	8.07±3.10	7.35±2.82	2.45	0.24
NEU (10^9/L)	59.09±15.72	56.63±14.23	1.67	0.054
MON (10^9/L)	6.83±2.39	7.54±2.62	−2.88	0.44
EC (10^9/L)	1.23±1.76	1.54±2.18	−1.62	0.24
PLT(10^9/L)	309.68±61.41	294.16±51.26	1.64	0.20
CRP (mg/L)	15.38±9.85	10.71±8.08	2.15	0.026
AST (U/L)	37.48±17.17	22.42±11.62	2.76	0.002
ALT (U/L)	25.14±11.18	13.16±7.40	4.12	<0.001
LDH (U/L)	376.49±67.89	246.27±38.66	8.84	<0.001
CK-MB (U/L)	23.21±18.59	17.79±5.81	3.99	<0.001
TnL (ug/L)	2.00±1.55	1.20±0.67	3.10	<0.001
FIB (g/L)	3.85±1.18	3.27±0.60	1.16	0.198
APTT (s)	33.02±4.75	33.68±3.64	−1.39	0.037
DD (mg/L)	0.79±0.51	0.15±0.08	4.62	<0.001

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Table 4 Chest Imaging Findings in Children with SMPP and GMPP

Imaging Findings, n (%)	SMPP (n=210)	GMPP (n=207)	χ2/t/Z	P
Infectious lesions			9.08	0.028
1 pulmonary lobe	70(33.3%)	92(44.4%)		
≥2 pulmonary lobes	74(35.2%)	52(25.1%)		
≥3 pulmonary lobes	46(21.9%)	38(18.4%)		
Lobar consolidation			11.24	0.01
1 pulmonary lobe	94(44.8%)	67(32.4%)		
≥2 pulmonary lobes	14(6.7%)	9(4.3%)		
≥3 pulmonary lobes	4(1.9%)	2(0.9%)		
Pleural adhesion/thickening	4(1.9%)	2(0.9%)	1.59	0.452
Atelectasis	15(7.1%)	3(1.4%)	8.26	0.004
Pleural effusion	49(23.3%)	1(4.8%)	52.19	<0.001
Pericardial effusion	5(2.4%)	0(0%)	5.01	0.025

Correlation Analysis Between Different Clinical Indicators and SMPP Occurrence

Univariate analysis of clinical data from all children with MPP indicated that length of hospital stay, TNF-α, IFN-γ, IL-4, IL-5, IL-6, IgM, IgE, CRP, AST, ALT, LDH, CK-MB, TnI, APTT, and DD were significantly associated with SMPP ($p < 0.05$). Multivariate binary logistic regression analysis including these statistically significant variables identified length of hospital stay ($p < 0.001$), CRP ($p = 0.026$), AST ($p = 0.011$), ALT ($p = 0.004$), LDH ($p < 0.001$), and DD ($p = 0.002$) as independent risk factors for SMPP (Table 5).

Predictive Value Analysis of Independent Correlative Factors for SMPP

ROC curve analysis evaluated the predictive performance of each independent risk factor for SMPP. LDH demonstrated the strongest predictive capability (AUC = 0.860, sensitivity = 77.4%, specificity = 90.1%), with levels exceeding 286.5 U/L effectively predicting SMPP. DD also demonstrated strong predictive value (AUC = 0.815, sensitivity = 73.8%, specificity = 77.6%), with levels above 0.1965 mg/L demonstrating high diagnostic utility. AST provided moderate predictive value (AUC = 0.675, sensitivity = 56.0%, specificity = 74.3%). CRP (AUC = 0.596, sensitivity = 53.0%) and ALT (AUC = 0.611, sensitivity = 57.1%) demonstrated weaker predictive performance (Table 6, Figure 2).

Table 5 Multivariate Logistic Regression Analysis of Independent Risk Factors for SMPP

Variable	β value	Standard error	Wald χ^2 value	p value	Exp (B)	95% CI
Length of hospital stay	−0.203	0.033	38.473	<0.001	0.817	0.766–0.871
TNF-α	0.013	0.029	0.220	0.639	1.014	0.958–1.072
IFN-γ	−0.031	0.250	0.015	0.902	0.970	0.594–1.584
IL-4	0.541	0.339	2.541	0.111	1.718	0.883–3.340
IL-5	−0.144	0.362	0.157	0.691	0.866	0.426–1.760
IL-6	−0.050	0.059	0.730	0.393	0.951	0.847–1.067
IgM	−0.045	0.625	0.005	0.942	0.956	0.281–3.252
IgE	−0.001	0.001	1.423	0.233	0.999	0.997–1.001
CRP	−0.057	0.026	4.988	0.026	0.944	0.898–0.993
AST	0.044	0.017	6.468	0.011	1.045	1.010–1.082
ALT	−0.062	0.021	8.429	0.004	0.940	0.902–0.980
LDH	−0.042	0.010	18.941	<0.001	0.959	0.941–0.977
CK-MB	−0.030	0.052	0.329	0.567	0.971	0.877–1.075
TnL	−0.447	0.359	1.546	0.214	0.640	0.316–1.294
APTT	0.040	0.078	0.258	0.612	1.040	0.893–1.212
DD	−7.942	3.184	6.222	0.013	0.000	0.00–0.182

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Table 6 Predictive Value of Independent Risk Factors for SMPP Based on ROC Curve Analysis

Variable	Optimal cutoff value	AUC	Standard error	*p*-value	95% *CI*	Sensitivity (%)	Specificity (%)
CRP	7.96	0.596	0.032	0.003	0.534–0.658	53.0	61.2
AST	25.05	0.675	0.030	0.000	0.616–0.733	56.0	74.3
ALT	13.25	0.611	0.031	0.001	0.549–0.672	57.1	65.1
LDH	286.5	0.860	0.023	<0.001	0.815–0.905	77.4	90.1
DD	0.1965	0.815	0.024	<0.001	0.769–0.862	73.8	77.6

Discussion

MPP is one of the most common forms of community-acquired pneumonia in children and demonstrates clear seasonality and clustering patterns. Due to the immature immune function in children, particularly in the preschool age group, susceptibility to infection is increased. Recent research has indicated that the age of onset of MPP has gradually shifted toward younger children. Clinically, fever and cough represent the predominant manifestations, and early targeted antibiotic administration typically results in substantial symptom improvement. However, some children experience slow recovery or prolonged illness, which may lead to varying levels of intrapulmonary lesions and extrapulmonary involvement. Reports have indicated that approximately 6% to 23% of affected children may develop pulmonary sequelae. As the inflammatory response intensifies, some patients progress to SMPP, which is characterized by persistent high fever, dyspnea, and multi-organ functional impairment. Early recognition of disease severity and accurate prognostic assessment remain essential for optimizing therapeutic strategies and improving clinical outcomes.

The findings of this analysis indicated notable differences between SMPP and GMPP in clinical manifestations and laboratory characteristics. The longer hospitalization duration observed in children with SMPP may reflect increased disease complexity and challenges in clinical management. Fever, cough, and extrapulmonary complications occurred more frequently in the SMPP group, indicating that severe disease may be associated with heightened immune responses or more extensive infection. Extrapulmonary involvement may be related to systemic inflammatory responses triggered when Mycoplasma enters the bloodstream, although the underlying mechanisms require further clarification.[15]

Interleukins (ILs) are cytokines secreted by immune cells that regulate immune responses and are closely associated with the development and progression of MPP.[16–18] IL-4 promotes B-cell proliferation, differentiation, and activation, stimulates airway epithelial cells to secrete mucus, and contributes to airway smooth muscle activation, all of which may provoke airway

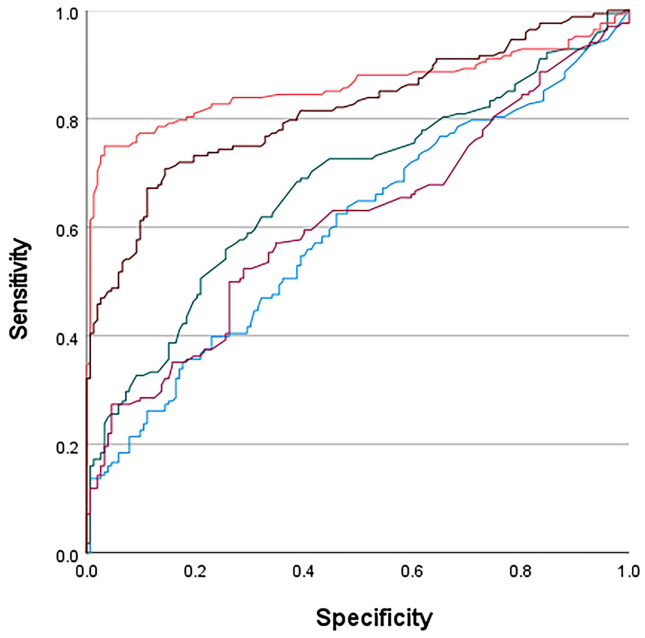

Figure 2 ROC curve analysis of each independent risk factors for SMPP. Blue: CRP; Green: AST; Purple: ALT; Red: LDH; Brown: DD.

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hyperresponsiveness. IL-4 is frequently used as an indicator of the risk of refractory MPP in children.[19] IL-5 plays a crucial role in the differentiation, recruitment, and activation of eosinophils, contributing to airway inflammation and remodeling and promoting Mycoplasma-associated or Mycoplasma-aggravated asthma.[20] IL-6 is involved in anti-infection responses through activation of immune cells, but persistently elevated IL-6 levels may result in tissue damage and immunopathological responses.[21] TNF-α facilitates the release of chemokines by macrophages and neutrophils, and the combined effect of these pathways results in rapid neutrophil migration and accumulation at inflammatory sites, leading to aggravated local inflammation and tissue injury.[22] Consistent with domestic and international research findings, the present results demonstrated that levels of IL-4, IL-5, IL-6, and IFN-γ were significantly higher in children with SMPP compared with those with GMPP.[23–25]

A recent retrospective, cross-sectional study involving CAP children in the pediatric intensive care unit has found that mortality was significantly associated with infectious agent type, respiratory support type, length of stay, lactate, and lactate/albumin ratio.[26] Their results suggest that CRP/mean platelet volume may be useful in assessing mortality, clinical severity, and respiratory support type, and the lactate/albumin ratio may be a more valuable predictor of mortality than lactate alone. Our study also incorporated laboratory indicators reflecting infection and inflammation (WBC, NEU, MON, EC, CRP, serum amyloid A [SAA], erythrocyte sedimentation rate [ESR], LDH), liver function (ALT, AST), myocardial involvement (CK-MB, TnI), coagulation function (FIB, APTT, DD), and PLT to provide a comprehensive assessment of inflammatory status, organ injury, and multi-system functional changes. Compared with the GMPP group, the SMPP group demonstrated significantly higher levels or activities of CRP, LDH, AST, ALT, CK-MB, TnI, APTT, and DD. CRP and LDH represent important inflammatory biomarkers, and their levels are closely related to the severity of inflammation.

These markers are considered sensitive for the early detection of MP infection in children.[27] Children with SMPP were also more likely to develop pulmonary and extrapulmonary complications. Although the mechanisms underlying MP-related extrapulmonary complications are not fully understood, existing evidence indicates that both direct pathogen-induced injury and excessive immune activation contribute to their development.

Elevated ALT and AST levels indicated that infection may affect hepatic function, while increased CK-MB and TnI levels implied possible myocardial or skeletal muscle involvement.[28,29] DD levels were also significantly higher in severe cases, consistent with prior evidence linking MPP to increased risks of thrombosis or embolism.[30] The markedly elevated DD levels observed in SMPP align with previous research reporting enhanced thrombosis risk in children with MPP.[31] However, no clinically confirmed thrombotic events, such as deep vein thrombosis or pulmonary embolism, were documented in this cohort, and none of the children had known predispositions to thrombophilia. The elevated DD levels may therefore reflect a localized pulmonary hypercoagulable state induced by Mycoplasma infection rather than secondary consequences of extrapulmonary thrombosis.

MP can involve the entire respiratory tract, including the bronchial, interstitial, and alveolar regions. Accordingly, MPP may present with diverse imaging manifestations. Although the distribution of lesions did not differ significantly between SMPP and GMPP, the infection in children with SMPP was more extensive, with significantly higher rates of lobar pneumonia, atelectasis, and pleural effusion. A prior analysis of 393 hospitalized children with MPP reported that pulmonary lobe or segmental consolidation was the most common imaging finding (37%).[32] These observations indicate that MP infection can lead to substantial pulmonary tissue injury, underscoring the importance of early assessment to prevent adverse outcomes.

Early prediction of SMPP in the initial stages of MP infection has substantial clinical relevance. Multiple serological indicators were evaluated to determine their predictive utility. Logistic regression analysis identified length of hospital stay, CRP, AST, ALT, LDH, and DD as independent risk factors for SMPP. ROC curve analysis further demonstrated that LDH levels of 286.5 U/L or greater and DD levels of 0.1965 mg/L or greater served as reliable predictors of SMPP. Another study reported that LDH levels of 360 U/L or greater and DD levels of 0.6 mg/L or greater were meaningful predictors of SMPP. Collectively, these findings indicate that LDH and DD hold promise as key serological markers for evaluating disease severity and predicting progression to severe disease. Larger multicenter studies will be essential for validating their clinical reliability and establishing optimal cutoff values.

Conclusions

In conclusion, children with SMPP exhibited higher frequencies of persistent high fever, extrapulmonary manifestations, notable pulmonary imaging abnormalities, coagulation dysfunction, and marked elevations in inflammation and tissue-

injury markers including CRP, LDH, AST, ALT, CK-MB, TnI, APTT, and DD compared with children with non-severe MPP. CRP, AST, ALT, LDH, and DD demonstrated meaningful value in predicting SMPP. When chest imaging at admission reveals substantial pulmonary consolidation and laboratory findings display markedly elevated levels of these biomarkers, clinicians should recognize the heightened risk of progression to SMPP. In such circumstances, prompt anti-Mycoplasma management alongside early use of glucocorticoids or immunomodulators may help reduce inflammation and prevent further disease progression. The indicators used in this analysis are easily obtainable in routine clinical practice, supporting their integration into early diagnostic and management strategies.

Abbreviations

MPP, *Mycoplasma pneumoniae* pneumonia; SMMP, severe *Mycoplasma pneumoniae* pneumonia; GMMP, gernal *Mycoplasma pneumoniae* pneumonia; CAP, community-acquired pneumonia; TNF-α, Tumor Necrosis Factor-α; IFN-γ, Interferon-γ; IL-2, Interleukin-2; IL-4, Interleukin-4; IL-5, Interleukin-5; IL-6, Interleukin-6; IL-17, Interleukin-17; IgG, Immunoglobulin G; IgM, Immunoglobulin M; IgA, Immunoglobulin A; IgE, Immunoglobulin E; WBC, White Blood Cell count; NEU, Neutrophil count; MON, Monocyte count; EC, Eosinophil count; PLT, Platelet count; CRP, C-reactive Protein; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; LDH, Lactate Dehydrogenase; CK-MB, Creatine Kinase-MB isoenzyme; TnI, Cardiac Troponin I; FIB, Fibrinogen; APTT, Activated Partial Thromboplastin Time; DD, D-dimer; ROC, Receiver Operating Characteristic curve; EOS, Eosinophil count.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author, Dr. Shunying Zhao, upon reasonable request.

Ethics Approval and Consent to Participate

The retrospective study was approved by ethics committee of The Seventh Medical Centre, Chinese PLA General Hospital (No. S2025-138-01). This study was conducted in accordance with the declaration of Helsinki. This was a retrospective study involving only the analysis of existing clinical data, with no direct patient contact or intervention; therefore, the ethics committee waived the requirement for informed consent.

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Disclosure

The authors declare that they have no competing interests.

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