

Original Research Article

Recognizing *Mycoplasma pneumoniae* early in children: a clinico-laboratory pattern and a simple hematology clue

Sandeep Garg^{1*}, Sunita Prasad², Riddhi Jadhav¹

¹Department of Pediatrics, Bhaktivedanta Hospital and research Institute, Thane, Maharashtra, India

²Department of Laboratory Medicine, Bhaktivedanta Hospital and research Institute, Thane, Maharashtra, India

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*Correspondence:

Dr. Sandeep Garg,

E-mail: drsandeep2109@gmail.com

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ABSTRACT

Background: Early diagnosis of *Mycoplasma pneumoniae* pneumonia (MPP) is challenging because its presentation overlaps with viral and typical bacterial pneumonia. Although polymerase chain reaction (PCR) is the diagnostic standard, it is not routinely available in most Indian laboratories, leaving clinicians dependent on serology, which has recognized limitations. We aimed to identify a practical clinicolaboratory pattern, including a simple hematological clue, for early recognition of pediatric MPP.

Methods: This retrospective observational study included 60 children managed during a three-month community outbreak of clinically suspected and/or serologically confirmed MPP at a tertiary care pediatric center. Clinical, laboratory, radiographic, treatment and outcome data were reviewed, emphasizing automated complete blood count (CBC) abnormalities and their correction after incubating EDTA samples at 37°C.

Results: Children presented with prolonged fever, persistent dry cough, mild respiratory distress despite radiographic pneumonia and poor response to β -lactam antibiotics. Inflammatory markers were modestly elevated, with normal or mildly increased leukocyte counts. Chest radiographs showed lower lobe consolidation, although bilateral pneumonia, pleural effusion, lobar collapse and severe extrapulmonary manifestations also occurred. Several children showed spuriously elevated mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) with reduced red blood cell counts due to cold agglutinin-mediated agglutination, which normalized after warming samples to 37°C. Most improved rapidly after azithromycin or doxycycline.

Conclusions: Recognition of a characteristic clinicolaboratory pattern, together with reversible cold agglutinin-mediated CBC abnormalities, may facilitate early suspicion of MPP where molecular diagnostics are unavailable. Awareness of this simple hematological clue may enable earlier targeted therapy, reduce unnecessary β -lactam exposure and improve outcomes.

Keywords: Community-acquired pneumonia, Cold agglutinins, Complete blood count, *Mycoplasma pneumoniae*, MCHC, Pediatric pneumonia

INTRODUCTION

Mycoplasma pneumoniae is an important cause of community-acquired pneumonia (CAP) in children, accounting for approximately 10–40% of cases, particularly among school-aged children and adolescents. The infection typically occurs in cyclical epidemics every 3–7 years and remains a significant cause of respiratory

morbidity worldwide. Following relaxation of COVID-19-related public health measures, many countries have reported a marked resurgence of *M. pneumoniae* infections, highlighting the continued importance of early recognition and appropriate treatment.^{1–5} Despite its clinical importance, early diagnosis of *M. pneumoniae* infection remains difficult because its presentation often overlaps with both viral and typical bacterial pneumonia.

Children commonly present with prolonged fever, persistent dry cough and relatively mild respiratory distress despite significant radiographic abnormalities a syndrome classically described as “walking pneumonia.” Routine laboratory investigations are frequently nonspecific and empirical β -lactam antibiotics are commonly prescribed as first-line therapy for CAP. Because *M. pneumoniae* lacks a peptidoglycan cell wall, β -lactam antibiotics are intrinsically ineffective, frequently resulting in delayed clinical improvement until appropriate anti-mycoplasma therapy is initiated.⁶⁻⁹

The diagnostic challenge is even greater in low- and middle-income countries such as India, where commercially available PCR testing is not routinely accessible in most clinical laboratories. Consequently, diagnosis relies largely on Mycoplasma IgM serology, which has important limitations, including delayed seroconversion, persistence of antibodies after previous infection and both false-positive and false-negative results. Accordingly, international guidelines recommend interpreting serological findings in conjunction with compatible clinical, radiological and laboratory features rather than in isolation.¹⁰⁻¹²

In this context, recognition of a characteristic clinicolaboratory pattern assumes particular importance. A combination of prolonged fever, persistent nonproductive cough, poor response to β -lactam antibiotics, relatively preserved general condition despite radiographic pneumonia, normal or mildly elevated leukocyte counts and modest inflammatory marker elevation may provide valuable clues to *M. pneumoniae* infection before microbiological confirmation becomes available. Such pattern recognition can facilitate earlier institution of appropriate antimicrobial therapy and potentially reduce unnecessary antibiotic exposure, disease duration, complications and healthcare utilization.

An additional but underrecognized diagnostic clue may be obtained from routine automated CBC analysis. *M. pneumoniae* infection is well known to induce cold agglutinin production, resulting in temperature-dependent red blood cell (RBC) agglutination. This phenomenon may produce spuriously reduced RBC counts together with markedly elevated MCH and MCHC, abnormalities that normalize after warming EDTA blood samples to 37°C before repeat analysis. Although the immunological basis of cold agglutinin-mediated RBC agglutination is well established, its practical value as an early diagnostic clue in pediatric respiratory infections has received relatively little attention in routine clinical practice.^{13,14}

During a community outbreak of pediatric *M. pneumoniae* infection at our tertiary care center, we observed a remarkably consistent combination of clinical, radiological and laboratory findings, together with reproducible CBC abnormalities attributable to cold agglutinin-mediated RBC agglutination. Authors hypothesized that this constellation of routinely available

findings could provide a practical bedside framework for early recognition of *M. pneumoniae* infection, particularly in settings where molecular diagnostics are unavailable. The present study therefore aimed to describe the clinicolaboratory profile of affected children and highlight a simple hematological clue that may assist both pediatricians and laboratory physicians in raising early diagnostic suspicion and initiating timely targeted therapy.

METHODS

Study design and setting

This retrospective observational study was conducted in the Department of Pediatrics, Bhaktivedanta Hospital and Research Institute, Mumbai, India, during a community outbreak of Mycoplasma pneumoniae infection over a three-month period in 2024.

Study population

Children aged 1 month to 18 years admitted with CAP and clinically suspected and/or *M. pneumoniae* IgM-positive infection were included. Clinical suspicion was based on prolonged fever (≥ 5 days), persistent cough, chest radiographic findings suggestive of atypical pneumonia, poor response to appropriate β -lactam antibiotics after 48–72 hours and subsequent clinical improvement following anti-mycoplasma therapy. Patients with an alternative confirmed diagnosis explaining the illness were excluded.

The primary objective of the study was to identify a practical constellation of clinical, radiological and routine laboratory findings that could facilitate early recognition of *M. pneumoniae* infection before definitive laboratory confirmation.

Data collection

Hospital records were reviewed for demographic characteristics, presenting symptoms, physical examination findings, laboratory investigations, chest radiographic findings, treatment received and clinical outcomes. Laboratory parameters included CBC, inflammatory markers, liver function tests and *Mycoplasma pneumoniae* IgM serology.

Hematological evaluation

Special attention was paid to automated CBC parameters suggestive of cold agglutinin-mediated RBC agglutination. Samples demonstrating analyzer flags for RBC agglutination or physiologically implausible erythrocyte indices (low RBC count with disproportionately elevated MCH and MCHC) underwent peripheral smear examination. EDTA blood samples were then incubated at 37°C for 15–30 minutes, followed by repeat CBC analysis to evaluate correction of

erythrocyte indices and disappearance of RBC agglutination.

Statistical analysis

Data were analyzed descriptively. Continuous variables are presented as mean±standard deviation or median (interquartile range), while categorical variables are expressed as frequencies and percentages.

Ethical considerations

There was no ethics approval as it was retrospective observational study.

RESULTS

During the study period, 60 children with clinically suspected and/or serologically confirmed *Mycoplasma pneumoniae* pneumonia were managed at our tertiary care center. The outbreak demonstrated a remarkably consistent clinicolaboratory profile, enabling early recognition of the disease before laboratory confirmation in many patients.

Most children presented after 5–10 days of fever accompanied by persistent, predominantly dry cough. Many had already received empirical β -lactam antibiotics before referral but showed little or no clinical improvement. Despite radiographic evidence of pneumonia, the majority remained relatively non-toxic with preserved oxygen saturation and only mild-to-moderate respiratory distress, consistent with the classical description of “walking pneumonia.”

Routine laboratory investigations demonstrated an intermediate inflammatory profile. Total leukocyte counts were generally normal or only mildly elevated, while inflammatory markers showed moderate elevation rather than the marked response usually observed in pyogenic bacterial pneumonia. Platelet counts remained largely preserved. Mild elevation of hepatic transaminases was observed in a subset of children.

Chest radiographs showed variable patterns of pulmonary involvement (Figure 2). Lower lobe consolidation was the commonest abnormality, although bilateral pneumonia, diffuse patchy infiltrates, pleural effusion and lobar collapse were also encountered. In several patients, radiological abnormalities appeared disproportionately extensive compared with the relatively mild clinical presentation.

Although most children experienced an uncomplicated course, a small number developed severe disease manifestations, including bilateral pneumonia requiring non-invasive or invasive ventilatory support, hemorrhagic pleural effusion, Stevens-Johnson syndrome and *Mycoplasma pneumoniae* infection complicating congenital pulmonary airway malformation. These

observations illustrate the broad clinical spectrum of pediatric *M. pneumoniae* infection.

A particularly noteworthy observation was the occurrence of characteristic abnormalities on automated CBC analysis in selected patients (Figure 3). Automated hematology analyzers demonstrated spuriously reduced RBC counts together with markedly elevated mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC), frequently accompanied by analyzer flags indicating RBC agglutination. Peripheral blood smear examination confirmed red blood cell clumping. Following incubation of EDTA blood samples at 37°C, repeat CBC analysis demonstrated normalization of erythrocyte indices with disappearance of analyzer flags and complete resolution of RBC agglutination on peripheral smear (Table 1, Figure 1).

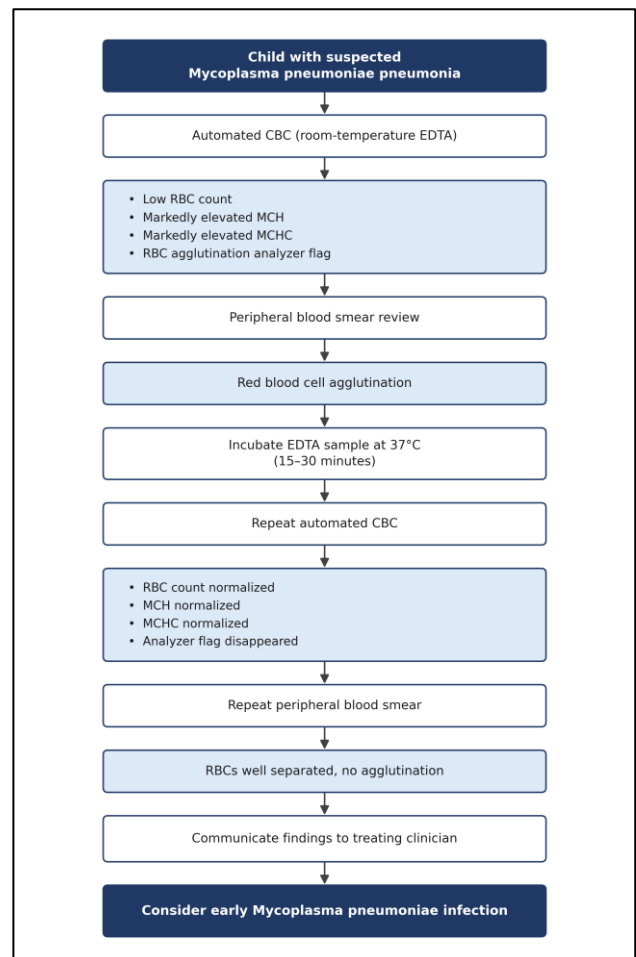


Figure 1: Proposed workflow for recognition of cold agglutinin-mediated CBC abnormalities. Workflow demonstrating correction of cold agglutinin-mediated CBC abnormalities following incubation of EDTA blood samples at 37°C. Automated CBC demonstrates spuriously reduced RBC count with elevated MCH and MCHC due to RBC agglutination. Following warming, erythrocyte indices normalize and RBC agglutination disappears on peripheral smear.

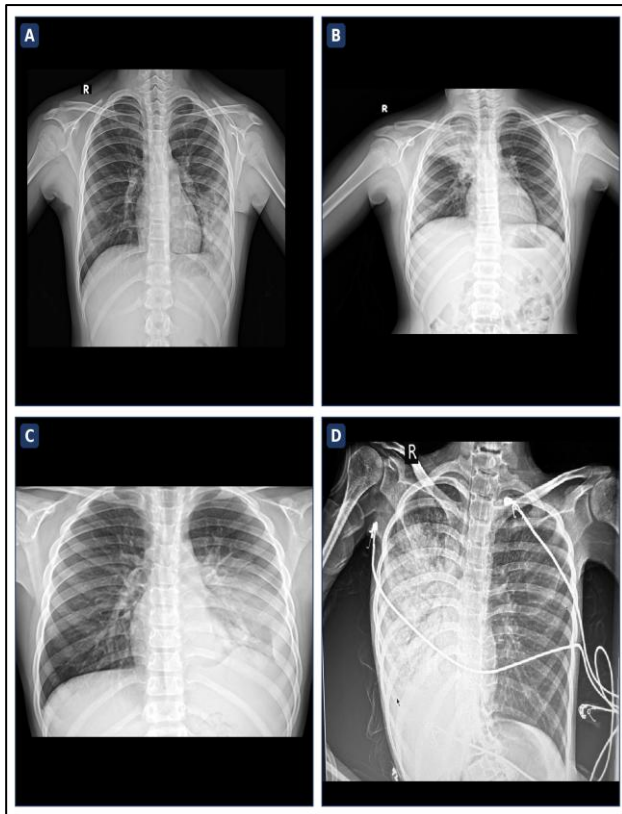


Figure 2 (A-D): Representative chest radiographs of children with *Mycoplasma pneumoniae* pneumonia.

Images illustrate the spectrum of pulmonary involvement observed during the outbreak, including lower lobe consolidation, bilateral pneumonia, pleural effusion, diffuse infiltrates and severe complicated disease.

This reversible phenomenon was consistent with cold agglutinin-mediated RBC agglutination, providing a

Table 1: Representative correction of complete blood count indices following incubation of EDTA blood sample at 37°C.

Parameters	Before warming	After warming (37°C)
RBC ($\times 10^6/\mu\text{l}$)	0.76	3.01
Hemoglobin (g/dl)	10.7	10.6
MCH (pg)	140.8	35.2
MCHC (g/dl)	159.7	38.4
Automated analyzer	RBC agglutination flag present	Analyzer flag resolved
Peripheral smear	Marked RBC clumping	RBCs well separated

Table 2: Clinical situations that should raise suspicion for *Mycoplasma pneumoniae* pneumonia.

Clinical situations	Diagnostic implication
Prolonged fever with persistent dry cough and relatively non-toxic (“walking pneumonia”) appearance	Classical presentation of MPP
Chest X-ray more extensive than expected from clinical examination	Clinical–radiological dissociation favors atypical pneumonia
Extrapulmonary manifestations accompanying pneumonia (rash, hepatitis, mucositis, hemolysis, neurological involvement)	Suggests immune-mediated <i>Mycoplasma</i> disease
CAP not improving after 48–72 hours of appropriate β -lactam	Consider atypical pneumonia because β -

Continued.

readily available laboratory clue that prompted consideration of *M. pneumoniae* infection even before serological confirmation. Most children demonstrated rapid clinical improvement following initiation of azithromycin. Patients with persistent fever despite macrolide therapy were managed as probable macrolide-resistant *M. pneumoniae* infection and responded to doxycycline (in age-appropriate children) or levofloxacin according to disease severity. Overall, clinical outcomes were favorable following institution of targeted anti-mycoplasma therapy.

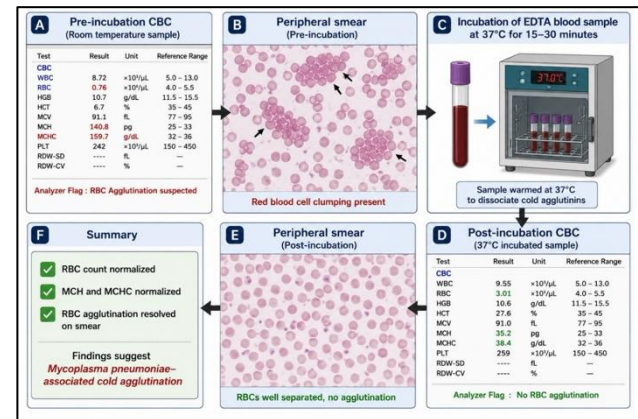


Table 3 (A-F): Six-panel illustration of the CBC warming phenomenon. Automated CBC before warming showing abnormal erythrocyte indices.

Peripheral smear demonstrating red blood cell agglutination. Incubation of EDTA blood sample at 37°C. Repeat CBC showing normalization of RBC count, MCH and MCHC. Repeat peripheral smear demonstrating resolution of RBC agglutination.

Clinical implication: early communication to the treating clinician prompting consideration of *Mycoplasma pneumoniae* infection.

Clinical situations	Diagnostic implication
therapy	lactams lack activity against <i>M. pneumoniae</i>
Clinical deterioration despite escalation of β -lactam antibiotics	Consider <i>M. pneumoniae</i> as the primary pathogen or coinfection
Persistent wheeze associated with pneumonia	Airway inflammation due to <i>M. pneumoniae</i> should be considered
Normal or mildly elevated leukocyte count with moderate CRP elevation despite radiographic pneumonia	Intermediate inflammatory profile between viral and pyogenic bacterial pneumonia
Unexpected CBC abnormalities (high MCHC/MCH, low RBC count, RBC agglutination corrected after warming)	Strong laboratory clue to cold agglutinin-mediated <i>M. pneumoniae</i> infection

Table 3: Practical clinicolaboratory framework for differentiating common causes of pediatric pneumonia.

Features	Viral pneumonia	<i>Mycoplasma pneumoniae</i>	Typical bacterial pneumonia
Fever	Variable	Prolonged	High
Cough	Variable	Persistent, dry	Productive
General condition	Usually well	Relatively well (“walking pneumonia”)	Toxic appearance
WBC count	Low/normal	Normal–mildly elevated	Elevated with neutrophilia
CRP	Low	Moderate	High
Chest X-ray	Interstitial/perihilar	Patchy, segmental or lobar±bilateral involvement	Dense lobar consolidation
Response to β -lactam antibiotics	Not applicable	Poor	Good
CBC clue	None	Elevated MCH/MCHC with RBC agglutination corrected by warming	None
Preferred therapy	Supportive	Azithromycin/Doxycycline*	β -lactam antibiotics

*Doxycycline in age-appropriate children, according to current recommendations.

DISCUSSION

Early recognition of pediatric MPP remains challenging because no single clinical feature or laboratory test reliably establishes the diagnosis during the initial stages of illness. Our study demonstrates that, even in the absence of routinely available molecular diagnostics, a characteristic constellation of clinical, radiological and routine laboratory findings can substantially increase early diagnostic suspicion. Rather than relying on an individual investigation, clinicians should recognize a reproducible clinicolaboratory pattern comprising prolonged fever, persistent dry cough, relatively mild clinical toxicity despite radiographic pneumonia, poor response to β -lactam antibiotics, modest inflammatory marker elevation and characteristic cold agglutinin-mediated CBC abnormalities (Table 2). This pattern formed the basis of early therapeutic decision-making during our outbreak and may be readily applicable in routine pediatric practice.^{1,10}

A useful conceptual framework is to consider *M. pneumoniae* as occupying an intermediate biological position between viral and typical bacterial pathogens. Like bacteria, it is susceptible to antimicrobial therapy; however, because it lacks a peptidoglycan cell wall, β -lactam antibiotics are intrinsically ineffective. Consequently, its inflammatory profile often lies between that of viral and pyogenic bacterial pneumonia. Viral infections generally produce low inflammatory markers

and lymphocyte-predominant responses, whereas typical bacterial pneumonia is characterized by neutrophilic leukocytosis, marked CRP elevation and rapid response to β -lactam therapy. In contrast, children with MPP frequently exhibit normal or mildly elevated leukocyte counts, moderate CRP elevation, persistent dry cough and failure to improve despite appropriate β -lactam treatment (Table 3). Appreciating this intermediate phenotype can improve bedside diagnostic reasoning and reduce unnecessary escalation of ineffective antibiotics.^{6,10}

The present study also highlights an important practical issue in many low- and middle-income countries. Although PCR remains the preferred diagnostic modality because of its high sensitivity and specificity, it is not routinely available in most clinical laboratories in India. In day-to-day practice, clinicians therefore depend largely on *Mycoplasma* IgM serology, which has well-recognized limitations. Antibody responses may be absent early in the disease, persist long after infection or occasionally yield false-positive results. Current international recommendations therefore emphasize interpreting serological results within the overall clinical context rather than in isolation.^{2,9,11} The findings support this approach and suggest that clinical pattern recognition should guide early management while awaiting or interpreting laboratory confirmation.

The most distinctive observation of our study was the reproducible correction of abnormal erythrocyte indices following incubation of EDTA blood samples at 37°C.

Children demonstrating spuriously low RBC counts together with markedly elevated MCH and MCHC consistently showed normalization of these parameters after warming, accompanied by disappearance of RBC agglutination on peripheral smear. This reversible phenomenon reflects cold agglutinin-mediated erythrocyte agglutination induced by *M. pneumoniae*. Although the immunopathogenesis of cold agglutinin formation is well established, its routine application as a practical diagnostic clue for pediatric respiratory infections has received relatively little attention.^{12,13,15}

The importance of this observation extends beyond hematology. Automated CBC results are often available before microbiological investigations or specialist review. Recognition of physiologically implausible erythrocyte indices particularly an unexpectedly elevated MCHC (>38 g/dl), disproportionate elevation of MCH, low RBC count or analyzer flags indicating RBC agglutination should prompt peripheral smear examination and repeat CBC analysis after warming the sample. If these abnormalities resolve, the hematology laboratory should communicate the finding to the treating pediatrician as a potential indicator of cold agglutinin-mediated disease. In a child with prolonged fever and persistent cough, this simple observation may provide an early clue to *M. pneumoniae* infection, allowing timely initiation of targeted therapy even before serological results become available.^{4,13-15} Analyzer flags indicating RBC agglutination were noted in only 11 of the 60 children in this series; even in this limited subset, however, the finding helped guide earlier diagnosis and treatment.

From a clinical perspective, the observations suggest that MPP should be suspected whenever the overall clinical picture appears discordant with either viral or typical bacterial pneumonia. This pattern-based approach is particularly valuable in resource-limited settings where definitive molecular diagnostics are unavailable. Rather than pursuing repeated courses or escalation of β -lactam antibiotics, clinicians should consider atypical pathogens early in the disease course and initiate appropriate anti-mycoplasma therapy when the constellation of findings is strongly suggestive. Early targeted treatment may shorten illness duration, reduce unnecessary antibiotic exposure, minimize complications and improve patient outcomes.^{6,10}

The study also underscores the importance of close collaboration between pediatricians, pathologists and hematology laboratories. Recognition of cold agglutinin-mediated CBC abnormalities requires awareness at the laboratory level, while their clinical significance becomes apparent only when integrated with the patient's history, examination findings and radiological features. This interdisciplinary communication represents a simple but potentially impactful strategy for improving early diagnosis without additional cost or specialized investigations.

The principal strength of the study is its reflection of real-world clinical practice during a community outbreak, allowing identification of a consistent diagnostic pattern applicable to routine pediatric care. In addition, we highlight a readily available hematological clue that can be recognized using standard automated hematology analyzers available in most hospitals. The study is limited by its retrospective design, single-center setting and the absence of molecular confirmation because PCR testing was not routinely available during the study period. Diagnosis therefore relied on a combination of compatible clinical features, radiological findings, serology, exclusion of alternative diagnoses and response to targeted therapy. Larger prospective multicenter studies incorporating standardized molecular diagnostics are needed to validate the diagnostic accuracy of the proposed clinicolaboratory framework and to determine its predictive value across different epidemiological settings.

Overall, the findings support a practical diagnostic strategy centered on pattern recognition rather than dependence on a single laboratory test. Increased awareness of this characteristic clinicolaboratory profile, together with the CBC warming phenomenon, may facilitate earlier diagnosis and more timely institution of targeted therapy, particularly in healthcare settings where molecular diagnostics remain inaccessible.

The central message of the study is that the diagnosis of pediatric MPP should not depend on a single laboratory investigation. Instead, it should emerge from the integration of clinical presentation, radiological findings, routine hematological parameters and therapeutic response. Such an approach is particularly relevant in resource-limited settings where molecular diagnostics remain inaccessible. The CBC warming phenomenon represents a simple yet valuable addition to this diagnostic framework, enabling the hematology laboratory to play an active role in the early recognition of this important pathogen.

CONCLUSION

Early diagnosis of pediatric Mycoplasma pneumoniae pneumonia remains challenging because of its nonspecific clinical presentation and the limited availability of rapid molecular diagnostics in many resource-constrained settings. The study demonstrates that diagnosis need not rely solely on microbiological confirmation. Instead, recognition of a characteristic clinicolaboratory pattern prolonged fever, persistent dry cough, relatively mild clinical illness despite significant radiological pneumonia, poor response to β -lactam antibiotics, normal or mildly elevated leukocyte counts, moderate inflammatory marker elevation and reversible cold agglutinin-mediated CBC abnormalities can substantially improve early diagnostic suspicion.

Among these observations, the CBC warming phenomenon represents a simple, inexpensive and readily available laboratory clue. Recognition of spuriously elevated MCH and MCHC with reduced RBC counts and analyzer flags for RBC agglutination, followed by normalization after warming EDTA blood samples to 37°C, should alert both pathologists and paediatricians to the possibility of *M. pneumoniae* infection. Awareness of this finding may facilitate earlier initiation of appropriate anti-mycoplasma therapy, reduce unnecessary β -lactam exposure and potentially shorten illness duration and associated morbidity.

Authors propose that this practical clinicolaboratory framework may serve as a useful bedside approach for the early recognition of pediatric *M. pneumoniae* pneumonia, particularly in regions where PCR testing is not routinely available. Prospective multicenter studies are warranted to validate its diagnostic performance and assess its incorporation into routine clinical practice.

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Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

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