

Impact of body mass index on clinical outcomes in pediatric patients with mycoplasma pneumoniae pneumonia: a retrospective cohort study

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Background: The incidence of mycoplasma pneumoniae pneumonia (MPP) has increased globally, particularly among children and adolescents. Obesity, a major public health challenge, may affect the severity and clinical manifestations of respiratory infections. However, the prognostic role of body mass index (BMI) in MPP remains unclear. This study aimed to investigate the relationship between BMI and clinical outcomes in pediatric patients with MPP.

Methods: This retrospective study included 530 children (aged 2–14 years) hospitalized with MPP at Shaoxing People's Hospital between May 2024 and August 2024. Participants were categorized into obesity, overweight, and normal weight groups based on BMI standards. Inclusion criteria required a confirmed diagnosis of MPP according to the National Health Commission's guidelines. Demographic characteristics, clinical factors such as fever, dyspnea, rash, pleural effusion, plastic bronchitis, and laboratory findings were compared among BMI groups. Prognostic outcomes (including duration of fever, length of hospital stay, incidence of plastic bronchitis, and pleural effusion) were assessed, with severe MPP (SMPP) serving as the primary endpoint. The Pearson correlation coefficient was used to evaluate associations between BMI and clinical variables, while binary logistic regression was performed to analyze the relationship between BMI and SMPP.

Results: Forty-two patients had obesity (7.9%), 45 were overweight (8.5%), and 443 (83.6%) had normal BMI. There were no significant differences in age, gender, and height distribution among three groups. A longer duration of fever (P=0.01), along with a greater incidence of dyspnea (P=0.006), plastic bronchitis (P=0.007), and SMPP (P=0.008) was observed in the obesity and overweight groups compared to the normal weight group. Laboratory results showed elevated white blood cell count (WBC, P<0.001), neutrophil count (N, P<0.001), and C-reactive protein (CRP, P=0.001) levels in the overweight and obesity groups compared to the normal weight group. Additionally, significant increases in red blood cell count (RBC, P=0.03), and alanine aminotransferase (ALT, P=0.006) levels were observed in the obesity group compared to the normal weight group. Correlation analysis revealed that higher BMI was positively associated with the duration of fever (P=0.03) and levels of WBC (P<0.001), N (P<0.001), CRP (P<0.001), RBC (P=0.009), and ALT (P<0.001). Logistic regression analysis demonstrated that higher BMI was associated with an increased risk of SMPP [odds ratio (OR) =1.143, 95% confidence interval (CI): 1.045–1.250]. This association remained significant after adjusting for age and gender (OR =1.120, 95% CI: 1.020–1.231).

Conclusions: This study underscored the critical role of overweight and obesity in exacerbating the

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severity of MPP in pediatric patients. Higher BMI was associated with an increased risk of more severe clinical manifestations, such as prolonged fever, dyspnea and complications like plastic bronchitis. Clinicians should prioritize BMI assessment when managing MPP to improve outcomes and implement targeted preventive strategies for obese patients.

Keywords: Mycoplasma pneumoniae pneumonia (MPP); children; body mass index (BMI); clinical outcomes

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Introduction

In recent years, respiratory infections caused by mycoplasma pneumoniae have increased significantly worldwide, with approximately 10–40% of cases progressing to mycoplasma pneumoniae pneumonia (MPP) (1,2). The most affected age groups are school-age children and adolescents, with an increasing number of reported cases of severe MPP (SMPP), posing significant challenges for healthcare providers (3,4). Although the pathogenesis of MPP is not yet fully elucidated, emerging research suggests that abnormal immune responses in the host are closely associated with the development of MPP, especially in severe cases (5,6). Several prognostic factors have been identified in pediatric MPP, such as erythrocyte sedimentation rate (ESR), lactate

Highlight box

Key findings

 Obese and overweight children with Mycoplasma pneumoniae pneumonia (MPP) had a longer fever duration and a greater incidence of dyspnea, plastic bronchitis, and severe MPP (SMPP). Elevated levels of white blood cell count (WBC), neutrophil count (N), C-reactive protein (CRP), red blood cell count (RBC), and alanine aminotransferase (ALT) were observed in these groups, and a positive correlation was found between body mass index (BMI) and these markers.

What is known and what is new?

- Obesity is known to exacerbate certain respiratory infections.
- The specific impact of BMI on MPP severity in children was previously unclear. This study provides new evidence that higher BMI is associated with worse clinical outcomes in MPP.

What is the implication, and what should change now?

BMI should be assessed in pediatric MPP cases, as overweight and
obesity are significant risk factors for severe disease. Clinicians
should implement BMI-focused strategies to improve management
and prevent complications in obese and overweight children.

dehydrogenase (LDH), interleukin (IL)-6, C-reactive protein (CRP), neutrophil percentage, and neutrophil/lymphocyte ratio (7). Given that inflammation plays a critical role in disease severity, metabolic conditions such as obesity, which are associated with chronic low-grade inflammation, may influence MPP outcomes. Our study aims to further explore the impact of obesity on the clinical manifestations and severity of MPP in children.

Obesity has emerged as a major global health challenge, increasingly affecting children's health by raising risks not only for metabolic disorders but also for greater susceptibility to various infections. A recent study has highlighted the complex interaction between obesity and infection, revealing that obesity is associated with an increased risk of various infections, including surgical site infections, skin infections, periodontal disease, and hospital-acquired infections (8). Furthermore, obesity has been associated with adverse outcomes in diseases such as influenza and the coronavirus disease 2019 (COVID-19), where obese individuals experience more severe disease progression and complications (9,10). However, it is currently unknown if obesity plays a role in the clinical manifestations of MPP in children.

Obesity, a chronic metabolic disease, is associated with sustained low-grade inflammation that can impair the body's immune function (11,12), thus it is possible that the clinical characteristics of MPP may vary among patients with different body mass index (BMI) levels. A strong rationale existed for hypothesizing that a high BMI may influence children's progression of the MPP. However, research exploring the prognostic role of BMI in MPP is limited. This study aimed to explore the clinical characteristics of MPP in children with varying BMI levels, focusing on symptom severity, and specific laboratory markers. Through a retrospective analysis of clinical data from pediatric patients diagnosed with MPP at our hospital, we evaluated

the association between BMI and disease severity, as well as examining the potential impact of obesity on clinical outcomes. By understanding these associations, targeted prevention and treatment strategies can be developed, potentially improving the prognosis for this vulnerable patient population. Furthermore, the study's results could inform public health policies aiming at reducing obesity-related infection risks in pediatric patients. We present this article in accordance with the STROBE reporting checklist (available at https://tp.amegroups.com/article/view/10.21037/tp-2024-494/rc).

Methods

Study design and subjects

This retrospective cohort study analyzed pediatric patients hospitalized with MPP at Shaoxing People's Hospital between May 2024 and August 2024. A total of 530 children aged 2–14 years were enrolled. Ethical approval was obtained from the Ethics Committee of Shaoxing People's Hospital (approval No. 2024-099-01). Requirement for informed consent was waived due to the retrospective nature of the study. The study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments.

Sample size estimation

The required sample size was estimated based on prior studies assessing the impact of BMI on respiratory infections. To achieve 80% statistical power (α =0.05) for detecting differences among BMI groups using ANOVA, an estimated 188 participants per group were required. While the overweight (n=45) and obesity (n=42) groups were smaller than this threshold, the study successfully enrolled a substantial cohort of 530 children, including 443 in the normal-weight group. Despite the estimated power of 55–65%, the study provides valuable preliminary insights into the relationship between BMI and MPP severity. The findings contribute to the growing body of evidence on this topic and highlight the importance of future research with larger, more balanced cohorts to further validate these associations.

Diagnostic criteria

The diagnoses of MPP and SMPP were based on the National Health Commission's Guidelines for Diagnosis and

Treatment of MPP in Children (2023 Edition). Imaging findings, including pleural effusion patterns, were also considered in diagnosis. A recent study has explored the potential of CT-based radiomics to differentiate pleural effusions in bacterial pneumonia and MPP, offering valuable insights into radiological distinctions (13). The diagnosis of MPP included the following criteria: (I) patients presenting with symptoms such as fever, cough, wheezing, dyspnea and other respiratory manifestations; (II) physical examination revealing rales in the lungs or imaging confirming pneumonia; (III) fulfillment of at least one of the following criteria: ≥1 item: (i) single serum MP antibody titer ≥1:160 (PA method) or a four-fold or greater increase in paired serum MP antibody titers (with a 2-week interval); (ii) positive MP-DNA or MP-RNA. The diagnosis of SMPP required meeting any of the following criteria: (I) persistent high fever (≥39 °C) lasting ≥5 days or fever lasting ≥7 days, with no decrease in peak temperature; (II) presence of any of the following symptoms: wheezing, shortness of breath, difficulty breathing, chest pain, or hemoptysis; (III) extrapulmonary complications not meeting the criteria for critical illness; (IV) oxygen saturation (SpO₂) ≤93% at rest while breathing ambient air; (V) imaging findings meeting any of the following: (i) involvement of $\geq 2/3$ of a single lung lobe with uniform, dense consolidation, or involvement of two or more lobes with high-density consolidation (regardless of affected area size), possibly accompanied by moderate to large pleural effusion, or localized bronchiolitis; (ii) diffuse involvement of a single lung or bronchiolitis affecting ≥4/5 of both lungs, possibly accompanied by bronchitis and mucus plugs leading to atelectasis; (VI) progressive worsening of clinical symptoms, with imaging showing a >50% increase in lesion size within 24–48 hours; (VII) significant elevation of any of the following: CRP, LDH, or D-dimer. This study adopted globally recognized BMI standards to define overweight and obesity, with specific thresholds for children aged 2-18 years from Expert consensus on diagnosis, assessment, and management of obesity in Chinese children (14).

Exclusion criteria

- (I) Patients aged under 2 years or over 14 years.
- (II) Children with chronic respiratory infections or other pulmonary diseases.
- (III) Community-acquired pneumonia caused by pathogens other than Mycoplasma pneumoniae, such as adenovirus pneumonia.

(IV) Patients who had recently received immunotherapy or corticosteroid treatment.

Clinical and laboratory measurements

Demographic, clinical, and laboratory data were retrieved from medical records.

- Clinical variables: length of stay, duration of fever, dyspnea, rash, pleural effusion, plastic bronchitis. Plastic bronchitis was defined as airway obstruction due to thick, branching mucus casts.
- ❖ Laboratory tests: blood samples collected on admission were analyzed using standard hospital laboratory protocols and included: routine blood test: white blood cell count (WBC), neutrophil count (N), lymphocyte count (L), red blood cell count (RBC). Inflammatory markers: CRP (measured by immunoturbidimetry), ESR, LDH, fibrinogen (FIB) (measured by Clauss method). Liver and protein markers: total protein (TP), albumin (ALB), alanine aminotransferase (ALT), aspartate aminotransferase (AST). Immunoglobulins (Ig): IgG, IgA, and IgM [measured using enzyme-linked immunosorbent assay (ELISA)].

Prognostic outcomes

The primary outcome was the severity of MPP, classified as either non-SMPP or SMPP. Secondary outcomes included length of stay (recorded from admission to discharge based on physician criteria), duration of fever (defined as the number of days until body temperature normalized), incidence of plastic bronchitis (diagnosed based on bronchoscopic findings), and pleural effusion (determined by CT imaging).

Statistical analysis

The demographic and clinical variables of the subjects were expressed as means \pm standard deviation for continuous variables. Intergroup comparisons were conducted using a variance test, followed by pairwise comparisons with the least significant difference (LSD) method. Categorical data were presented as frequencies and percentages, with intergroup comparisons performed using the χ^2 test. Associations between patients' BMI and other clinical variables were assessed via Pearson's correlation coefficient. All statistical analyses were performed using SPSS Version

24.0 (IBM Corp.), and figures were generated using GraphPad Prism Version 9.0 (GraphPad Software). For all analyses, a two-sided P value of <0.05 was considered statistically significant.

Results

Comparison of demographic and clinical characteristics among different groups based on BMI

A total of 530 patients with MPP were enrolled in this study, comprising 259 boys and 271 girls, with a mean age of 7.1±2.4 years (range: 2 to 14 years). The male-tofemale ratio was 0.96:1. Patients were divided into three groups based on BMI: obesity (42 cases, 7.9%), overweight (45 cases, 8.5%), and normal weight (443 cases, 83.6%). Demographic and clinical characteristics were compared and presented in Table 1. There were no statistically significant differences in age, height, or sex distribution among the three groups (all P>0.05). A longer duration of fever (P=0.01), and a greater incidence of dyspnea (P=0.006), plastic bronchitis (P=0.007), and SMPP (P=0.008) were noted in the obesity and overweight groups compared to the normal weight group. However, no significant differences were observed in the length of hospital stay (P=0.09) or the incidence of rash (P=0.94) and pleural effusion (P=0.11) among the groups.

Comparison of laboratory results among different groups based on BMI

The laboratory results for the MPP patients are detailed in *Table 2* and *Figure 1*. Laboratory analyses revealed that WBC (P<0.001), N (P<0.001), and CRP (P=0.001) levels were significantly elevated in the obesity and overweight groups compared to the normal weight group (all P<0.05). Furthermore, the obesity group showed notably higher RBC (P=0.03) and ALT (P=0.006) levels compared to the normal weight group. However, no significant differences were observed among the three groups in L (P=0.10), ESR (P=0.31), TP (P=0.18), ALB (P=0.10), AST (P=0.93), LDH (P=0.19), FIB (P=0.08), IgG (P=0.74), IgA (P=0.91), or IgM (P=0.17).

Correlation between BMI and different variables

We employed the Pearson's correlation test to analyze the relationship between BMI and various clinical

Table 1 Comparison of demographic and clinical characteristics among different groups based on BMI

Variables Obesity (n=42) Overweight (n=45) Normal (n=443) P value Age (years) 7.0±1.7 7.4±2.8 7.0±2.4 0.60 Gender (male) 27 (64.3) 20 (44.4) 212 (47.8) 0.30 Weight (kg) 33.2±9.6 31.7±14.1 23.2±7.9 <0.001* Height (cm) 125.4±13.4 127.3±20.0 123.3±16.7 0.30 BMI (kg/m²) 20.7±2.6 18.5±2.1 14.9±1.6 <0.001* Length of stay (days) 5.5±1.9 4.9±1.4 4.8±1.2 0.09 Duration of fever (days) 6.0±2.0 6.2±3.1 5.2±2.0 0.01* Dyspnea 6 (14.3) 4 (8.9) 15 (3.4) 0.006* Rash 3 (7.1) 4 (8.9) 39 (8.8) 0.94 Pleural effusion 4 (9.5) 2 (4.4) 13 (2.9) 0.11 Plastic bronchitis 4 (9.5) 5 (11.1) 12 (2.7) 0.007* SMPP 12 (28.5) 10 (22.2) 48 (10.8) 0.008*	1 01		0 0 1		
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Rash 3 (7.1) 4 (8.9) 39 (8.8) 0.94 Pleural effusion 4 (9.5) 2 (4.4) 13 (2.9) 0.11 Plastic bronchitis 4 (9.5) 5 (11.1) 12 (2.7) 0.007*	Duration of fever (days)	6.0±2.0	6.2±3.1	5.2±2.0	0.01*
Pleural effusion 4 (9.5) 2 (4.4) 13 (2.9) 0.11 Plastic bronchitis 4 (9.5) 5 (11.1) 12 (2.7) 0.007*	Dyspnea	6 (14.3)	4 (8.9)	15 (3.4)	0.006*
Plastic bronchitis 4 (9.5) 5 (11.1) 12 (2.7) 0.007*	Rash	3 (7.1)	4 (8.9)	39 (8.8)	0.94
	Pleural effusion	4 (9.5)	2 (4.4)	13 (2.9)	0.11
SMPP 12 (28.5) 10 (22.2) 48 (10.8) 0.008*	Plastic bronchitis	4 (9.5)	5 (11.1)	12 (2.7)	0.007*
	SMPP	12 (28.5)	10 (22.2)	48 (10.8)	0.008*

Data are presented as mean \pm standard deviation or numbers (%). P values are determined by analysis of variance test (continuous data) or χ^2 test (categorical data). *, P<0.05 indicates significant difference. BMI, body mass index; SMPP, severe mycoplasma pneumoniae pneumonia.

Table 2 Comparison of laboratory results among different groups based on BMI

Variables	Obesity (n=42)	Overweight (n=45)	Normal (n=443)	P value
WBC (×10 ⁹ /L)	9.2±3.9	8.3±2.3	6.7±2.1	<0.001*
N (×10 ⁹ /L)	5.9±2.9	5.4±1.8	4.1±1.7	<0.001*
L (×10 ⁹ /L)	2.5±1.6	2.1±0.8	2.0±0.8	0.10
RBC (×10 ¹² /L)	4.7±0.3	4.6±0.3	4.5±0.4	0.03*
CRP (mg/L)	26.5±22.1	26.4±27	15.4±13.5	0.001*
ESR (mm/h)	36.4±18.1	32.7±14.7	32.3±15.1	0.31
TP (g/L)	70.4±4.2	69.9±3.9	69.3±3.9	0.18
ALB (g/L)	41.6±2.3	41.3±2.8	40.8±2.5	0.10
ALT (U/L)	21.5±14.2	25±36.0	15±10.8	0.006*
AST (U/L)	34.1±10.4	32.9±17.8	33.2±14.9	0.93
LDH (U/L)	308.6±64.3	282.9±52.5	297.4±67.8	0.19
FIB (g/L)	4.4±0.8	4.4±0.8	4.2±0.7	0.08
IgG (g/L)	9.6±2.1	9.3±1.7	9.4±2.1	0.74
IgA (g/L)	1.4±0.7	1.5±0.6	1.5±0.6	0.91
IgM (g/L)	1.2±0.4	1.4±0.5	1.4±0.5	0.17

Data are presented as mean ± standard deviation. P values are determined by analysis of variance test. *, P<0.05 indicates significant difference. ALB, albumin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; FIB, fibrinogen; IgG, immunoglobulin G; IgA, immunoglobulin A; IgM, immunoglobulin M; L, lymphocyte count; LDH, lactate dehydrogenase; N, neutrophil count; TP, total protein; RBC, red blood cell count; WBC, white blood cell count.

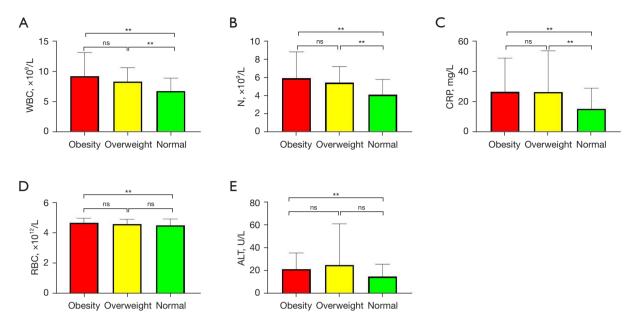


Figure 1 Comparative analysis of laboratory results in MPP patients among different groups based on BMI. (A) Comparison of the peripheral WBC between groups. (B) Comparison of N between groups. (C) Comparison of CRP between groups. (D) Comparison of RBC between groups. (E) Comparison of ALT between groups. Statistical analyses were performed using the Least Significant Difference (LSD) method. **, P<0.05 denotes statistical significance. "ns" denotes no significant difference, P≥0.05. ALT, alanine aminotransferase; BMI, body mass index; CRP, C-reactive protein; MPP, mycoplasma pneumonia; N, neutrophil count; RBC, red blood cell count; WBC, white blood cell count.

and laboratory variables. As shown in *Figure 2*, BMI demonstrated positive correlations with the duration of fever (P=0.03) and levels of WBC (P<0.001), N (P<0.001), CRP (P<0.001), RBC (P=0.009), and ALT (P<0.001).

Correlation analysis between BMI and clinical classification

We performed binary logistic regression analysis to assess the impact of BMI on clinical classification (non-SMPP vs. SMPP). As shown in *Table 3*, a higher BMI was associated with a more severe clinical classification before adjustment [odds ratio (OR) =1.143, 95% confidence interval (CI): 1.045–1.250], and this association remained significant after adjusting for age and gender (OR =1.120, 95% CI: 1.020–1.231), suggesting that BMI is an independent risk factor for disease severity.

Discussion

Globally, childhood obesity has become a major public health concern, especially given its association with increased severity of respiratory infectious diseases (15,16). Our results demonstrated that children with MPP in the

obesity and overweight groups had a longer duration of fever, and a higher incidence of dyspnea, plastic bronchitis, and SMMP compared to their normal-weight peers. Furthermore, significantly elevated levels of inflammatory markers, including WBC, N, and CRP were observed in both the obesity and overweight groups, with these markers showing a positive correlation with BMI. These markers, which are commonly associated with heightened inflammatory responses, suggest that overweight or obesity exacerbates the inflammatory process in MPP. Our findings confirm that childhood obesity and overweight are critical risk factors for the severity of MPP, supporting our initial hypothesis. Previous studies on obesity and pneumonia have reported similar findings. For instance, a large prospective study involving hospitalized patients with community-acquired pneumonia found that overweight or obese children had worse outcomes compared to their normal-weight counterparts, including higher rates of ICU admission, increased need for mechanical ventilation, and longer hospital stays (17). Clinical studies on COVID-19 pneumonia also demonstrated significantly higher WBC, N, CRP levels, and mortality rates in obese patients compared to their normal-weight counterparts, emphasizing the

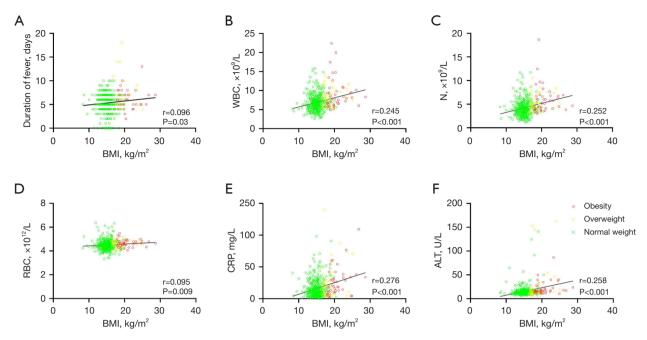


Figure 2 Correlation analysis between BMI and other clinical variables in MPP patients. (A) Correlation between duration of fever and BMI (r=0.096, P=0.03). (B) Correlation between WBC and BMI (r=0.245, P<0.001). (C) Correlation between N and BMI (r=0.252, P<0.001). (D) Correlation between RBC and BMI (r=0.095, P=0.009). (E) Correlation between CRP and BMI (r=0.276, P<0.001). (F) Correlation between ALT and BMI (r=0.258, P<0.001). ALT, alanine aminotransferase; BMI, body mass index; CRP, C-reactive protein; MPP, mycoplasma pneumonia; N, neutrophil count; RBC, red blood cell count; WBC, white blood cell count.

Table 3 Correlation analysis of BMI and clinical classification

Model	OR (95% CI)	P value
1	1.143 (1.045, 1.250)	0.003
2	1.120 (1.020, 1.231)	0.02

Model 1: unadjusted; Model 2: adjusted for age and gender. BMI, body mass index; CI, confidence interval; OR, odds ratio.

critical role of high BMI as a significant risk factor for adverse outcomes in respiratory infections (18-22). In this study, we found that an increased BMI was associated with enhanced inflammatory responses and poorer clinical outcomes, which was consistent with existing literature. These findings indicate that in the management of MPP, it is essential to recognize obesity and overweight as risk factors for disease exacerbation.

Although obesity and overweight pose potential risks to the progression of MPP, their exact roles in disease development remains unclear. However, previous studies have suggested that multiple factors contributed to this association. The pathogenesis of MPP is currently believed to involve two primary mechanisms: direct microbial

damage and excessive activation of the host immune system (5). Excessive immune activation can lead to an overproduction of cytokines, resulting in a cytokine storm, which plays a critical role in the progression of MPP from mild to severe disease (4). Fang et al. (23) conducted a retrospective study involving 96 children with MPP and found that abnormal expression of cytokines such as tumor necrosis factor (TNF)-α, IL-2, IL-6, and interferon (IFN)-γ, correlated positively with disease severity. Additionally, recent meta-analysis findings indicate that IL-17 levels are significantly elevated in children with SMPP, suggesting its potential as a biomarker for disease progression and severity assessment (24). Obesity is characterized by chronic low-grade inflammation, oxidative stress, and alterations in various hormone levels, resulting in immune system dysfunction. A previous study has shown that, due to inherent immune dysfunction, obese individuals are more prone to excessive immune activation during immune responses, which may trigger a cytokine storm (25). Therefore, obesity may contribute to MPP progression by altering immune system function, promoting proinflammatory cytokine release, increasing systemic inflammation, thereby influencing both the severity and course of MPP.

Moreover, immune dysfunction associated with obesity plays a significant role in adipose tissue inflammation, which is characterized by excessive fat accumulation and dysregulated secretion of adipokines (26). Leptin, the most well-studied adipokine, is typically elevated in obese individuals. Ubags et al. (27) reported a positive correlation between plasma leptin levels and the risk of respiratory infections in mouse models of obesity and pneumonia. Elevated plasma leptin levels have been shown to impair immune responses, which correlates with increased pneumonia severity (27). Additionally, leptin has been implicated in the activation of signal transduction pathways. During pneumonia, leptin stimulates the inflammatory pathway, including STAT3. Compared with normal-weight pneumonia mice, obese pneumonia mice exhibit higher leptin levels, greater lung STAT3 activation, and elevated inflammatory markers such as serum IL-6 and TNF-α, along with increased lung neutrophil infiltration (28). Conversely, adiponectin is another important adipokine that plays a protective role in preventing airway smooth muscle hypertrophy, airway hyperreactivity, and peribronchial inflammation (29). Research involving adipokines and human bronchial epithelial cells has shown that adiponectin can modulate the inflammatory response of these cells (30). However, significantly reduced adiponectin concentrations in obese individuals may increase their susceptibility to pulmonary infections and exacerbate the severity of these diseases (30). These findings suggest that specific adipokines may play critical roles in the pathogenesis and progression of MPP.

Furthermore, obese individuals often have fat deposits around the chest, which restricts diaphragm and chest wall movement, thereby reducing lung compliance (31). Combined with airway narrowing and increased respiratory resistance, this exacerbates dyspnea in pneumonia patients (32). Additionally, obesity increases the likelihood of comorbidities such as obstructive sleep apnea, further compromising pulmonary function and raising the risk of cardiovascular events due to chronic hypoxia (33). Notably, severe obesity, even in children, can cause hemodynamic changes, leading to cardiac remodeling and eventual heart dysfunction (34). Given that obese patients may experience chronic pulmonary and cardiac impairment, they are more likely to suffer from decompensation during MPP, which can lead to respiratory failure and heart failure, further complicating their clinical presentation. Our study also found that obese

patients had higher rates of dyspnea and severe pneumonia compared to their normal-weight counterparts.

Our findings have important implications for clinical practice. As overweight and obesity are key risk factors for the severity of MPP, clinicians should prioritize BMI assessment when evaluating patient risk and formulating treatment strategies. During peak MPP seasons, preventive measures should specifically target overweight and obese populations to reduce infection risks.

However, there are several limitations in this study. It relied solely on BMI to assess overweight and obesity, without considering other important measures such as waist circumference, waist-to-hip ratio, and visceral fat distribution. Additionally, the retrospective, single-center design may limit the generalizability of our findings. Therefore, further multi-center, large-sample studies are needed to validate these results and gain a deeper understanding of the mechanisms linking overweight and obesity to the severity of MPP.

Conclusions

This study underscored the critical role of overweight and obesity in exacerbating the severity of MPP in pediatric patients. Higher BMI was associated with an increased risk of more severe clinical manifestations, such as prolonged fever, dyspnea and complications like plastic bronchitis. Clinicians should prioritize BMI assessment when managing MPP to improve patient outcomes and implement targeted preventive strategies for obese patients. Future research should further investigate the complex relationship between childhood obesity and respiratory infections, employing larger sample sizes and multi-center designs to validate these findings and refine management strategies for this vulnerable population.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Ethical approval was obtained from the Ethics Committee of Shaoxing People's Hospital (approval No. 2024-099-01). Requirement for informed consent was waived due to the retrospective nature of the study. The study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments.

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References

- Kutty PK, Jain S, Taylor TH, et al. Mycoplasma pneumoniae Among Children Hospitalized With Community-acquired Pneumonia. Clin Infect Dis 2019;68:5-12.
- Choi YJ, Jeon JH, Oh JW. Critical combination of initial markers for predicting refractory Mycoplasma pneumoniae pneumonia in children: a case control study. Respir Res 2019;20:193.
- Tan J, Chen Y, Lu J, et al. Pathogen distribution and infection patterns in pediatric severe pneumonia: A targeted next-generation sequencing study. Clin Chim Acta 2025;565:119985.

- Han Q, Jiang T, Wang T, et al. Clinical value of monitoring cytokine levels for assessing the severity of mycoplasma pneumoniae pneumonia in children. Am J Transl Res 2024;16:3964-77.
- 5. Zhu Y, Luo Y, Li L, et al. Immune response plays a role in Mycoplasma pneumoniae pneumonia. Front Immunol 2023;14:1189647.
- 6. Jiang C, Bao S, Shen W, et al. Predictive value of immunerelated parameters in severe Mycoplasma pneumoniae pneumonia in children. Transl Pediatr 2024;13:1521-8.
- 7. Liu F, Chen L, Wang MY, et al. Exploring high-risk factors for the prediction of severe mycoplasma pneumonia in children. Transl Pediatr 2024;13:2003-11.
- 8. Muscogiuri G, Pugliese G, Laudisio D, et al. The impact of obesity on immune response to infection: Plausible mechanisms and outcomes. Obes Rev 2021;22:e13216.
- 9. Peters SAE, MacMahon S, Woodward M. Obesity as a risk factor for COVID-19 mortality in women and men in the UK biobank: Comparisons with influenza/pneumonia and coronary heart disease. Diabetes Obes Metab 2021;23:258-62.
- Noye EC, Bekkering S, Sng JDJ, et al. Obesity Is a Risk Factor for Severe Influenza Virus Infection and COVID-19 in Children. J Pediatric Infect Dis Soc 2025;14:piae123.
- Heymsfield SB, Wadden TA. Mechanisms, Pathophysiology, and Management of Obesity. N Engl J Med 2017;376:1492.
- 12. Fang X, Henao-Mejia J, Henrickson SE. Obesity and immune status in children. Curr Opin Pediatr 2020;32:805-15.
- 13. Li J, Si J, Yang Y, et al. The value of CT-based radiomics for differentiation of pleural effusions in bacterial pneumonia and Mycoplasma pneumoniae pneumonia in children. Transl Pediatr 2025;14:70-9.
- 14. Subspecialty Group of Endocrinologic, Hereditary and Metabolic Diseases, the Society of Pediatrics, Chinese Medical Association; Subspecialty Group of Child Health Care, the Society of Pediatrics, Chinese Medical Association; Subspecialty Group of Clinical Nutrition, the Society of Pediarics, Chinese Medical Association; et al. Expert consensus on diagnosis, assessment, and management of obesity in Chinese children. Zhonghua Er Ke Za Zhi 2022;60:507-15.
- 15. Moser JS, Galindo-Fraga A, Ortiz-Hernández AA, et al. Underweight, overweight, and obesity as independent risk factors for hospitalization in adults and children from influenza and other respiratory viruses. Influenza Other

- Respir Viruses 2019;13:3-9.
- Yang W, Yang Y, Guo Y, et al. Obesity and risk for respiratory diseases: a Mendelian randomization study. Front Endocrinol (Lausanne) 2023;14:1197730.
- 17. Bramley AM, Reed C, Finelli L, et al. Relationship Between Body Mass Index and Outcomes Among Hospitalized Patients With Community-Acquired Pneumonia. J Infect Dis 2017;215:1873-82.
- Zhang J, Xu Y, Shen B, et al. The Association between Obesity and Severity in Patients with Coronavirus Disease 2019: a Retrospective, Single-center Study, Wuhan. Int J Med Sci 2021;18:1768-77.
- 19. Bettini S, Bucca G, Sensi C, et al. Higher Levels of C-Reactive Protein and Ferritin in Patients with Overweight and Obesity and SARS-CoV-2-Related Pneumonia. Obes Facts 2021;14:543-9.
- Kalligeros M, Shehadeh F, Mylona EK, et al. Association of Obesity with Disease Severity Among Patients with Coronavirus Disease 2019. Obesity (Silver Spring) 2020;28:1200-4.
- Chetboun M, Raverdy V, Labreuche J, et al. BMI and pneumonia outcomes in critically ill covid-19 patients: An international multicenter study. Obesity (Silver Spring) 2021;29:1477-86.
- 22. Malik VS, Ravindra K, Attri SV, et al. Higher body mass index is an important risk factor in COVID-19 patients: a systematic review and meta-analysis. Environ Sci Pollut Res Int 2020;27:42115-23.
- 23. Deng F, Cao H, Liang X, et al. Analysis of cytokine levels, cytological findings, and MP-DNA level in bronchoalveolar lavage fluid of children with Mycoplasma pneumoniae pneumonia. Immun Inflamm Dis 2023;11:e849.
- 24. Leerach N, Sitthisak S, Kitti T, et al. Association of serum interleukin-17 level and Mycoplasma pneumoniae pneumonia in children: a systematic review and meta-

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- analysis. Transl Pediatr 2024;13:1588-99.
- 25. Chen J, Wei H. Immune Intervention in Sepsis. Front Pharmacol 2021;12:718089.
- 26. Fischer-Posovszky P, Möller P. The immune system of adipose tissue: obesity-associated inflammation. Der Pathologe. 2020;41:224-9.
- Ubags ND, Stapleton RD, Vernooy JH, et al.
 Hyperleptinemia is associated with impaired pulmonary host defense. JCI Insight 2016;1:e82101.
- 28. Bodilly L, Williamson L, Howell K, et al. Obese mice with pneumonia have hyperleptinemia and increased pulmonary signal transducer and activator of transcription 3 activation. Shock 2023;59:409-16.
- Zhu L, Chen X, Chong L, et al. Adiponectin alleviates exacerbation of airway inflammation and oxidative stress in obesity-related asthma mice partly through AMPK signaling pathway. Int Immunopharmacol 2019;67:396-407.
- Salvator H, Grassin-Delyle S, Naline E, et al. Contrasting Effects of Adipokines on the Cytokine Production by Primary Human Bronchial Epithelial Cells: Inhibitory Effects of Adiponectin. Front Pharmacol 2020;11:56.
- 31. Cesanelli L, Cesanelli F, Degens H, et al. Obesity-related reduced spirometry and altered breathing pattern are associated with mechanical disadvantage of the diaphragm. Respir Physiol Neurobiol 2024;325:104267.
- 32. Dupuis A, Thierry A, Perotin JM, et al. Obesity Impact on Dyspnea in COPD Patients. Int J Chron Obstruct Pulmon Dis 2024;19:1695-706.
- 33. Feher M, Hinton W, Munro N, et al. Obstructive sleep apnoea in Type 2 diabetes mellitus: increased risk for overweight as well as obese people included in a national primary care database analysis. Diabet Med 2019;36:1304-11.
- 34. Wang H, Min J, Zhong L, et al. Life-course obesity and heart failure: a two-sample Mendelian randomization study. Intern Emerg Med 2025;20:171-80.