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Clinical Characteristics and Predictive Factors Analysis of Mycoplasma Pneumoniae Pneumonia Complicated with Pleural Effusion in Children

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ABSTRACT

Mycoplasma pneumoniae pneumonia (MPP) is a prevalent cause of respiratory infections in children, sometimes leading to pleural effusion (PE). This study aimed to identify risk factors and clinical features associated with PE in pediatric MPP patients.

We conducted a retrospective case-control study involving 412 children with MPP and 82 with MPP+PE at the Third Affiliated Hospital of Wenzhou Medical University from January 2021 to January 2024. Demographic, clinical, and laboratory data were analyzed using multivariate logistic regression and receiver operating characteristic (ROC) curves.

Significant findings included a higher incidence of immunocompromised states in the MPP+PE group (18.29% vs. 8.98%). At admission, children with MPP+PE exhibited higher respiratory rates (29.94 vs. 29.16 breaths/min), lower oxygen saturation (82.33% vs. 83.14%), longer fever duration (5.75 vs. 4.83 days), elevated white blood cell counts (WBC) (11.64×10^9/L vs. 10.12×10^9/L), and increased erythrocyte sedimentation rates (ESR) (20.66 vs. 19.49 mm/h). Patients with PE also experienced longer antibiotic treatment (9.14±4.91 vs. 7.46±3.29 days) and extended hospital stays (13.58 ± 4.18 vs. 12.37 ± 3.52 days). Multivariate analysis identified several significant predictors of PE, and a joint prediction model achieved an area under the curve (AUC) of 0.842, sensitivity of 0.796, and specificity of 0.793.

These findings suggest that specific clinical and laboratory factors can help identify children at higher risk for PE, facilitating timely interventions.

Keywords: Diagnostic markers; Mycoplasma pneumonia; Pediatric patients; Pleural effusion; Retrospective case-control study

INTRODUCTION

Mycoplasma pneumoniae (MP) is a well-recognized cause of community-acquired pneumonia (CAP) in children and adolescents, accounting for up to 20% of all CAP cases in this age group.[1] Despite its common occurrence, MP infections can present with a wide spectrum of clinical manifestations, ranging from mild upper respiratory tract symptoms to severe pneumonia necessitating hospitalization.[2] Among the serious complications of MP pneumonia (MPP), pleural effusion (PE) represents a significant clinical concern, often associated with prolonged hospital stays, increased morbidity, and a more complicated course of illness.[3,4]

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Pleural effusion complicating MPP typically presents as a sterile exudate, with a high protein content and a predominantly lymphocytic profile.[5] The pathogenesis of PE in the context of MP infection is thought to involve an exaggerated immune response characterized by the release of pro-inflammatory cytokines and chemokines, leading to increased vascular permeability and fluid accumulation in the pleural space.[6,7] However, the specific mechanisms underlying the development of PE in some patients with MPP remain incompletely understood, and the identification of clinical and laboratory parameters that predict this complication is crucial for early recognition and effective management.[8]

Previous studies have explored various demographic, clinical, and laboratory factors associated with the development of PE in MPP.[9] These include age, gender, comorbid conditions, and laboratory markers of inflammation.[10] However, the literature is inconsistent regarding the predictive value of these factors, partly due to differences in study designs, patient populations, and statistical approaches.[11] Moreover, the impact of antibiotic treatment regimens and adjunctive therapies on the development of PE has not been thoroughly elucidated.[11]

A thorough understanding of the clinical characteristics and predictive factors for PE in children with MPP can guide clinicians in anticipating and managing this complication.[12] Early recognition of children at risk for PE could lead to closer monitoring, earlier intervention, and potentially improved outcomes.[13] Given the paucity of comprehensive data addressing the clinical characteristics and predictive factors for PE in children with MPP, our study aims to fill this gap in knowledge. By analyzing a large cohort of pediatric patients treated at our institution for MPP over a three-year period, we sought to identify demographic, clinical, and laboratory features that are associated with the development of PE. We hypothesized that specific demographic and clinical parameters, as well as laboratory markers of inflammation, would be predictive of PE in children with MPP.

MATERIALS AND METHODS

Study Design

This study is a retrospective case-control study that analyzed pediatric patients with MPP treated at the Third Affiliated Hospital of Wenzhou Medical University from January 2021 to January 2024. Patients were classified into two groups based on the presence or absence of PE: the MPP group (n=412) and the MPP+PE group (n=82). This study was approved by the Ethics Committee of Wenzhou Provincial Children's Hospital (approval number: YJ2024001). As this was a retrospective study using anonymized data, informed consent was waived by the ethics committee.

Inclusion Criteria

Participants had to meet the diagnostic criteria[14,15] for MPP confirmed through clinical examinations such as pulmonary imaging and serology, have normal metabolic and neurological functions, and have complete clinical data. Patients with incomplete datasets were excluded from the study.

Exclusion Criteria

Excluded were individuals with infections from viruses, chlamydia, or other pathogens, those with concurrent tuberculosis or viral pneumonia, children with mental disorders, abnormal coagulation function, significant heart, liver, or kidney impairment, neurological disorders, or other infectious diseases.

Data Collection

Demographic data collection included age, gender, body mass index (BMI), number of siblings, family size, history of respiratory diseases, allergic rhinitis, immunocompromised status (defined as the presence of primary or secondary immunodeficiency, including but not limited to HIV infection, primary immunodeficiency disorders, malignancy, chronic use of immunosuppressive medications like corticosteroids or other immunosuppressive agents, and history of organ transplantation), duration of breastfeeding, exposure to smoke at home, family history of asthma, pet contact, and urban residence. Clinical symptoms and laboratory data collection involved respiratory rate, wheezing, blood oxygen saturation, duration of fever, white blood cell count (WBC), C-reactive protein (CRP) levels, erythrocyte sedimentation rate (ESR), and procalcitonin levels. Radiological findings included infiltrates, bronchial wall thickening, and atelectasis. Details on antibiotic treatment were recorded, including duration of antibiotic use, inhaled corticosteroids (ICS), treatment response, and length of hospital stay.

Blood Sample Analysis

In this study, blood test data were collected from enrolled patients at the time of their first hospitalization, including white blood cell count (WBC), C-reactive protein (CRP), procalcitonin (PCT), and erythrocyte sedimentation rate (ESR). All samples were obtained as fasting venous blood, adhering to strict collection and processing protocols.

Radiological Examination

On the first day of admission, pediatric patients underwent chest computed tomography (CT) scans (from the chest to the bottom of the lungs) using a spiral CT scanner (Philips CT64) with a slice thickness of 5mm, a voltage of 120 kV, current of 220 mA. Following routine scanning, iodixanol injection (Shanghai SITAI Pharmaceutical, approval number H20203432, 100 mL:32 g (I)) of 80 mL was given along with 30 mL normal saline was administered, followed by an AI-triggered scan with a 5-second delay.[16] Two attending physicians and two radiologists simultaneously analyzed the images to observe the distribution, morphological and density characteristics, and surrounding lesions of suspicious lesions.

On the first day of admission, pediatric patients underwent chest computed tomography (CT) using a spiral CT scanner (GE CT 62 slice), with a slice thickness of 5 mm, an voltage of 120 kV, and a current of 135 mA. After the routine scan was completed, the data was reconstructed and simultaneously uploaded to the PACS and AI system (Shenrui Medical, Deepwise). Two attending physicians analyzed the images in conjunction with the AI system, observing the distribution, morphology, and density characteristics of any suspicious lesions.

Statistical Methods

Data analysis was performed using SPSS 29.0 statistical software (SPSS Inc., Chicago, IL, USA). Categorical variables were expressed as [n (%)] and Chi-square tests were applied using the basic formula when the sample size was≥40 and theoretical frequency (T) was≥5. For sample sizes≥40 but theoretical frequency between 1 and<5, the corrected formula for the Chi-square test was used. When the sample size was<40 or the theoretical frequency was<1, statistical analysis was conducted using Fisher's exact probability method. Continuous variables were tested for normal distribution using the Shapiro-Wilk method. Normally distributed continuous variables were presented as (Mean±SD) and analyzed using a t-test with corrected variance. Multivariate logistic regression analysis was performed to identify independent predictors of PE. A joint prediction model was then constructed incorporating all significant independent factors identified in the multivariate analysis, and its predictive performance was assessed using receiver operating characteristic curve (ROC) analysis. A two-tailed p value<0.05 was considered statistically significant.

RESULTS

Demographic Characteristics

The immunocompromised state was significantly higher in the MPP+PE group (18.29% vs. 8.98%; p=0.021) (Table 1). These findings suggest that while demographic and several environmental factors do not discriminate between the two groups, immunocompromised status may play a role in the development of PE in children with Mycoplasma pneumonia.

Clinical Symptoms and Labs Test at Admission

Upon admission, children with MPP complicated by PE (MPP+PE) exhibited significantly higher respiratory rates (29.94/min vs. 29.16/min; p=0.003), lower oxygen saturation (82.33% vs. 83.14%; p=0.012), longer fever duration (5.75 days vs. 4.83 days; p=0.007), and elevated WBC (11.64 $\times 10^9$/L vs. 10.12$\times 10^9$/L; p=0.003) compared to those with uncomplicated MPP (MP) (Table 2). Additionally, the ESR were higher in the MPP+PE group (20.66 mm/h vs. 19.49 mm/h; p=0.006), and procalcitonin levels were also significantly increased (0.29 ng/L vs. 0.25 ng/L; p=0.019) These findings highlight the distinct clinical and laboratory profiles associated with the complication of PE in pediatric patients with MPP.

Radiological Findings

Radiological findings revealed significant differences in infiltrates between children with MPP and those with MPP+PE, occurring in 21.95% vs 12.14% respectively ($\chi 2$=4.754, p=0.029) (Table 3) These data underscore the association between PE and the increased prevalence of infiltrates in radiological images of pediatric patients with MPP.

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Table 1. Demographic Characteristics of the Study Population

Parameter	MPP (n=412)	MPP+PE (n=82)	t/χ^2	*p*
Age (years)	7.05±1.05	7.11±1.15	0.386	0.700
Gender (M/F)	217 (52.67%)/195 (47.33%)	38 (46.34%)/44 (53.66%)	0.858	0.354
BMI (kg/m^2)	12.25±2.25	12.37±2.41	0.411	0.682
Number of Siblings	2.38±1.04	2.51±1.28	0.83	0.409
Household Size	4.86±2.15	5.14±2.17	1.079	0.283
Previous Respiratory Diseases (%)	124 (30.1%)	22 (26.83%)	0.211	0.646
Allergic Rhinitis (%)	103 (25%)	23 (28.05%)	0.193	0.660
Immunocompromised State (%)	37 (8.98%)	15 (18.29%)	5.347	0.021
Breastfeeding Duration (months)	13.54±3.28	12.96 ± 2.94	1.579	0.117
Exposure to Smoke at Home (%)	107 (25.97%)	20 (24.39%)	0.026	0.872
Family History of Asthma (%)	90 (21.84%)	21 (25.61%)	0.361	0.548
Exposure to Pets (%)	98 (23.79%)	22 (26.83%)	0.199	0.656
Living in Urban Area (%)	268 (65.05%)	50 (60.98%)	0.333	0.564

MPP: P pneumonia; PE:pleural effusion; BMI: body mass index.

Table 2. Clinical Symptoms and Labs test at Admission

Parameter	MPP (n=412)	MPP+PE (n=82)	t/χ^2	*p*
Respiratory Rate (/min)	29.16±2.36	29.94±2.07	3.029	0.003
Presence of Wheezing (%)	26 (6.31%)	7 (8.54%)	0.245	0.621
Oxygen Saturation (%)	83.14±2.26	82.33 ± 2.68	2.554	0.012
Fever Duration (days)	4.83±2.35	5.75±2.86	2.739	0.007
White Blood Cell (×10^9/L)	10.12±4.78	11.64 ± 4.06	3.01	0.003
C-reactive Protein (mg/L)	22.35±3.08	22.71±2.83	1.040	0.300
Erythrocyte Sedimentation Rate (mm/h)	19.49±3.05	20.66±3.51	2.829	0.006
Procalcitonin Levels (ng/L)	0.25±0.12	0.29±0.14	2.373	0.019

Table 3. Radiological Findings

Parameter	MPP (n=412)	MPP+PE (n=82)	t/χ^2	*p*
Infiltrates (%)	50 (12.14%)	18 (21.95%)	4.754	0.029
Bronchial Wall Thickening (%)	47 (11.41%)	8 (9.76%)	0.059	0.809
Atelectasis (%)	24 (5.83%)	4 (4.88%)	0.006	0.938

Antibiotic Treatment Details

Children with MPP+PE received antibiotics for a significantly longer duration than those without PE (9.14±4.91 days vs 7.46 ± 3.29 days; p=0.004) (Figure 1). Furthermore, the use of ICS was markedly higher in the MPP+PE group (39.02% vs. 4.85%; χ^2=81.193, p<0.001), as was the rate of treatment response (40.24% vs 27.43%; χ^2=4.798, p=0.028) (Table 4). Additionally, the length of hospital stay was significantly longer for patients with PE (13.58±4.18 days vs. 12.37±3.52 days; p=0.015). These findings indicate that the presence of PE is associated with more intensive antibiotic therapy, greater use of adjunctive treatments, improved treatment response, and prolonged hospitalization in children with MPP.

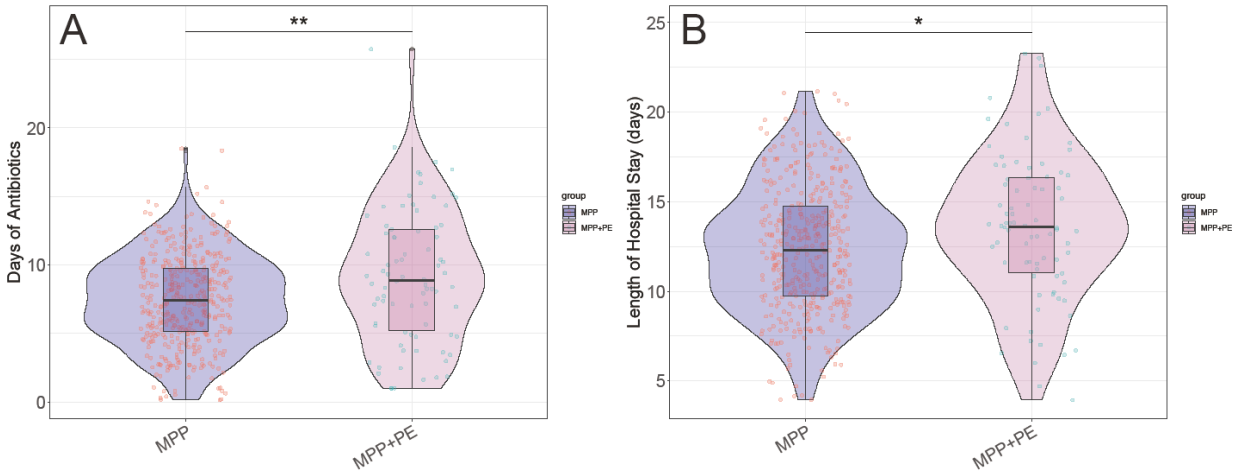

Figure 1. Antibiotic Treatment Time. (A) Days of Antibiotics; (B) Length of Hospital Stay (days). MPP: P pneumonia; PE: pleural effusion.

Table 4. Antibiotic Treatment Details

Parameter	MPP (n=412)	MPP+PE (n=82)	t/χ^2	*p*
Use of Inhaled Corticosteroids (%)	20 (4.85%)	32 (39.02%)	81.193	*0*<0.001
Treatment Response (%)	113 (27.43%)	33 (40.24%)	4.798	0.028

Multivariate Logistic Regression Analysis

Multivariate logistic regression analysis identified several parameters significantly associated with the complication of PE in children with MPP (Table 5). Increased fever duration (OR, 1.227; 95% CI, 1.090-1.383; p<0.001), higher WBC (OR, 1.103; 95% CI, 1.034-1.178; p=0.003), elevated ESR (OR, 1.132; 95% CI, 1.032-1.242; p=0.009), increased procalcitonin levels (OR, 42.099; 95% CI, 3.811-464.991; p=0.002), prolonged antibiotic treatment (OR, 1.122; 95% CI, 1.038-1.212; p=0.004), and the use of ICS (OR, 17.476; 95% CI, 8.013-38.112; p<0.001) were predictive of PE. Decreased oxygen saturation (OR, 0.870; 95% CI, 0.766-0.988; p=0.032) and longer hospital stays (OR, 1.094; 95% CI, 1.008-1.187; p=0.031) were also linked to the complication. Respiratory rate, presence of infiltrates, and treatment response did not show significant associations.

ROC Analysis of Independent Influencing Factors

ROC analysis was conducted to evaluate the diagnostic accuracy of various clinical and laboratory

parameters for predicting MPP complicated with PE in children (Table 6). Among the assessed parameters, the use of ICS showed the highest area under the curve (AUC) of 0.671 with a Youden index of 0.341, indicating moderate discriminatory power. Length of hospital stay had an AUC of 0.591, and days of antibiotics had an AUC of 0.599. Other parameters, including oxygen saturation, fever duration, WBC, ESR, and procalcitonin levels, demonstrated poor to moderate discriminatory ability with AUCs ranging from 0.575 to 0.596. Sensitivities and specificities varied across parameters, with oxygen saturation showing high specificity (0.954) but low sensitivity (0.207), and fever duration and ESR exhibiting moderate sensitivities and specificities. The joint prediction model (Figure 2) utilizing all independent influencing factors achieved an AUC value of 0.842, suggesting good discriminatory capacity for identifying MPP complicated with PE in children. The optimal cut-off point for this model was determined to be 0.158, with a sensitivity of 0.796 and specificity of 0.793. This indicates that combining these factors can effectively predict the likelihood of developing PE in pediatric patients with MPP.

Table 5. Multivariate logistic regression analysis between Complicated PE and various Parameters

Parameter	Coefficient	Std Error	Wald Stat	*p*	OR	95% CI
Immunocompromised State (%)	0.356	0.433	0.821	0.411	1.427	0.610-3.338
Respiratory Rate (/min)	0.124	0.067	1.870	0.061	1.132	0.994-1.290
Oxygen Saturation (%)	-0.139	0.065	-2.149	0.032	0.870	0.766-0.988
Fever Duration (days)	0.205	0.061	3.375	0<0.001	1.227	1.090-1.383
White Blood Cell ($\times10^9$/L)	0.098	0.033	2.956	0.003	1.103	1.034-1.178
Erythrocyte Sedimentation Rate (mm/h)	0.124	0.047	2.628	0.009	1.132	1.032-1.242
Procalcitonin Levels (ng/L)	3.740	1.226	3.052	0.002	42.099	3.811-464.991
Infiltrates (%)	0.552	0.385	1.435	0.151	1.737	0.817-3.694
Days of Antibiotics	0.115	0.039	2.917	0.004	1.122	1.038-1.212
Use of Inhaled Corticosteroids (%)	2.861	0.398	7.191	0<0.001	17.476	8.013-38.112
Treatment Response (%)	0.472	0.315	1.498	0.134	1.603	0.865-2.971
Length of Hospital Stay (days)	0.090	0.042	2.161	0.031	1.094	1.008-1.187

PE:pleural effusion

Table 6. ROC analysis

Parameter	Sensitivities	Specificities	AUC	Youden index
Oxygen Saturation (%)	0.207	0.954	0.575	0.161
Fever Duration (days)	0.415	0.755	0.587	0.17
White Blood Cell ($\times10^9$/L)	0.646	0.534	0.596	0.18
Erythrocyte Sedimentation Rate (mm/h)	0.439	0.75	0.606	0.189
Procalcitonin Levels (ng/L)	0.341	0.816	0.578	0.157
Days of Antibiotics	0.317	0.91	0.599	0.227
Use of Inhaled Corticosteroids (%)	0.39	0.951	0.671	0.341
Length of Hospital Stay (days)	0.561	0.612	0.591	0.173

ROC: receiver operating characteristic

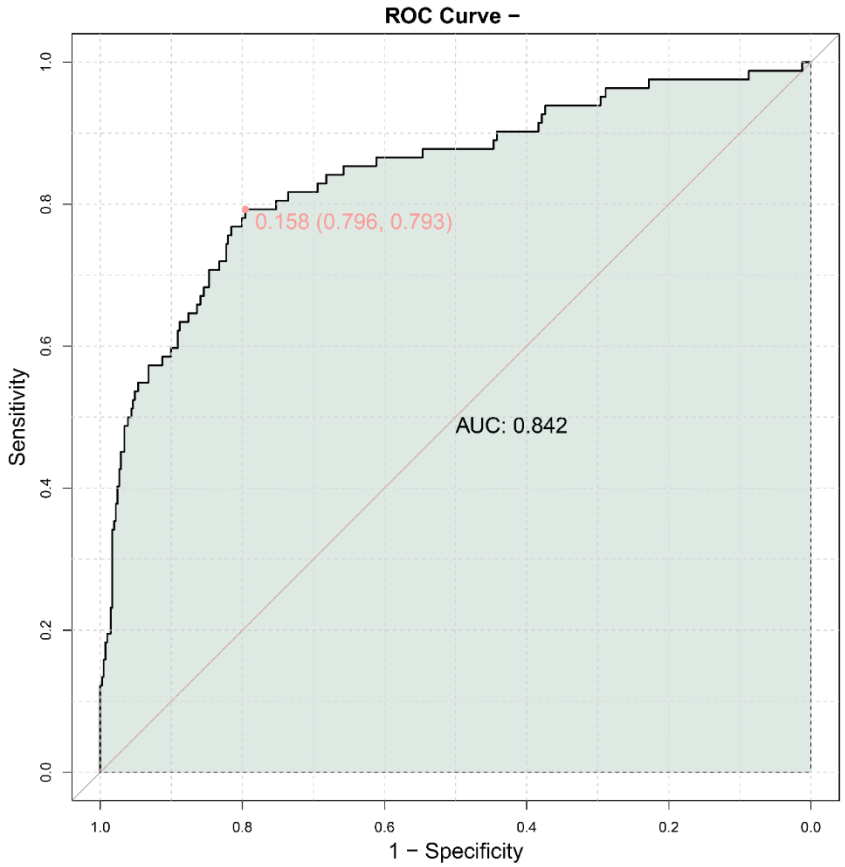

Figure 2. The ROC curve of the joint prediction model utilizing all independent influencing factors. ROC: receiver operating characteristic; AUC: area under the curve.

DISCUSSION

Our study on the clinical characteristics and predictive factors for MPP complicated by PE in children provides insights into the pathophysiological mechanisms and clinical management implications of this condition. Our findings underscore the complex interplay between host immune response, disease severity, and treatment outcomes in pediatric MPP cases.

We observed that immunocompromised status was significantly more prevalent in the MPP+PE group compared to those without PE, highlighting its potential role as a risk factor for PE development. Immunocompromised states may predispose children to more severe disease manifestations due to impaired clearance of MPP, potentially leading to excessive inflammatory responses and pleural involvement.[17,18] This finding aligns with previous research, such as a recent analysis highlighting that patients with a-hypogammaglobulinemia can be at increased risk of severe MP infections, including those complicated by pleural effusions.[19] While MP is not traditionally considered an opportunistic pathogen, these findings suggest that individuals with specific antibody deficiencies may be more susceptible to severe MP manifestations. Further research is needed to elucidate the precise mechanisms underlying this increased susceptibility and determine whether routine immunoglobulin screening is warranted in children diagnosed with MP, particularly those with severe disease or risk factors for immunodeficiency. This aligns with previous research[20] suggesting that compromised immune systems contribute to more severe MP infections.

This susceptibility to more severe inflammatory responses in immunocompromised individuals could be linked to several factors. Firstly, these individuals may have a reduced capacity to effectively clear the initial MP infection, leading to prolonged antigen exposure and the stimulation of the immune system. This prolonged stimulation can trigger an excessive release of pro-

inflammatory cytokines, such as IL-6, IL-8, and TNF-α, which are known to play a crucial role in the pathogenesis of MPP and its complications. These cytokines contribute to increased vascular permeability in the lungs, facilitating the leakage of fluid and inflammatory cells into the pleural space, ultimately leading to the development of PE. Secondly, the immune dysregulation often observed in immunocompromised states could further exacerbate the inflammatory response. For instance, an imbalance in T helper cell subsets, particularly a decrease in regulatory T cells (Tregs), which are crucial for suppressing excessive inflammation, could contribute to the uncontrolled inflammation seen in MPP+PE. This heightened and prolonged inflammatory cascade, driven by persistent MP antigen and potential immune dysregulation, underscores the vulnerability of immunocompromised children to developing PE.

Clinical symptoms and laboratory findings at admission revealed distinct patterns in children with MPP+PE. Higher respiratory rates, lower oxygen saturation, longer fever duration, and elevated WBC were noted. These findings are indicative of more severe lung inflammation and compromised gas exchange, which are hallmarks of MP-induced pleuritis and PE.[21] The elevated WBC count suggests an ongoing acute inflammatory response, possibly exacerbated by MP-induced cytokine storm, leading to pleural fluid accumulation.[22,23]

The observed association between elevated WBC count and MPP+PE further supports the role of a dysregulated inflammatory response in PE development. The increase in WBC, particularly neutrophils in the early stages of infection, is indicative of the body's attempt to clear the MP infection. However, in severe cases, this inflammatory response can become excessive and contribute to tissue damage. Neutrophils, while essential for pathogen clearance, release various inflammatory mediators and reactive oxygen species that can damage the pleural membrane, increasing its permeability and contributing to fluid accumulation. This uncontrolled inflammatory response, potentially driven by a cytokine storm, underscores the importance of monitoring inflammatory markers like WBC in identifying children at risk for developing PE.

The radiological finding of increased infiltrates in the MPP+PE group supports the notion that PE is associated with more extensive lung involvement. While bronchial wall thickening and atelectasis were not significantly different between groups, the presence of infiltrates may reflect more severe lung parenchymal damage, which could facilitate pleural fluid accumulation through increased capillary permeability.[24,25]

Our data on antibiotic treatment details reveal that children with MPP+PE required longer antibiotic courses, likely reflecting more severe disease and delayed resolution of infection. The increased use of ICS in the MPP+PE group is noteworthy. ICS can help mitigate airway inflammation and improve lung function in severe pneumonia cases.[26,27] the association between ICS use and PE in our study should be interpreted cautiously. While ICS can be beneficial in managing severe respiratory symptoms, its increased use in the MPP+PE group might reflect a treatment bias rather than a direct causal link to PE development. It is plausible that clinicians were more likely to prescribe ICS to children with more severe MPP, who were also more likely to develop PE due to their underlying disease severity. Therefore, further research is needed to elucidate the precise role of ICS in the context of MPP+PE and determine whether its use directly contributes to PE development or simply reflects a marker of disease severity. The higher treatment response rate in the MPP+PE group might be attributed to the more aggressive treatment regimen, including ICS, which could have contributed to better clinical outcomes despite the complexity of the disease.[28,29]

The multivariate logistic regression analysis identified several key predictors of PE in children with MPP. Longer fever duration, higher WBC count, increased ESR, elevated procalcitonin levels, prolonged antibiotic treatment, and the use of ICS were all positively associated with PE. These findings reinforce the concept that PE in MPP is associated with a heightened systemic inflammatory response, possibly compounded by a dysregulated immune reaction to MP infection.

Decreased oxygen saturation and longer hospital stays were also linked to PE. Hypoxemia could exacerbate the inflammatory cascade, leading to further lung injury and pleural fluid accumulation.[30,31] Longer hospital stays could be reflective of the more severe clinical course and complications associated with MPP+PE.

ROC analysis of independent influencing factors revealed that ICS use had the highest discriminatory power for predicting MPP+PE, suggesting that ICS

administration could serve as a marker for more severe disease requiring additional therapeutic interventions. However, it is important to note that the predictive value of individual parameters was only moderate, underscoring the multifactorial nature of MPP+PE development. The joint prediction model utilizing all independent influencing factors achieved a higher AUC, indicating that a combination of factors can more accurately predict the likelihood of developing PE in MPP.

Our study has several implications for clinical practice. It underscores the importance of early recognition and aggressive management of immunocompromised children with MPP to prevent PE. The findings also suggest that close monitoring of oxygen saturation, fever duration, and inflammatory markers could aid in the timely identification of children at risk for PE. Limitations of our study include its retrospective design, which precludes causal inference, and the potential for selection bias and unmeasured confounding variables. The single-center nature of our study limits the generalizability of our findings, and further research in diverse populations is needed to validate these results. Additionally, while we identified several clinical predictors of PE, our study did not delve into the specific immunological mechanisms underlying PE development in MPP. Furthermore, we did not consider the presence of MP-related extra-pulmonary diseases, which have been linked to severe MP infections and could potentially contribute to PE development. Future studies should incorporate this aspect for a more comprehensive analysis. Future prospective studies with larger sample sizes are needed to confirm our findings and explore the underlying mechanisms linking MPP to PE in greater depth, particularly focusing on the roles of specific inflammatory mediators and immune cell subsets.

In conclusion, our study provides valuable insights into the clinical characteristics and predictive factors associated with PE in children with MPP. Specifically, we found that a combination of factors, including longer fever duration, higher WBC count, increased ESR, elevated procalcitonin levels, prolonged antibiotic treatment, use of ICS, decreased oxygen saturation, and longer hospital stays, can effectively predict the likelihood of developing PE in these patients. These findings have important implications for the clinical management of MPP and suggest potential avenues for targeted interventions aimed at preventing or mitigating pleural complications.

STATEMENT OF ETHICS

This study was approved by the Ethics Committee of the third Affiliated Hospital of Wenzhou Medical University (YJ2024001).

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

ACKNOWLEDGMENTS

Not applicable.

DATA AVAILABILITY

The data are available from the corresponding author on reasonable request (lichu72061@163.com).

AI ASSISTANCE DISCLOSURE

Not applicable.

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