Pediatr Pol 2025; 100 (3): 281-285
DOI: HTTPS://DOI.ORG/10.5114/POLP.2025.154302
Submitted: 08.04.2025, Accepted: 22.06.2025, Published: 24.09.2025

PEDIATRIA Polska
PISMO POLSKIEGO TOWARZYSTWA PEDIATRYCZNEGO
Polish Journal of PAEDIATRICS
OFFICIAL JOURNAL OF POLISH SOCIETY OF PAEDIATRICS

CASE REPORT

Different clinical manifestations of *Mycoplasma pneumoniae* infection in children: a case series

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ABSTRACT

Among bacterial diseases occurring in children, *Mycoplasma pneumoniae* infection remains a diagnostic challenge due to different clinical presentation. Its most common manifestation is atypical pneumonia, with a mild course and persistent dry cough. Detection of the pathogen is based on immunological methods and polymerase chain reaction (PCR) testing; chest X-ray may also be helpful. Infection can lead to severe complications in many organs, especially in children and elderly individuals. Non-obvious laboratory and imaging findings, along with the increasing resistance of bacteria to antibiotics, make it increasingly challenging to treat effectively and quickly. This work aimed to report several cases of *M. pneumoniae* infection in pediatric patients, who developed myocarditis, meningitis, and Stevens-Johnson syndrome in the course of or as a result of the disease.

KEY WORDS:

myocarditis, atypical pneumonia, *Mycoplasma pneumoniae*, Stevens-Johnson syndrome.

INTRODUCTION

Mycoplasma pneumoniae, a small Gram-negative bacterium, is the most common cause of atypical pneumonia in children, accounting for up to 40% of out-of-hospital cases [1]. Infections peak in autumn and winter, but occur year-round, especially in crowded settings. Transmission is via droplets, with an incubation period of one to three weeks [2]. The primary presentation is atypical pneumonia, though extra-pulmonary complications may occur, notably affecting the nervous (e.g., meningitis, myelitis), cardiovascular (e.g., myocarditis), lymphatic, muscular, and joint systems [3]. This manuscript described four pediatric cases, one with pulmonary and three with extra-pulmonary involvements.

CASE DESCRIPTIONS

CASE 1

A previously healthy 16-year-old male, engaged in sports, and without chronic conditions or regular medications, was admitted to the pediatric department due to a prolonged respiratory tract infection unresponsive to outpatient treatment. His history included a fever up to 39.7°C, with only moderate response to antipyretics, and respiratory symptoms beginning two weeks prior. Initial lab results showed elevated inflammatory markers (C-reactive protein [CRP]: 109.33 mg/l; N < 5 mg/l) and mild thrombocytopenia (platelet count [PLT]: $158 \times 10^3/\mu l$). He was started on amoxicillin-clavulanic

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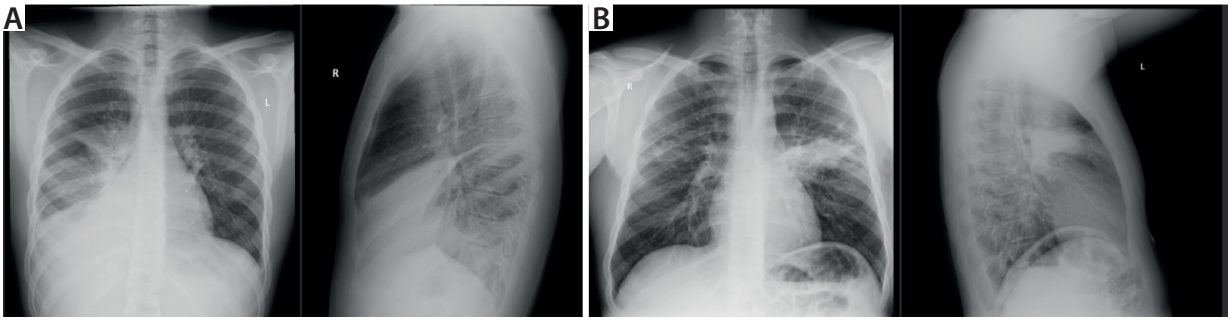

FIGURE 1. Patients' chest X-rays. Case 1 showing features of lobar pneumonia (**A**), and case 2 revealing atelectasis-inflammatory changes in the left lung (**B**)

acid and symptomatic therapy. Despite a decrease in CRP (63.45 mg/l), his fever persisted, and clinical status worsened. A third-generation cephalosporin was introduced without significant improvement. New-onset right subcostal pain led to referral for further evaluation.

On admission, he was febrile, in average general condition, with impaired respiratory function but no increased respiratory effort. Vital signs included SpO_2 of 94%, bood pressure (BP) of 110/80 mmHg, and heart rate (HR) of 100 bpm. Physical examination revealed diminished breath sounds, right-sided basal crackles, and severe right subcostal tenderness.

Laboratory tests showed markedly elevated CRP (153,13 mg/l), normal procalcitonin (PCT) (0.209 ng/ml; N < 0.50 ng/ml), neutrophilia (8.11×10^3/µl), elevated D-dimer (18,379 ng/ml; N < 500 ng/ml), and a slightly increased International Normalized Ratio (INR) (1.27; N: 0.8–1.2). Blood cultures were negative.

Chest X-ray revealed confluent maculopapular and dehiscent inflammatory or atelectatic lesions in the right middle and lower lung fields, obliteration of the right diaphragmatic angle, and pleural effusion (Figure 1A). While awaiting microbiological results, third-generation cephalosporin therapy was continued, but later switched to a glycopeptide due to suspected abscess formation.

Following a drop in oxygen saturation to 92% and lack of clinical improvement, a chest computed tomography (CT) revealed extensive parenchymal consolidation with air bronchograms in the right lung, consistent with lobar pneumonia involving the entire middle lobe and most of the lower lobe, sparing segment VIII. Minor subpleural consolidations were noted in the left lung, with a small right pleural effusion (10 mm) present. Polymerase chain reaction (PCR) confirmed *M. pneumoniae*, with no other common bacterial pathogens. Immunoglobulin M (IgM) titers were significantly elevated (> 200 U/ml).

Clarithromycin was initiated, with vancomycin initially continued, and then replaced with a third-generation cephalosporin. The patient's condition progressively improved, with resolution of right-sided crackles, reduced fatigue, and lung ultrasound showing regression of atelectasis and pleural effusion. Inflammatory markers declined (CRP: 10.16 mg/l; D-dimer: 3,635 ng/ml). Follow-up chest X-ray demonstrated partial resolution of right lung opacities and decreased hilar enlargement.

The patient was discharged with a 21-day clarithromycin regimen. Two-week follow-up ultrasound showed a reduced consolidation (2 × 1.8 cm), with complete resolution after four weeks.

CASE 2

A 17-year-old boy was brought to the hospital by emergency medical services (EMS) due to a 20-minute episode of compressive retrosternal chest pain (6/10 NRS), which resolved spontaneously without medication or loss of consciousness. The pain was non-respiratory-related, with no dyspnea or reduced exercise tolerance. At home, BP was 87/37 mmHg and HR 80/min. EMS recorded BP of 120/80 mmHg, HR of 87/min, and electrocardiogram (ECG) showed ST elevation in II, III, and aVF leads. He had been on a first-generation cephalosporin for pharyngitis with fever.

The patient had no chronic conditions and was fully vaccinated. On admission, he appeared well but had a dry paroxysmal cough, a 2/6 systolic murmur, and mild crackles in the left lung. Lab results showed elevated inflammatory markers, creatine kinase isoenzyme MB (CK-MB) (14 ng/µl, N < 6.22 ng/µl), and troponin (272 µg/l, N < 14.0 ng/µl), with normal pro-B-type natriuretic peptide (pro-BNP) (61.2 ng/l, N < 486 ng/l). Chest X-ray revealed atelectasis-inflammatory changes in the left lung (segment 5) (Figure 1B). Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), influenza, and respiratory syncytial virus (RSV) were ruled out. He was started on symptomatic treatment and amoxicillin-clavulanic acid.

The next day, CK-MB rose to 19.05 ng/µl and troponin to 617 µg/l. Echocardiography demonstrated slightly reduced left ventricular function (ejection fraction [EF]: 60%). Repeat ECG showed persistent ST elevation and tachycardia (HR: 100/min). PCR from sputum identified *M. pneumoniae*, prompting addition of clarithromycin and a third-generation cephalosporin due to rising inflammation.

The patient improved clinically; fever resolved by day three. Cardiac enzymes and inflammatory markers nor-

malized. Cardiology consultation found no echo abnormalities, and ECG showed a negative T wave in lead III and repolarization changes in the left precordial leads. He was discharged in good condition.

One week later, lung ultrasound showed significant resolution of inflammatory changes. Cardiac magnetic resonance imaging confirmed acute active myocarditis per 2018 Lake Louise criteria [4], with post-inflammatory damage in the basal and mid-inferior/inferolateral segments of the left ventricle, but preserved right ventricular function and borderline normal left ventricular systolic function.

No pharmacologic cardiac support was needed. Holter monitoring was normal. He was discharged with lifestyle recommendations, BP monitoring, and a cardiology follow-up in 3–4 weeks.

CASE 3

A 14-year-old boy was admitted due to severe dehydration, general deterioration, and behavioral changes. He had a week-long cough and, on the day of admission, experienced severe vomiting and headache without fever, unresponsive to paracetamol. His history included allergies to grasses and mites, and full vaccination per schedule.

On admission, he appeared pale and dehydrated, with two-finger neck stiffness. Despite normal blood work, electrolytes, and negative antigen tests (influenza A/B, RSV, SARS-CoV-2), a head CT was unremarkable. Cerebrospinal fluid (CSF) analysis showed mild pleocytosis (26 cells/μl) and elevated protein (69 mg/dl), with PCR negative for common pathogens. Acyclovir was initiated for suspected viral meningitis.

Due to persistent cough without auscultatory findings, lung ultrasound was performed revealing left-sided consolidation consistent with pneumonia. Serology confirmed *M. pneumoniae* infection (IgM and IgG > 200 U/ml [> 25 positive]), indicating mycoplasmal pneumonia with extra-pulmonary meningitis. Clarithromycin was started, but later switched to doxycycline after an infectious disease consultation.

The patient's condition steadily improved, with follow-up ultrasound confirming regression of lung inflammation.

CASE 4

A 7-year-old boy was admitted with extensive stomatitis, conjunctivitis, and a persistent fever up to 39°C lasting for two days despite symptomatic treatment. Initially managed as an outpatient, he showed temporary improvement before symptom recurrence, with a dry cough, chest pain during swallowing and coughing, worsening oral lesions, and new purulent and blistering eruptions on the upper lip. Amoxicillin-clavulanic acid and choline salicylate gel were added, but symptom progression, including a productive cough with blood-streaked sputum, prompted hospital referral.

The child had no chronic illnesses, allergies, or ongoing medications, and was fully vaccinated. His history included erythema nodosum. On admission, he was febrile, mildly dehydrated, and weak, with conjunctival injection and painful mucocutaneous lesions. Oral examination revealed edema, vesicles, aphthae, and necrotic lesions involving the lips, palate, and right cheek. Multiple small vesicles on erythematous bases were present on the shoulder, scapula, clavicle, and right foot.

Lab results showed elevated inflammatory markers and D-dimers. Chest X-ray indicated atypical pneumonia, and Stevens-Johnson syndrome was diagnosed based on clinical presentation. Microbiological tests excluded Epstein-Barr virus (EBV), but serology showed strongly positive *M. pneumoniae* IgM and negative IgG.

Empirical broad-spectrum antibiotics were started, and then adjusted to clarithromycin following microbiological confirmation. The patient also received IV immunoglobulin, corticosteroid pulses, and topical treatments [5]. Ophthalmology and ENT consultations led to the addition of antibiotic and steroid eye drops [6]. Fever resolved the next day, and skin lesions stabilized, with only a few minor new lesions.

On day five, a urethral ulcer appeared, with intermittent pain on urination; topical treatment was applied. The patient's condition gradually improved, with healing of mucosal and skin lesions.

DISCUSSION

Pediatric cases of *M. pneumoniae* infection show considerable variability in symptom progression. Although common features include persistent cough, poor general condition, and fever unresponsive to initial treatment, diagnostic complexity arises from heterogeneous and sometimes extra-pulmonary symptoms. Accurate diagnosis and appropriate pharmacotherapy are essential, particularly in severe pediatric cases [7].

M. pneumoniae circulates year-round globally, with epidemics in Europe every 1–3 years due to decreasing immunity in general population and bacterial variation [8]. COVID-19-related non-pharmacological measures temporarily reduced its transmission [9]; however, a reduced exposure and resumption of group childcare settings have increased susceptibility and disease severity, including extra-pulmonary complications. The rising antibiotic resistance highlights the need for improved clinical recognition and disease management.

Pathogenesis involves ciliary dysfunction and epithelial damage from bacterial hydrogen peroxide and dismutase production [1]. Autoimmune phenomena may result from molecular mimicry between bacterial glycolipids and host antigens [1]. The infection typically presents as atypical pneumonia, most commonly in children

TABLE 1. Antibiotics most commonly used in *Mycoplasma pneumoniae* infections

Antibiotics	Macrolides	Tetracyclines	Quinolones
	Azithromycin, clarithromycin, roxithromycin	Doxycycline (high safety), minocycline (strong effect)	Levofloxacin, ciprofloxacin, moxifloxacin
Use	Preferred group of antibiotics In mild *M. pneumonia*	Alternative treatment In the absence of response to macrolides Treatment of infections with macrolide-resistant strains (MRMP) Severe and untreatable pneumonia	Treatment of infections caused by MRMP strains
Advantages	Strong trans-tissue penetration Fewer gastrointestinal side effects	Effective in infections with MRMP strains	Very effective, but not recommended for children due to the risk of cartilage and tendon damage Negligible resistance of *M. pneumoniae* to antibiotics of this group
Recommended dose and duration of treatment	10 mg/kg once daily *p.o./i.v.* for 3 days	First dose, 4 mg/kg (max. 200 mg), then 2 mg/kg after 12, twice a day for 10 days	Levofloxacin: • children 6 months – 5 years old: 8–10 mg/kg twice a day • 5–16 years old: 8–10 mg/kg once a day • > 16 years old: 500 mg/d (max. 750 mg/d) for 7–14 days Moxifloxacin: • 10 mg/kg/d for 7–14 days
Disadvantages	Resistance of *M. pneumoniae* to antibiotics of this group is steadily increasing	Tetracyclines should not be given to children < 8 years old	Not recommended for children < 18 years old

aged 5–15 years, but can affect all ages [10]. Prodromal symptoms include rhinitis, fever, headache, malaise, and sometimes otalgia, hoarseness, and myalgia. A severe dry cough is characteristic, potentially progressing to dyspnea and pertussis-like symptoms [11].

Extra-pulmonary manifestations may result from direct infection, immune, or vascular responses, including [3, 12]:

- neurological: meningitis, encephalitis, optic neuritis, Guillain-Barré syndrome,
- cardiovascular: myocarditis, pericarditis, arrhythmias, thrombosis,
- dermatological: erythema nodosum, leukocytoclastic vasculitis, Stevens-Johnson syndrome, Kawasaki disease, etc.,
- renal: acute tubular necrosis, glomerulonephritis, interstitial nephritis,
- gastrointestinal: abdominal pain, nausea, diarrhea, pancreatitis,
- hematological: hemolytic anemia due to antibody cross-reaction,
- musculoskeletal: myalgia and arthralgia.

Furthermore, *M. pneumoniae* frequently co-infects with viruses (e.g., adenovirus, RSV, influenza) and bacteria (e.g., *Streptococcus pneumoniae*, *Haemophilus influenzae*) [13]. Adenoviral co-infection is associated with wheezing and increased extra-pulmonary complications [14], whereas bacterial co-infections contribute to more severe disease and complications, such as otitis media and meningitis. Timely identification and combination of antimicrobial therapy improve outcomes [13, 14].

Diagnosis primarily relies on immunoassays detecting IgM, IgG, and IgA antibodies; PCR is increasingly favored for its high sensitivity and specificity (80–100%) [15]. IgM typically appears within a week of symptom onset, peaking in the third week. A fourfold rise in IgG or elevated IgM titers confirm infection. However, serology has limitations, including delayed or absent responses, particularly in immunocompromised children and prolonged IgG persistence [16].

Cold agglutinins are elevated in over half of infected patients, while leukocyte counts are usually normal. Chest X-rays often reveal non-specific unilateral bronchial lesions, primarily in the lower and middle lung zones. Exudative pleuritis and mediastinal lymphadenopathy are rare.

Although often self-limiting, delayed antibiotic treatment may lead to severe extra-pulmonary complications [15]. Due to the absence of a cell wall, *M. pneumoniae* is resistant to β-lactams, glycopeptides, and fosfomycin [17]. Macrolides are first-line therapy; if ineffective, tosufloxacin or tetracyclines are alternatives, but tetracyclines are contraindicated in children under the age of 8 [18]. Table 1 lists commonly used antibiotics [16].

The overuse of macrolides, especially in mild or unconfirmed *M. pneumoniae* cases, drives resistance and should be avoided, as most such infections in children are self-limiting and do not require antibiotics [19]. Despite growing resistance, macrolides are still widely used, often alongside broad-spectrum antibiotics in pediatric pneumonia patients [20]. Stewardship efforts should fo-

cus on reserving macrolides for confirmed or severe infections [19, 21]. The emergence of macrolide-resistant *M. pneumoniae* (MRMP), especially in children, complicates treatment and prolongs fever due to immune-mediated inflammation [22]. In resistant cases, corticosteroids may be used [18]. For severe extra-pulmonary manifestations (e.g., CNS involvement, mucocutaneous lesions, hematologic complications), intravenous immunoglobulin G (IVIG) at 1 g/kg/day for 1–2 days is recommended [5, 6, 23]. Vaccine development efforts are ongoing [24].

CONCLUSIONS

M. pneumoniae infection presents with diverse clinical manifestations, complicating diagnosis and treatment. Four illustrative cases are presented: (1) pneumonia of *M. pneumoniae* etiology; (2) pneumonia with myocarditis; (3) pneumonia with meningitis; and (4) multi-organ *M. pneumoniae* infection with Stevens-Johnson syndrome. Common symptoms include persistent cough and general malaise, but extra-pulmonary complications, i.e., neurological, cardiovascular, or mucocutaneous, vary in severity.

Symptoms' expression depends on various factors, such as patient age, co-infections, and immune status. Accurate diagnosis requires immunoassays and PCR, and treatment must be tailored to disease severity and account for rising macrolide resistance. The infection's clinical variability underscores the need for deeper understanding and targeted therapeutic strategies.

DISCLOSURES

1. Institutional review board statement: Not applicable.
2. Assistance with the article: None.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

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