Infection and Drug Resistance

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Clinical Characteristics of *Mycoplasma pneumoniae* Pneumonia in Children and Analysis of the Risk Factors for Severe *Mycoplasma pneumoniae* Pneumonia

Guang Yang, Yunxia Qiao, Xinxin Sun, Tiandan Yang, Aiying Lv, Min Deng, Qi Yu

Department of Pediatrics, Shanxi Medical University, Taiyuan, Shanxi, 030001, People's Republic of China

Correspondence: Qi Yu, Department of Pediatrics, Shanxi Medical University, No. 56 of Xinjian South Street, Yinze District, Taiyuan, Shanxi, 030001, People's Republic of China, Tel +86-0351-4135143, Email yuqi241@126.com412506

Purpose: To analyse the clinical features of *Mycoplasma pneumoniae* pneumonia (MPP) and the independent risk factors of severe MPP (SMPP) and to provide a theoretical basis for the early diagnosis of SMPP and timely judgment of changes in the disease's progress.

Patients and Methods: The clinical data of children with MPP who were treated in our hospital from January 2021 to December 2022 were collected and retrospectively analysed. 83 cases were assigned to the severe group (SMPP group) and 152 cases were assigned to the general group (MPP group). The epidemiology, clinical characteristics and related influencing factors of SMPP were analysed.

Results: Compared with the MPP group, the SMPP group were older, had a longer fever time, had more imaging manifestations of large patchy consolidation and experienced a longer hospital stay. The peripheral blood white blood cell (WBC) count, C-reactive protein (CRP), serum IgA, serum IgG, serum IgM, lactate dehydrogenase (LDH) and ferritin (SF) in the SMPP group were significantly higher than those in the MPP group ($P < 0.05$). Logistic regression analysis showed that fever days (odds ratio [OR] = 1.31, 95% confidence interval [CI] = 1.03–1.65, $P = 0.021$), CRP (OR = 1.16, 95% CI = 1.02–1.10, $P = 0.006$), LDH (OR = 1.021, 95% CI = 1.01–1.02, $P = 0.002$) and large patchy consolidation (OR = 10.38, 95% CI = 4.31–31.02, $P = 0.001$) were independent factors of SMPP.

Conclusion: All patients with MMP had a fever and a cough, and patients with SMPP had a high fever. Chest imaging examination of patients with SMPP showed extensive consolidation shadows in the lungs, and laboratory examinations showed significant increases in WBCs, CRP, LDH and SF. The fever time, CRP, LDH and imaging showed large patchy consolidation shadows are related to the severity of the disease.

Keywords: *Mycoplasma pneumoniae* pneumonia, clinical features, severe patients, children

Introduction

Mycoplasma pneumoniae (MP) pneumonia (MPP) refers to lung inflammation caused by MP infection, which can affect the bronchi, bronchioles, alveoli and pulmonary interstitium.[1] It is the most common community-acquired pneumonia (CAP) among children aged five and above in China. Among hospitalised children with CAP, 10–40% have MPP, and this proportion can reach 50% in epidemic years.[1,2] It is mainly seen in school-age children and adolescents, but there has been a trend towards younger children in recent years, and infant infections are not uncommon.[3]

When MPP is severe and meets the criteria for severe CAP assessment, it is called severe *MPP* (SMPP).[4] The pathogenesis of SMPP is not yet clear, and it is currently believed to be related to excessive immune response, delayed use of macrolide antibiotics and drug resistance.[5] With the increase of MPP's resistance to macrolide antibiotics, the incidence rate of SMPP in children's CAP increased year by year. It progresses rapidly and has complex clinical

https://doi.org/10.2147/IDR.S412506
Received: 22 March 2023
Accepted: 16 June 2023
Published: 13 February 2026

manifestations, which can easily cause functional damage to the digestive, neurological, cardiovascular, skin mucosal and musculoskeletal systems.[6]

The relationship between the severity of MPP and clinical and relevant laboratory indicators is not fully understood, and there are currently no diagnostic and treatment guidelines for SMPP. Due to doctors' insufficient understanding of SMPP, it is easy to misdiagnose it as other serious diseases, and the optimal treatment time is often delayed, and excessive medical treatment occurs. Research has shown that early diagnosis and timely and effective treatment of SMPP can significantly improve the prognosis for children.[7] Early detection of severe cases, reasonable treatment and avoidance of death and sequelae are the core and key issues in the diagnosis and treatment of MPP.

Based on the above research background, a retrospective analysis was conducted on the clinical data of patients with MPP in our hospital from January 2021 to December 2022. The aim was to explore the clinical characteristics of MPP and the independent risk factors of SMPP to provide a theoretical basis for the early and timely diagnosis of disease changes in SMPP.

Materials and Methods

Study Subjects

A total of 235 children with acute MPP who were hospitalised in the Department of Respiratory Medicine of our hospital from January 2021 to December 2022 were selected as subjects by the convenient sampling method. The diagnostic criteria were based on the expert consensus on the diagnosis and treatment of MMP in children (2017).[4]

Inclusion criteria: (1) with cough, with or without fever and other respiratory manifestations and dry and wet rales or consolidation signs could be heard in bilateral auscultation; (2) imaging showed lobar infiltration, lobular patchy infiltration or interstitial changes in the lungs; and (3) met the criteria for MP infection of a single MP antibody (mainly MP-IgM) titre $\geq$ 1:160.

Exclusion criteria: (1) children with immune deficiency, (2) children with basic lung diseases (tuberculosis, tracheomalacia, etc.); or (3) children with other pathogenic infections.

Grouping criteria: According to the guidelines for the management of paediatric acquired pneumonia (revised in 2013),[8] children with any of the following manifestations were included in the SMPP group: (1) significantly increased breathing: respiratory rate >70 times/min in infants or >50 times/min in older children; (2) dehydration or refusal to eat; (3) consciousness disorders; (4) difficulty in breathing (moaning, nasal fan or triple concave sign); (5) the range of lung infiltration is multi-lobed involvement or $\geq$⅔ lung involvement; (6) pleural effusion; (7) pulse oxygen saturation $\leq$0.92; or (8) having extrapulmonary complications. Finally, 83 children were included in the SMPP group and 152 children were included in the MPP group.

Method

Clinical data: General information, such as the age and gender of all enrolled children, as well as clinical manifestations, such as high fever, cough severity, wheezing and wet rale, were collected.

Laboratory indicators: The white blood cell (WBC) count, C-reactive protein (CRP, kit purchased from Hunan Mindray Medical Technology Co., Ltd.), procalcitonin (PCT, kit purchased from Shandong Boke Biological Industry Co., Ltd.), immunoglobulin (IgA, IgG and IgM), lactate dehydrogenase (LDH, kit purchased from Tianjin Taide Hezhong Biotechnology Development Co., Ltd.), ferritin (SF, reagent kit purchased from Wuhan Mingde Biotechnology Co., Ltd.), etc. were collected. On the day of admission and about 10 d after the course of the disease, the serum MP antibody (MP IgM) titre was detected using a fully automated enzyme-linked immunosorbent assay analyser purchased from BioTck Company in the United States and the kit purchased from Shanghai Biyuntian Biotechnology Co., Ltd.

Imaging examination: All children admitted to the hospital underwent lung imaging examinations, such as chest X-ray (Shanghai Siemens Medical Equipment Co., Ltd.) and/or computed tomography (Shanghai Siemens Medical Equipment Co., Ltd.) to check for the presence of atelectasis, pleural effusion, etc.

Statistical Methods

The SPSS™ v21.0 statistical software was used for data analysis. The measurement data were analysed by the independent *t*-test and the count data were analysed by the x^2 test. After screening the factors with $P < 0.05$, the binary logistic regression analysis was performed. A value of $P < 0.05$ was considered statistically significant.

Results

Epidemiological Characteristics

Among the 235 patients, 113 were male (48.1%) and 122 were female (51.9%). They were aged between 8 months and 14 years, with an average age of 6.8 ± 4.7 years. The highest number of children by season was 96 (40.9%) in autumn, followed by 90 (38.3%) in winter, 32 (13.6%) in summer and at least 17 (7.2%) in spring.

Clinical Features

The main clinical manifestations of MPP were fever and cough. Most of them (211 cases, 89.3%) had a fever, and the duration of the fever was 6.4 ± 2.2 d. All children had a cough, mainly a dry cough (90 cases, 38.3%). Some (112 cases, 47.7%) had extrapulmonary system damage, and the cardiovascular system was dominated by myocardial damage (51 cases, 21.7%), arrhythmia (9 cases, 3.8%) and myocarditis (12 cases, 5.1%). The digestive system manifestations were mainly liver damage (15 cases, 11.1%), followed by vomiting and abdominal pain. The blood system manifestation was mainly neutropenia (26 cases, 6.7%). The SMPP group had pleural effusion (23 cases, 9.8%), followed by atelectasis (10 cases, 4.3%), pleurisy in two cases and emphysema in one case.

Comparison of Clinical Features and Laboratory Tests Between the Two Groups

The average age of patients with MMP was 5.68 ± 3.34 years, with more females than males (53.3% vs 46.7%), and the duration of the fever was 4.41 ± 2.77 d. The proportion of children with a wet cough was higher than that with a dry cough (47.4% vs 33.6%), and 29 patients (19.0%) had symptoms of wheezing. Imaging showed 31 (20.4%) of the patients had a large leakage, 100 (65.8%) had lobularity on imaging, and 21 (13.8%) had an interstitial. The average hospitalisation time was 9.34 ± 2.43 d.

The average age of patients with SMPP was 7.72 ± 4.08 years, with more males than females (50.6% vs 49.4%), and the duration of the fever was 6.42 ± 2.36 d. The proportion of children with a dry cough was higher than that with a wet cough (49.4% vs 47.0%), and 3 patients (3.6%) had symptoms of wheezing. Imaging showed 57 (69.5%) of the patients had a large leakage, 9 (11.0%) had lobularity on imaging, and 16 (19.5%) had an interstitial. The average hospitalisation time was 10.26 ± 3.12 d.

There were statistically significant differences between the two groups in terms of average age, fever duration, cough type, imaging manifestations and average hospitalisation days ($P < 0.05$). See Table 1.

The laboratory examination results showed that the average WBC count of patients with MPP was 9.21 ± 2.74 × 10% L, CRP was 11.52 ± 9.01 mg/L, PCT was 0.18 ± 0.29 ng/mL, IgA was 125.30 ± 83.25 mg/dL, IgG was 949.05 ± 240.52 mg/dL, IgM was 156.19 ± 80.19 mg/dL, LDH was 274.83 ± 68.21 U/L and SF was 115.59 ± 68.10 ng/mL.

In patients with SMPP, the mean WBC count was 10.6 ± 2.56 × 10% L, CRP was 23.65 ± 12.32 mg/l, PCT was 0.19 ± 0.39 ng/mL, IgA was 151.10 ± 68.95 mg/dL, IgG was 1103.50 ± 269.35 mg/dL, IgM was 179.21 ± 68.89 mg/dL, LDH was 347.75 ± 113.58 u/L and SF was 179.01 ± 121.71 ng/mL.

Except for PCT, the levels of WBCs, CRP, IgA, IgG, IgM, LDH and SF in the SMPP group were higher than those in the MPP group ($P < 0.05$). See Table 2.

Risk Factors for Severe *Mycoplasma pneumoniae* Pneumonia

Logistic regression analysis was performed with SMPP as the dependent variable, and age, fever duration, WBC count, CRP, IgA, IgG, IgM, LDH, SF and imaging manifestations of lobar consolidation as independent variables. The results showed that fever days (odds ratio [OR] = 1.31, 95% confidence interval [CI] = 1.03–1.65, $P = 0.021$), CRP (OR = 1.16,

Table 1 Comparison of Clinical Characteristics Between MPP and SMPP Groups

Index	MPP Group (n=152)	SMPP Group (n=83)	x^2/t	*P*
Age/year	5.68 ± 3.34	7.72 ± 4.08	3.21	0.002
Sex/M(%)	71(46.7)	42(50.6)	0.120	0.620
Fever time/d	4.41±2.77	6.42±2.36	5.231	0.001
Cough nature/n(%)				
Dry cough	51(33.6)	41(49.4)	4.533	0.033
Damp cough	72(47.4)	39(47.0)		
Gasp/n(%)	29(19.0)	3(3.6)	10.230	0.001
Imaging manifestation/n(%)				
Large leafy	31(20.4)	57(69.5)	75.791	<0.001
Lobularity	100(65.8)	9(11.0)		
Interstitial	21(13.8)	16(19.5)		
Hospital days/d	9.34±2.43	10.26 ± 3.12	7.458	<0.001

Abbreviations: MPP, Mycoplasma pneumoniae pneumonia; SMPP, Severe mycoplasma pneumoniae pneumonia.

Table 2 Comparison of Laboratory Indexes Between MPP and SMPP

Index	MPP Group (n=152)	SMPP Group (n=83)	*t*	*P*
WBC / (×10% L)	9.21±2.74	10.6±2.56	2.506	0.013
CRP / (mg/L)	11.52±9.01	23.65±12.32	5.781	0.001
PCT / (ng/mL)	0.18±0.29	0.19±0.39	0.062	0.937
IgA / (mg/dL)	125.30±83.25	151.10±68.95	2.536	0.030
IgG / (mg/dL)	949.05±240.52	1103.50±269.35	2.219	0.048
IgM / <mg/dL	156.19±80.19	179.21±68.89	1.943	0.046
LDH / (U/L)	274.83±68.21	347.75±113.58	4.616	<0.001
SF / (ng/mL)	115.59±68.10	179.01±121.71	4.060	<0.001

Abbreviations: WBC, white blood cell count; CRP, C-reactive protein; PCT, procalcitonin; IgA, immunoglobulin A; IgG, immunoglobulin G; IgM, immunoglobulin M; LDH, lactate dehydrogenase; SF, ferritin; MPP, Mycoplasma pneumoniae pneumonia; SMPP, Severe mycoplasma pneumoniae pneumonia.

Table 3 Logistic Regression Analysis Results of SMPP Related Influencing Factors

Variable	Regression Coefficient (β)	Standard Error (*SE*)	Wald x^2 value	*P*	OR (95% CI)
Constant	−7.70	1.71	19.00	<0.001	–
Fever time	0.25	0.14	4.74	0.021	1.31(1.03~1.65)
CRP	0.07	0.03	7.88	0.006	1.16(1.02~1.10)
LDH	0.02	0.004	11.47	0.002	1.021(1.01~1.02)
Is it large leafy	2.46	0.51	23.49	0.001	10.38(4.31~31.02)

Abbreviations: CRP, C-reactive protein; LDH, lactate dehydrogenase.

95% CI = 1.02–1.10, $P = 0.006$), LDH (OR = 1.021, 95% CI = 1.01–1.02, $P = 0.002$) and large patchy consolidation (OR = 10.38, 95% CI = 4.31–31.02, $P = 0.001$) were the independent risk factors for SMPP. See Table 3.

Discussion

This study investigated the clinical characteristics of MPP and the independent risk factors of SMPP. There is a lot of discussion about the epidemiological characteristics of MPP. Regarding the onset season, MPP can occur in all seasons of the year, but due to different regions, the epidemic season varies slightly. Onozuka et al[9] found that if the average temperature in Japan increased by 1°C, the number of people infected with MP increased by 16.9%; an increase of 1% in relative humidity resulted in a 4.1% increase in the number of MP infections. A research report in China[10] stated that the

incidence rate of MPP is high in autumn and winter in northern China, while the incidence rate is high in summer and autumn in southern China, which is related to the difference in temperature and humidity in the different latitudes. The incidence rate of MPP in children in this study is mainly in autumn and winter, with 40.9% in autumn and 38.3% in winter, which is consistent with the conclusion of Liu et al.[11] Regarding the age of onset, according to statistics,[12] preschool and school-age children are the peak age for the onset of MPP, and 8–40% of CAP in children aged 3–15 years is caused by MP infection. Other studies[13] have shown that the risk of severe pneumonia in preschool children infected with MP is higher than that in school-age children. It is considered this is related to the low immune function of young children, and the pulmonary infection is more likely to spread and persist. In this study, the incidence of SMPP in preschool children was the highest, and the gender ratio of the two groups was similar, suggesting that SMPP is not significantly associated with gender, which is consistent with previous studies.[14]

The main clinical features of MPP are fever and cough. Fever is usually seen as a moderate to high fever (>38°C), but it may occur as a low fever (37.3–38°C) or even no fever. Fever can occur in almost all children with SMPP, 88.5% of whom have a high fever (>39°C).[15] In the early stages of the disease, children who had a paroxysmal dry cough experienced a gradual aggravation of the cough. Some children may have a whooping cough-like spasmodic cough that can last up to two weeks.

The results of the present study showed that 89.3% of the patients had a fever and a cough. The imaging manifestations of MPP vary.[5] The lesions were interstitial or alveolar inflammations with consolidation of the lobes or segments, pleural effusion or thickening of the pleura, thickening of the bronchial wall, hilar lymphadenopathy and ground-glass opacities. In the MPP group, 65.8% of the children showed patchy, blurry shadows on imaging. In the SMPP group, 69.5% had large patchy consolidation. It is suggested that the duration of fever and imaging findings are related to the severity of the disease. Previous reports[16] have made similar claims: the longer the fever lasts, the more severe the imaging findings and the greater the likelihood of SMPP.

In addition to respiratory and imaging manifestations, MP infection has a variety of extrapulmonary manifestations, among which the incidence of skin and mucosal damage is the highest, followed by liver function damage and nervous system damage.[6] Some children had liver damage, neutropenia and arrhythmia. The reason was related to the immune response caused by MP infection. As MP spreads to various tissues and organs of the body, it induces cytokines through lipoproteins, causing a series of inflammatory reactions. At the same time, MP antigen has the same antigen structure with the heart, liver, kidney, brain and smooth muscle of the human body. When the body is infected with MP, it can produce autoantibodies in the corresponding tissues and form immune complexes to cause cross immunity, resulting in extrapulmonary damage, myocarditis, erythema multiforme, Steven–Johnson syndrome and haemolytic anaemia.[17]

The results of the present study showed that the IgA, IgG and IgM values of children in the SMPP group were higher than those in the MPP group ($P < 0.05$), which is similar to previous research results. In a previous study, the levels of IgM, IgA and IgG in the MPP group with different disease degrees were statistically significant, while they were significantly higher in the SMPP group than in the moderate and mild groups.[18] In the early stage of MP infection, the levels of IgM and IgA increase, followed by an increase in IgG levels, forming autoantibodies and producing pathological immune effects.[19] Research has shown that the dynamic level of IgM can be used to understand the severity and prognosis of children with MPP.[20] When IgG forms immune complexes by binding to MP, this leads to airway angiogenesis and prolongs the course of the MP infection.[21,22] Therefore, detecting the dynamic changes of immunoglobulins and other factors in children with MPP has certain clinical significance for judging their condition and prognosis.[23]

The results of logistic regression analysis in this study showed that CRP (OR = 1.16, $P = 0.006$) and LDH (OR = 1.021, $P= 0.002$) were the independent risk factors for SMPP, which indicates that the higher the CRP and LDH values, the more severe the patient's condition. This is similar to the findings of Seo et al[24] and Su et al.[25] The acute-phase reactive protein CRP is an important component of the body's nonspecific immune function and has immunomodulatory functions.[26] When tissue damage, inflammation, infection or tissue injury occurs, it can rise sharply within a few hours, increasing several-fold or hundreds of times, playing an important protective role in the body's natural immune process, and gradually decreasing and returning to normal levels when the condition improves.[27] During MPP, CRP is positively correlated with the severity of the disease and is suggestive for the determination of disease severity. The key enzyme

LDH in the glycolytic pathway is present in various organs. Even small amounts of lung tissue damage can cause changes in serum LDH concentrations.[28] In particular, when lung tissue undergoes hypoxic necrosis, resulting in increased cell membrane permeability, LDH is released into the bloodstream in increased concentrations.[29] In addition, LDH release into the blood can also be significantly increased when MP infects extra-pulmonary tissue injury, such as myocardial injury. The results of Zheng et al showed a significant increase in serum LDH in children with SMPP.[30]

There are some shortcomings in this study: (1) All the research subjects are limited to our hospital, the experimental subjects are limited by the region, the sample size is not large enough and the conclusion is limited; (2) This study did not discuss D-dimers, nor did it compare in detail the lung symptoms and signs in children with MPP, such as whether there was rale during lung auscultation and the specific nature of the rale; (3) The research subject of this paper is hospitalised children with MPP, and the condition is more serious than that of outpatient children. Therefore, the current conclusion may not have great reference value for non-hospitalised children. Further multi-centre and large sample studies are needed.

Conclusion

In conclusion, MPP is common in school age, and the incidence of MPP is high in autumn and winter. All patients with MPP had a fever and a cough. Patients with SMPP had a high fever, most of the inflammatory indexes were obviously elevated, the lungs showed a large shadow and there was airway mucus embolism. Logistic regression analysis showed that fever duration, CRP, LDH and large patchy consolidation were predictive factors for SMPP, which has important significance for the early recognition of SMPP.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committee of Shanxi Medical University. We obtained signed informed consent from the participants / legal guardians in this study.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Li J, Liu C, Wang X. Clinical characteristics and risk factors for *Mycoplasma pneumoniae* pneumonia in children. *J Tianjin Med Univ*. 2019;25 (04):396–399.
2. Lee KL, Lee CM, Yang TL, et al. Severe *Mycoplasma pneumoniae* pneumonia requiring intensive care in children, 2010–2019. *J Formos Med Assoc*. 2021;120(1 Pt 1):281–291. doi:10.1016/j.jfma.2020.08.018
3. Gao LW, Yin J, Hu YH, et al. The epidemiology of paediatric *Mycoplasma pneumoniae* pneumonia in North China: 2006 to 2016. *Epidemiol Infect*. 2019;147:e192. doi:10.1017/S0950268819000839
4. Liu H, Ma R. Expert consensus on diagnosis and treatment of *Mycoplasma Pneumoniae* pneumonia in children by integrated traditional chinese and western medicine (2017). *Chin J Pract Pediat*. 2017;32(12):881–885. doi:10.19538/j.ek2017120601
5. Tsai TA, Tsai CK, Kuo KC, Yu HR. Rational stepwise approach for *Mycoplasma pneumoniae* pneumonia in children. *J Microbiol Immunol Infect*. 2021;54(4):557–565. doi:10.1016/j.jmii.2020.10.002
6. Yu W, Chen X, Wang H, He L. Advances in imaging studies of severe *Mycoplasma pneumoniae* pneumonia. *Chin Imag J Integr Trad West Med*. 2022;20(05):494–496+500.
7. Expert Committee on Rational Use of Medicines for Children Pharmaceutical Group, National Health and Family Planning Commission. Expert consensus on laboratory diagnostics and clinical practice of *Mycoplasma pneumoniae* infection in children in China (2019). *Chin J Pediatr*. 2020;58 (05):366–373. doi:10.3760/cma.j.cn112140-20200304-00176
8. Lin L, Li C. Interpretation of guideline for management of community acquired pneumonia in children (2013 Revision). *Chi J Obstet & Gynecol Pediatr*. 2014;10(06):728–732. doi:10.3877/cma.j.issn.1673-5250.2014.06.006
9. Onozuka D, Chaves LF. Climate variability and nonstationary dynamics of *Mycoplasma pneumoniae* pneumonia in Japan. *PLoS One*. 2014;9(4): e95447. doi:10.1371/journal.pone.0095447
10. Wang F, Tian Z, Guo W. Epidemiology of *Mycoplasma Pneumoniae* in an area of liaoning province during 2013–2017. *Chin Contin Med Educ*. 2019;11(04):68–70. doi:10.3969/j.issn.1674-9308.2019.04.0031
11. Liu Q, Li H, Wan J, Zhang L. Analysis on epidemiological and clinical characteristics of severe *Mycoplasma* pneumonia in children. *Anhui Med J*. 2017;38(12):1553–1556. doi:10.3969/j.issn.1000-0399.2017.12.014
12. Chen K, Jia R, Li L, Yang C, Shi Y. The aetiology of community associated pneumonia in children in Nanjing, China and aetiological patterns associated with age and season. *BMC Public Health*. 2015;15:113. doi:10.1186/s12889-015-1422-1
13. Lin S, Ou W. Research progress on the relationship between community-acquired respiratory distress syndrome toxins and *Mycoplasma pneumoniae* infection and wheezing. *Chin J Diffic and Compl Cas*. 2020;19(12):1288–1291+1296. doi:10.3969/j.issn1671-6450.2020.12.026

14. Inchley CS, Berg AS, Vahdani Benam A, Kvissel AK, Leegaard TM, Nakstad B. *Mycoplasma Pneumoniae*: a cross-sectional population-based comparison of disease severity in preschool and school-age children. *Pediatr Infect Dis J*. 2017;36(10):930–936. doi:10.1097/INF.0000000000001628
15. Huang Y, Zhang Y. Analysis of related risk factors of severe *Mycoplasma pneumoniae* pneumonia in children. *Chin Clin Dr*. 2018;46(07):863–865. doi:10.3969/j.issn.2095-8552.2018.07.037
16. Cao J, Zheng K, Feng S, Huang L, Hou L. Clinical characteristics and factors related to the severity of symptoms of children with *Mycoplasma* pneumonia. *J Molecul Imag*. 2018;41(03):388–391. doi:10.3969/j.issn.1674-4500.2018.03.25
17. Li J, Luu LDW, Wang X, et al. Metabolomic analysis reveals potential biomarkers and the underlying pathogenesis involved in *Mycoplasma pneumoniae* pneumonia. *Emerg Microbes Infect*. 2022;11(1):593–605. doi:10.1080/22221751.2022.2036582
18. Chen D, Wu P, Liu D, et al. Clinical role of M. pneumoniae typing antibody detected by chemiluminescent immunoassay in the diagnosis of *Mycoplasma pneumoniae* pneumonia in children. *Int Immunopharmacol*. 2022;112:109196. doi:10.1016/j.intimp.2022.109196
19. Chen J, Yin Y, Zhao L, Zhang L, Zhang J, Yuan S. *Mycoplasma pneumoniae* infection prediction model for hospitalized community-acquired pneumonia children. *Pediatr Pulmonol*. 2021;56(12):4020–4028. doi:10.1002/ppul.25665
20. Choo S, Kim SH, Lee E. Clinical significance of *Mycoplasma pneumoniae* specific IgM titer in children hospitalized with Mycoplasma pneumoniae pneumonia. *BMC Infect Dis*. 2022;22(1):470. doi:10.1186/s12879-022-07456-6
21. Ge M, Song Q, Guo Q, et al. Diagnostic Value of Serum ELISA of *Mycoplasma Pneumoniae* IgM and IgG and passive particle agglutination for *Mycoplasma Pneumoniae* Pneumonia in Children. *Clin Lab*. 2021;67(1). doi:10.7754/Clin.Lab.2020.200321
22. Wu L, Ye M, Qin X, Liu Y, Lv Z, Zheng R. Diagnostic value of quantitative MP-IgG for *Mycoplasma pneumoniae* pneumonia in adults. *Clin Chim Acta*. 2020;503:76–83. doi:10.1016/j.cca.2020.01.004
23. Lee E, Young Lee Y. Risk factors for the development of post-infectious bronchiolitis obliterans after *Mycoplasma pneumoniae* pneumonia in the era of increasing macrolide resistance. *Respir Med*. 2020;175:106209. doi:10.1016/j.rmed.2020.106209
24. Seo YH, Kim JS, Seo SC, et al. Predictive value of C-reactive protein in response to macrolides in children with macrolide-resistant *Mycoplasma pneumoniae* pneumonia. *Korean J Pediatr*. 2014;57(4):186–192. doi:10.3345/kjp.2014.57.4.186
25. Su G, Ding L, Zhang Z, Liu X. Effects of different doses of Methylprednisolone on immune function and prognosis in children with refractory *Mycoplasma pneumoniae* pneumonia. *Chin J Front Med Sci*. 2019;11(10):71–74. doi:10.12037/YXQY.2019.10-15
26. Pathak A, Agrawal A. Evolution of C-Reactive Protein. *Front Immunol*. 2019;10:943. doi:10.3389/fimmu.2019.00943
27. State N. CRP and the Prognosis of Patients with Cirrhosis. *Maedica*. 2021;16(3):353–361. doi:10.26574/maedica.2021.16.3.353
28. Claps G, Faouzi S, Quidville V, et al. The multiple roles of LDH in cancer. *Nat Rev Clin Oncol*. 2022;19(12):749–762. doi:10.1038/s41571-022-00686-2
29. Gordon JS, Wood CT, Luc JGY, et al. Clinical implications of LDH isoenzymes in hemolysis and continuous-flow left ventricular assist device-induced thrombosis. *Artif Organs*. 2020;44(3):231–238. doi:10.1111/aor.13565
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