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ORIGINAL RESEARCH

Clinical Diagnosis and Treatment of 31 Children with *Mycoplasma pneumoniae* Necrotizing Pneumonia

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Objective: This study aimed to summarize the clinical characteristics and outcomes of necrotizing pneumonia caused by *Mycobacterium pneumoniae* in children.

Methods: The clinical features, accessory examinations, treatments, and prognoses of 31 children with M. pneumoniae necrotizing pneumonia admitted to our hospital between January 2020 and June 2023 were retrospectively analyzed.

Results: There were 31 patients, including 14 males and 17 females, with an average age of (6.77 ± 0.98) years. The length of hospitalization was 23.38 ± 5.56 days, with an average length of fever (22.10 ± 5.64) days. At the beginning of the disease, all patients had high fever and cough. Respiratory sounds were reduced in 29 cases, rales were audible in 13 cases, and wheezing sounds were audible in 4 cases. Pulmonary imaging revealed multiple alveolar or cavity formations in the lung consolidation areas and pleural effusion in 29 cases. The peak value of WBC in peripheral blood was (17.84 ± 3.72) × 10^9/L, and the proportion of neutrophils was (80.83 ± 4.19) %. C-reactive protein peak was (131.68 ± 36.47) mg/L, and D-dimer peak value was (6.79 ± 2.22) mg/L; The course of pulmonary necrosis was (11.61 ± 3.48) days. Bronchoscopy and lavage were performed on 30 patients. Two patients underwent closed thoracic drainage, and one patient underwent pulmonary lobectomy. After comprehensive treatment with macrolide antibiotics and glucocorticoids, the patient's prognosis improved.

Conclusion: Mycoplasma pneumoniae necrotizing pneumonia is more common in preschool and school-aged children with a long heat course and hospital stay. When WBC, neutrophil ratio, D-dimer, and C-reactive protein levels increase significantly, combined with pleural effusion, CT examination of the lungs should be performed to identify M. pneumoniae necrotizing pneumonia at an early stage. Despite a long disease course, most patients have a good prognosis after active treatment.

Keywords: *Mycoplasma pneumoniae*, necrotizing pneumonia, children

Introduction

Mycoplasma pneumoniae is a leading causative pathogen of community-acquired pneumonia in children, particularly among school-aged children and adolescents, accounting for 20–40% of such cases in the general population and even higher rates during epidemic cycles.[1,2] While M. pneumoniae pneumonia (MPP) is typically a self-limiting disease, it can present with severe and extra-pulmonary manifestations. Among its most severe complications is necrotizing pneumonia (NP), a condition characterized by the development of multiple thin-walled cavities within an area of consolidated lung tissue, resulting from parenchymal destruction.[3]

In recent years, an increase in the number of reported cases of M. pneumoniae necrotizing pneumonia (MPNP) has been observed both globally and within China, largely attributed to a greater awareness of the disease among clinicians and the widespread use of chest computed tomography (CT) for diagnosis.[4–7] Despite this apparent rise, MPNP remains a relatively uncommon condition, and the available literature consists predominantly of small case series or isolated case

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reports. This scarcity of large-scale data limits a comprehensive understanding of its clinical trajectory, optimal management strategies, and long-term outcomes. Consequently, clinicians often face diagnostic and therapeutic challenges, as the disease is associated with a protracted febrile course, prolonged hospitalization, and significant morbidity.

The general approach to treating MPP involves the use of macrolide antibiotics. However, in cases that progress to NP, this treatment is often insufficient due to the intense inflammatory response and associated complications like pleural effusion and necrosis. Adjunctive therapies, such as systemic glucocorticoids to modulate inflammation, anticoagulants to address hypercoagulable states, and interventional procedures like bronchoscopy with lavage or even surgical drainage, are frequently required. The lack of standardized guidelines for MPNP, stemming from the limited evidence base, makes management particularly complex.

Therefore, to strengthen the understanding of this severe disease and improve clinical diagnosis and treatment, we conducted a retrospective analysis. This study aims to summarize the clinical characteristics, treatment modalities, and outcomes of 31 children diagnosed with MPNP and hospitalized at our institution between January 2020 and June 2023, thereby providing a more detailed context for this patient population.

Object and Method

Patients

All 31 children with M. pneumoniae necrotizing pneumonia met the diagnostic criteria for MPP:[5] (1) fever and cough were the main clinical manifestations; (2) Chest X-ray or chest CT indicated pneumonia; (3) Single MP antibody titer ≥ 1:160; MP antibody titer was increased or decreased by ≥4 times in the convalescent and acute stages; and MP-IgM was positive in a single test. Nasopharyngeal swabs or alveolar lavage fluid were positive for MP-DNA.

Inclusion Criteria

Chest radiography or CT showed liquefaction necrosis in the lung consolidation area, forming multiple thin-walled voids or vesicles, which could fuse into large voids, thus meeting the diagnostic criteria for necrotic pneumonia.[6]

Exclusion Criteria

(1) Exclusion of pulmonary tuberculosis, congenital lung cyst, lung abscess, and other cavitary lung diseases; (2) previous bronchial asthma, repeated respiratory infection, primary or secondary immune deficiency, congenital heart disease, etc.; and (3) incomplete medical records. Among the 31 patients, 14 were males and 17 were females, with the average age of (6.77 ± 0.98) years.

Data Collection

The age, clinical manifestations, imaging data, laboratory examination, bronchoscopy, treatment, and prognosis of the children were retrospectively analyzed, including routine blood examinations and C-reactive protein, lactate dehydrogenase, and D-dimer levels.

Treatment Rationale and Protocols

All patients received a standardized, stepwise treatment approach based on their clinical status and laboratory findings. The rationale for each intervention was as follows:

Antibiotic Therapy

Intravenous azithromycin (10 mg/kg/day) was administered as the first-line anti-mycoplasmal agent to all patients, as per guidelines. This was followed by sequential oral therapy to complete a 3–4 week course. A β-lactam antibiotic (eg., ceftriaxone or cefotaxime) was added if there was clinical, laboratory, or microbiological evidence of bacterial co-infection (eg., positive culture for bacteria, lack of defervescence after 72 hours of macrolide monotherapy, or a procalcitonin level suggestive of bacterial infection).

Glucocorticoid Therapy

Intravenous methylprednisolone (1–2 mg/kg/day) was initiated in patients exhibiting a persistent, excessive systemic inflammatory response. The specific indications were recurrent high fever (>39°C) lasting beyond 5–7 days of appropriate antibiotic therapy, accompanied by significant toxic symptoms and very high levels of inflammatory markers (eg., CRP > 100 mg/L). The goal was to modulate the hyperinflammation thought to contribute to disease progression and necrosis.

Anticoagulant Therapy

Patients with a peak D-dimer level exceeding 5 mg/L were considered to be in a hypercoagulable state and at risk for thrombotic complications. These patients received low molecular weight heparin at a prophylactic dose of 10–30 U/kg every 8 hours. Heparin was discontinued once the D-dimer level dropped below 5 mg/L.

Interventional Procedures

Bronchoscopy with Alveolar Lavage

This was performed on patients with persistent fever, clinical or radiological evidence of airway obstruction (atelectasis), or to obtain lower respiratory tract specimens for etiological diagnosis. The goal was to clear mucus plugs and inflammatory exudates, thereby improving ventilation and potentially reducing the local pathogen and inflammatory load.

Pleural Drainage

Thoracentesis or closed thoracic drainage was performed on patients with moderate to large pleural effusions causing respiratory distress or persistent fever. This aimed to relieve symptoms and prevent empyema.

Surgical Intervention

Surgical intervention (eg., lobectomy) was reserved for patients who developed complications such as a bronchopleural fistula or did not respond to maximal medical and minimally invasive interventional management.

Statistical Methods

Statistical software (SPSS 21.0) was used to analyze the data, and the measurement data were represented as mean ± standard deviation (x ± s). A descriptive analysis of the imaging data was performed.

Results

Clinical Manifestations

All 31 children had high fever and cough, an initially dry cough, some cough with white or yellow sputum, one case of coughing rust color sputum, two cases of chest pain on the affected side, and one case of abdominal pain. Respiratory sounds were reduced in 29 cases, rales were audible in 13, and wheezing in 4. One patient experienced dyspnea and required a nasal catheter for oxygen inhalation. The fever duration was (22.10 ± 5.64) days, the duration of pulmonary necrosis was (11.61 ± 3.48) days, and the length of hospital stay was (23.38 ± 5.56) days.

Laboratory Test Results

(1) Blood routine: WBC peak was (17.84 ± 3.72) × 10^9/L, with neutrophil ratio peak (80.83 ± 4.19) %; The peak value of C-reactive protein was (131.68 ± 36.47) mg/L, including C-reactive protein >100 mg/L in 23 cases; (2) Coagulation function: the peak value of D-dimer was (6.79 ± 2.22) mg/L, and 23 cases were >5 mg/L; (3) Lactate dehydrogenase: 12 cases were >500U/L, and 2 cases were higher than 1000U/L (Table 1).

Etiological Examination Results

Three cases were confirmed to be complicated by bacterial infection, one case was positive in pleural effusion culture, and one case was positive in alveolar lavage fluid culture, both of which showed Staphylococcus aureus growth. Another case showed Streptococcus pneumoniae in the sputum smear and staining; however, the blood cultures were negative.

Table 1 Laboratory Results of Mycoplasma pneumoniae Necrotizing Pneumonia in Children

Peak Value	Mean ± Standard Deviation
WBC ($\times 10^9$/L)	17.84 ± 3.72
Neutrophil ratio (%)	80.83 ± 4.19
C-reactiveprotein (mg/L)	131.68 ± 36.47
D-dimer (mg/L)	6.79 ± 2.22
Lactate dehydrogenase (U/L)	505.58 ± 201.35

Imageological Examination

Imaging results showed that unilateral or bilateral lung consolidation occurred in the early stage, and liquefaction necrosis occurred in the lung consolidation area, forming multiple thin-walled voids or vesicles that could fuse into large voids (Figure 1). There were 29 cases of unilateral lung involvement, including 19 cases of right lung involvement, 10 cases of left lung involvement, and two cases of bilateral lung involvement.

Results of Bronchoscopy

Thirty patients underwent bronchoscopy and alveolar lavage (Figure 2), 4 patients underwent only one examination, and the rest underwent repeated alveolar lavage (2–6 times). Endoscopic results are presented in Table 2.

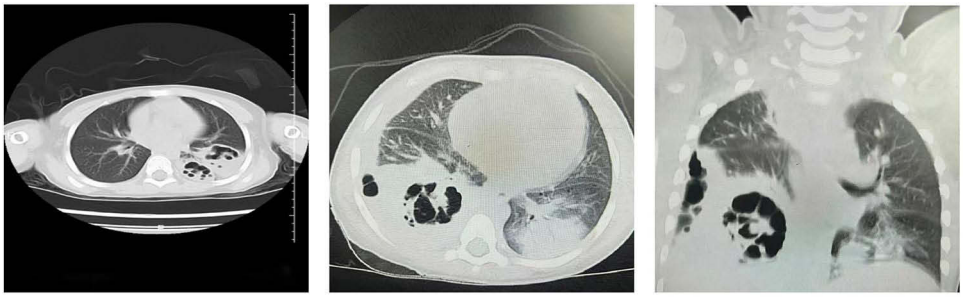

Figure 1 Lung imaging of children with Mycoplasma pneumoniae necrotizing pneumonia.

Figure 2 Alveolar lavage in children with Mycoplasma pneumoniae necrotizing pneumonia.

Table 2 Clinical Features and Bronchoscopic Changes in Children with Mycoplasma pneumoniae Necrotizing Pneumonia

Research Project	Number of Cases	Occurrence Rate (%)
Clinical feature		
Hyperpyrexia (>39°C)	31	100
Cough	31	100
Respiratory sounds reduced	29	93.5
Moist rale	13	41.9
Wheezing rale	4	12.9
Chest pain	2	6.5
Complications		
Liver injury	10	32.3
Myocardial damage	2	6.5
Pleural effusion	29	93.5
Rash	1	3.2
Bronchoscopic findings		
Mucosal hyperemia and edema	27	87.1
White or yellow sticky discharge	23	83.8
Sputum suppository formation	15	48.4
Mucosal fold hyperplasia	12	38.7
Lumen stenosis	11	35.5
Mucosal roughness	8	25.8
Mucosal erosion	6	19.4
Molding	2	6.5
Granulation tissue hyperplasia	1	3.2

Complications

Complications included liver injury in 10 cases, myocardial injury in 2 cases, skin rash in 1 case, and pleural effusion in 29 cases. Of these, 9 cases had moderate pleural effusion and 20 cases had small pleural effusion (Table 2).

Treatment

All 31 patients were administered intravenous azithromycin treatment, followed by oral azithromycin sequential treatment after discharge, with a total course of 3–4 weeks; Patients with bacterial infection combined with β-lactam antibiotics were considered. Patients with recurrent high fever and obvious symptoms of poisoning were considered to have excessive systemic inflammation and intravenous glucocorticoids were administered. In this group, 26 patients were given methylprednisolone 1–2 mg/(kg.d) for (12.9 ± 7.3) days. Twenty-three cases with D-dimer levels >5 mg/L, indicating hypercoagulability, were given heparin at 10–30U/(kg), once every 8 hours. Heparin was discontinued when D-dimer was lower than 5 mg/L. Two patients underwent closed thoracic drainage, 9 patients with moderate pleural effusion underwent thoracocentesis (1–2 times), and a small number of patients were absorbed by themselves. One patient with a bronchopleural fistula underwent lobectomy and pleurodesis.

After more than 3 months of follow-up, all clinical symptoms disappeared. To 3–6 months after discharge, CT examination of the lungs showed that the images had returned to normal, with a few remaining cystic changes, line shadows, or atectasis.

Discussion

NP represents a severe complication of community-acquired pneumonia, characterized by the destruction of lung parenchyma with the formation of multiple thin-walled cavities within areas of consolidation.[1,2] While historically associated with bacterial pathogens like Staphylococcus aureus, Mycoplasma pneumoniae has emerged as an important

causative agent in children. Prior to this study, the diagnosis of MPNP was largely dependent on the appearance of cavities on chest CT, which typically occurs in the second or third week of illness.[8] This reactive approach meant that the diagnosis was often delayed. Furthermore, while treatments such as macrolides, corticosteroids, and anticoagulants were used empirically in severe MPP, there were no clear, evidence-based protocols guiding their specific application in the context of MPNP. The clinical decision-making process regarding which patient should receive which adjunctive therapy remained unclear. Our study aimed to address this gap by identifying early clinical and laboratory predictors of MPNP and by outlining a structured treatment approach based on predefined criteria.

Our findings confirm that MPNP is predominantly a disease of preschool and school-aged children (mean age 6.77 ± 0.98 years), with a slight female predominance. The protracted clinical course, characterized by mean fever duration of 22.10 ± 5.64 days and hospitalization of 23.38 ± 5.56 days, underscores the severity of this condition and aligns with previous reports.[2,9] This prolonged course, often resistant to macrolide monotherapy, was the clinical reality that prompted the search for more aggressive, combined treatment strategies.

A key finding of our study is the identification of specific laboratory markers that can serve as early warning signs for the development of necrosis. We observed markedly elevated peak values of WBC (17.84 ± 3.72 × 10^9/L), neutrophil ratio (80.83 ± 4.19%), CRP (131.68 ± 36.47 mg/L), and D-dimer (6.79 ± 2.22 mg/L). While elevated inflammatory markers were a known feature of severe MPP, their magnitude in our cohort—with 74% of patients having CRP >100 mg/L and D-dimer >5 mg/L—provides a quantifiable threshold for concern. Previously, clinicians might have noted these elevations but lacked a clear framework for action. Our study suggests that when these markers reach such levels, especially in a child with persistent fever and consolidative changes on initial chest X-ray, the probability of progression to NP is high. This finding directly informs clinical practice by providing an evidence-based trigger for early chest CT, moving from a reactive to a proactive diagnostic approach. Early CT imaging, in turn, allows for the timely implementation of the comprehensive treatment protocol described below.

The treatment of MPNP in our center was guided by a structured protocol based on clinical and laboratory parameters. All patients received macrolides as the backbone of anti-mycoplasmal therapy. However, the recognition that macrolides alone are insufficient to control the excessive inflammatory response in MPNP led to the incorporation of adjunctive therapies, a practice that was evolving but not standardized before this study.

The decision to add systemic glucocorticoids was based on the presence of a persistent hyperinflammatory state (recurrent high fever, toxic appearance, very high CRP). While the immunomodulatory role of corticosteroids in severe MPP had been previously discussed,[5] our study provides specific, practical criteria for their initiation in the context of MPNP. The favorable outcome in the 26 patients who received methylprednisolone supports this targeted approach, suggesting that dampening the host inflammatory response is crucial to limiting tissue destruction.

Similarly, the use of low molecular weight heparin was guided by a specific D-dimer threshold (>5 mg/L). D-dimer elevation, reflecting coagulation activation and fibrinolysis, was a known phenomenon in severe infections. However, its role as a direct guide for anticoagulation therapy in MPNP was not well-established. Our protocol, which led to the treatment of 23 patients with heparin and discontinuation upon D-dimer normalization, represents a practical application of this biomarker to mitigate the risk of thrombotic complications, a significant refinement over prior, more empirical approaches.

Bronchoscopy with alveolar lavage was performed in 30 patients, not only for diagnostic purposes but also therapeutically. Before this study, the role of bronchoscopy in MPP was primarily diagnostic. Our experience supports an expanded therapeutic role. By clearing tenacious mucus plugs and inflammatory casts that cause airway obstruction and atelectasis, bronchoscopy likely improves ventilation, reduces the local pathogen and inflammatory burden, and may contribute to fever resolution and prevention of disease progression.

Despite the severity of the illness, the long-term prognosis was excellent, with all patients achieving clinical resolution and most showing significant radiographic improvement within 3–6 months. Only one patient with a bronchopleural fistula required surgical intervention. This favorable outcome, consistent with other reports,[2] is reassuring and suggests that with aggressive, multidisciplinary medical management, surgery can be avoided in the vast majority of cases.

This study has several important limitations that must be acknowledged when interpreting our findings. First, the sample size is relatively small (n = 31), which limits the statistical power of our analyses and precludes more sophisticated multivariable modeling. Second, as a single-center retrospective study, our findings may not be generalizable to other populations or healthcare settings, and they are subject to the inherent biases of retrospective data collection. Third, the absence of a control group (eg., children with severe MPP without necrosis or NP caused by other pathogens) makes it impossible to identify predictors that are specific to MPNP. Our findings regarding clinical and laboratory characteristics should therefore be viewed as descriptive rather than as validated predictive markers. Fourth, our follow-up period was limited to 3–6 months and focused on symptomatic and radiological resolution. We did not perform long-term pulmonary function tests, which would be valuable for assessing potential chronic sequelae such as restrictive lung disease or small airway dysfunction. Finally, while we performed exploratory analyses linking laboratory and imaging findings to outcomes, these analyses were underpowered and should be considered hypothesis-generating. Future multi-center, prospective studies with larger sample sizes, appropriate control groups, and long-term follow-up including pulmonary function testing are urgently needed to validate our observations and to establish evidence-based guidelines for the management of MPNP.

In conclusion, this study demonstrates that MPNP, while a severe and prolonged illness, carries a good prognosis with timely and comprehensive intervention. By connecting specific, quantifiable laboratory markers (WBC, neutrophil ratio, CRP, D-dimer) to the risk of necrosis, our findings provide clinicians with an evidence-based framework for early diagnosis via chest CT. Furthermore, by detailing a structured treatment protocol that integrates macrolides, glucocorticoids, anticoagulation, and bronchoscopy based on predefined clinical and laboratory criteria, we offer a practical roadmap for management. This study advances the field by moving the approach to MPNP from reactive and empirical to proactive and evidence-based, ultimately aiming to improve patient outcomes.

Data Sharing Statement

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

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of Weihai Central Hospital Affiliated to Qingdao University (Approval No.LL-2024-289). Written informed consent was obtained from the parents. This study was performed in line with the principles of the Declaration of Helsinki. All methods were carried out in accordance with relevant guidelines and regulations.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; 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.

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Disclosure

The authors declare that they have no competing interests.

References

1. Jain S, Williams DJ, Arnold SR, et al. Community-acquired pneumonia requiring hospitalization among U.S. children. *N Engl J Med*. 2015;372 (9):835–845. doi:10.1056/NEJMoa1405870

2. Waites KB, Xiao L, Liu Y, et al. Mycoplasma pneumoniae from the respiratory tract and beyond. *Clin Microbiol Rev*. 2017;30(3):747–809. doi:10.1128/CMR.00114-16
3. Nicolaou EV, Bartlett AH. Necrotizing pneumonia. *Pediatric Annals*. 2017;46(2):e65. doi:10.3928/19382359-20170120-02
4. Barreira ER, Souza DC, Góes PF, et al. Septic shock, necrotizing pneumonitis, and meningoencephalitis caused by Mycoplasma pneumoniae in a child: a case report. *Clin Pediatrics*. 2009;48(3):320. doi:10.1177/0009922808327108
5. Wang Y, Xu D, Li S, et al. Mycoplasma pneumoniae-associated necrotizing pneumonitis in children. *Pediatrics Int*. 2012;54(2):293–297. doi:10.1111/j.1442-200X.2011.03415.x
6. Zhang Y, Xie L, Feng T, et al. Clinical analysis of Mycoplasma pneumoniae necrotizing pneumonia in children. *BMC Infect Dis*. 2026;26(1). doi:10.1186/s12879-025-12495-w
7. Yang B, Zhang W, Gu W, et al. Differences of clinical features and prognosis between Mycoplasma pneumoniae necrotizing pneumonia and non-Mycoplasma pneumoniae necrotizing pneumonia in children. *BMC Infect Dis*. 2021;21(1):1–11. doi:10.1186/s12879-021-06469-x
8. Hou J, Sun R, Zhang X, et al. Chest CT characterization of children with necrotizing pneumonia due to Mycoplasma pneumoniae infection. *Sci Rep*. 2025;15(1):4283. doi:10.1038/s41598-025-88418-1
9. Teresinha Mocelin H, Bueno Fischer G, Danezi Piccini J, et al. Necrotizing Pneumonia In Children: a Review. *Paediatric Respir Rev*. 2024;52:51–57. doi:10.1016/j.prrv.2024.02.003