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Case Report

From pneumonia to pulmonary embolism: refractory *Mycoplasma pneumoniae* in a 13-year-old girl

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ABSTRACT

Macrolide-resistant *Mycoplasma pneumoniae* pneumonia (MRMP) is increasingly reported in children and may present with extrapulmonary complications, including pulmonary thromboembolism and immune-mediated organ dysfunction. Thromboembolic events related to *M. pneumoniae* are rare but potentially life-threatening and often associated with transient antiphospholipid antibodies such as anti-beta-2 glycoprotein. A previously healthy 13-year-old girl presented with 7 days of high-grade intermittent fever and productive cough, unresponsive to Oseltamivir and Amoxicillin-Clavulanate. Examination and imaging showed right upper lobe consolidation with minimal pleural effusion. Laboratory evaluation revealed raised ESR and elevated CRP, LDH, ferritin, D-dimer, and procalcitonin, with subsequent maculopapular rash, transaminitis, positive cold agglutinins, following which *M. pneumoniae* IgM was positive. CTPA confirmed pulmonary thromboembolism, thrombophilia workup showed positive beta-2 glycoprotein antibodies. She was treated with Ceftriaxone and Azithromycin, escalated to Meropenem and Linezolid, and then Doxycycline for suspected MRMP. Persistent fever, high inflammatory markers, and radiological progression warranted Methylprednisolone, resulting in defervescence and clinical improvement. Progressive pleural effusion required thoracentesis, and pulmonary emboli were managed with Enoxaparin. Refractory *M. pneumoniae* pneumonia in children can be complicated by pulmonary embolism (PE) and transient antiphospholipid antibodies, even without structural heart disease or limb thrombosis. Early suspicion of thrombosis in the presence of elevated D-dimer, prompt CT pulmonary angiography, and combined second-line antibiotics, corticosteroids, and anticoagulants are crucial to optimize outcomes.

Keywords: Refractory pneumonia, Macrolide resistance, Pulmonary embolism, Antiphospholipid antibodies, Pediatric mycoplasma

INTRODUCTION

Mycoplasma pneumoniae is a common cause of community-acquired pneumonia in school-aged children, usually producing mild, self-limiting disease. In the last decade, macrolide-resistant *M. pneumoniae* (MRMP) and refractory *M. pneumoniae* pneumonia (RMPP) have been increasingly recognized, characterized by prolonged fever, progressive radiological lesions, and extrapulmonary manifestations.[1-3] Thromboembolic phenomena, including PE and deep vein thrombosis, are rare but serious complications, thought to involve hypercoagulability, endothelial injury, and transient antiphospholipid antibodies.[4-10]

Several pediatric series describe *M. pneumoniae*-associated PE in older children with severe pneumonia, markedly elevated D-dimer, and antiphospholipid antibody positivity, with most improving on combined anti-infective and anticoagulant therapy.[1,4-7,9,10] This report describes a 13-year-old girl with RMPP complicated by bilateral pulmonary artery emboli, right-sided exudative pleural effusion, and beta-2 glycoprotein antibody positivity, with good outcome following stepwise

antimicrobial escalation, corticosteroids, and anticoagulation, highlighting the need to consider PE when D-dimer is disproportionately elevated.[1,4-7,9]

CASE REPORT

A previously healthy 13-year-old girl presented with 7 days of high-grade, intermittent fever and productive cough without diurnal or postural variation. There was no history of breathing difficulty, cyanosis, abdominal pain, loose stools, rash at onset, hematuria, blood in stools or recent travel.

On admission, she was febrile and tachypneic but hemodynamically stable. Respiratory examination revealed reduced air entry over the right side with whispering pectoriloquy. Chest radiograph showed right upper lobe consolidation, and chest ultrasonography demonstrated mild right-sided pleural effusion. Baseline blood counts were normal, with raised ESR (22 mm/hr) and CRP (127.12 mg/L).

She was started on ceftriaxone and oral Azithromycin for lobar pneumonia with synpneumonic effusion consistent with empirical management of pediatric community-acquired pneumonia when both typical and atypical pathogens are suspected.[12,13] Despite this, repeat investigations showed rising LDH (1250 IU/l), CRP (117.35 mg/l), procalcitonin (1.3 ng/ml), ferritin (441.2 ng/mL), and D-dimer (10 µg/ml), and she developed a generalized maculopapular rash. Tropical infection screen and sputum CBNAAT for *Mycobacterium tuberculosis* were negative.

Cold agglutinin positivity supported the diagnosis of *M. pneumoniae* pneumonia, prompting initiation of Doxycycline suspecting MRMP; this was subsequently confirmed by elevated serum *M. pneumoniae* IgM. Fever and radiologic infiltrates progressed, and transaminitis developed, while viral hepatitis panel and echocardiography were normal. Repeat chest ultrasonography showed moderate-to-severe right pleural effusion. Ultrasound-guided diagnostic and therapeutic thoracentesis yielded 60-70 mL of straw-colored exudative fluid, compatible with parapneumonic effusion described in severe *M. pneumoniae* pneumonia.

Persistent fever beyond 48 hours of Doxycycline, rising inflammatory markers, progressive rash, and worsening radiology led to a diagnosis of RMPP. Given markedly elevated D-dimer in the setting of systemic inflammation, CT pulmonary angiography was done and showed bilateral segmental pulmonary artery emboli with ongoing parenchymal consolidation. Doppler ultrasonography of limbs excluded peripheral arterial or venous thrombosis, and echocardiogram remained normal, a pattern similar to other pediatric *M. pneumoniae*-associated PE cases. Thrombophilia evaluation showed positive beta-2 glycoprotein IgG and IgM with otherwise unremarkable results, suggesting infection-related antiphospholipid antibody-mediated thrombosis.

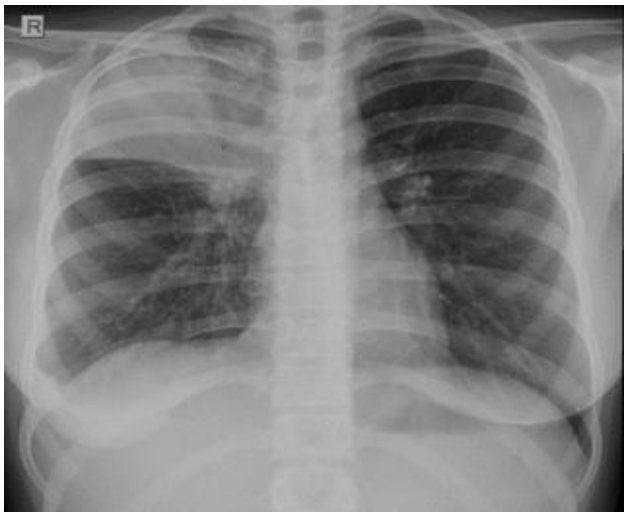

Figure 1: Radiograph showing right upper-lobe consolidation with synpneumonic effusion.

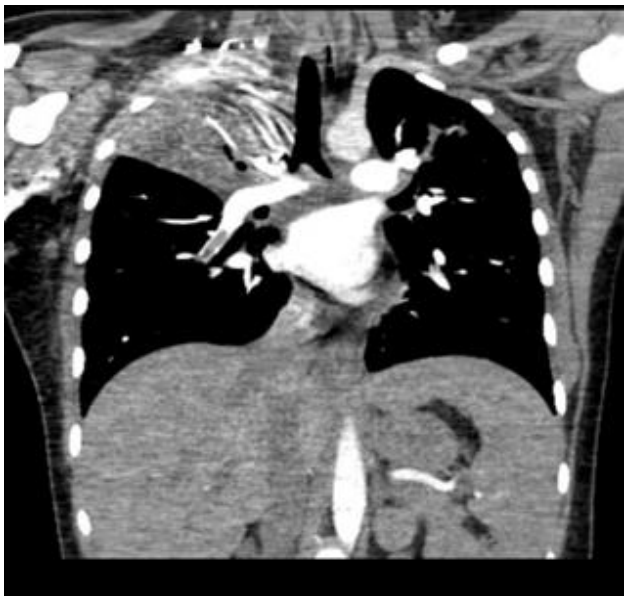

Figure 2: CTPA showing tram-track sign of pulmonary thrombus.

Management and outcome

Empirical Ceftriaxone plus Azithromycin for lobar community-acquired pneumonia with mild effusion was appropriate where atypical pathogens are possible and macrolide resistance status is unknown. Persistent fever, radiologic progression and rising inflammatory markers prompted escalation to intravenous Meropenem and Linezolid to broaden antibacterial coverage, although cultures remained negative.

Cold agglutinin positivity and ongoing fever with radiologic worsening despite adequate macrolide therapy

raised suspicion of MRMP, and Doxycycline was commenced as a second-line agent. However, fever spikes, rash, elevated inflammatory markers and worsening liver enzymes persisted despite hepatoprotective measures, consistent with severe systemic inflammation and sepsis-related transaminitis.

Methylprednisolone (10 mg/kg/day on day 1 and 5 mg/kg/day on day 2) was started followed by an oral taper, in line with regimens used in RMPP to modulate excessive immune responses. The child became afebrile soon after steroid initiation and showed rapid improvement, similar to reports where early corticosteroids accelerate radiologic recovery in severe *M. pneumoniae* pneumonia.

Progressive pleural effusion was managed with thoracentesis, which confirmed an exudative, non-empyematous parapneumonic effusion. Markedly elevated D-dimer and severe *M. pneumoniae* infection prompted CT pulmonary angiography, confirming bilateral segmental pulmonary emboli; similar pediatric cases report elevated D-dimer, segmental or lobar emboli, and good outcomes with timely anticoagulation.

In consultation with cardiology, therapeutic low molecular weight heparin (Enoxaparin 40 IU twice daily; weight-adjusted) was initiated after obtaining thrombophilia samples, following pediatric PE management principles. Repeat echocardiography showed normal function and limb Doppler was negative for deep vein thrombosis. She completed 10 days of intravenous Meropenem and Linezolid, with ongoing Doxycycline and tapering oral steroids.

Serial monitoring showed objective improvement: chest radiograph demonstrated resolution of right upper lobe consolidation, and repeat chest ultrasonography confirmed complete resolution of effusion. Inflammatory markers fell markedly (CRP 8.48 mg/l, D-dimer 2.83 µg/ml), with improving liver enzymes. She remained hemodynamically stable without bleeding or anticoagulant-related complications.

Thrombophilia workup showed normal complement, negative ANA, antiphospholipid IgG, and anticardiolipin antibodies, but initial beta-2 glycoprotein IgG and IgM positivity was consistent with transient antiphospholipid antibodies described in *M. pneumoniae*-associated thrombosis. She was discharged on day 14 on tapering oral steroids and continued anticoagulation. Low molecular weight heparin was given for 2 months, followed by Apixaban after hematology review; subsequent genetic testing and repeat beta-2 glycoprotein IgG/IgM were normal.

DISCUSSION

This case illustrates typical features of MRMP and RMPP in children: delayed response to macrolides, progressive radiologic disease, and extrapulmonary complications including thromboembolism.[1-3,5,6,9] Macrolide resistance in *M. pneumoniae* is associated with prolonged fever, longer hospitalization and greater need for second-line antibiotics and corticosteroids.[1-3,5,9] In this patient, clinical failure with Azithromycin and worsening pneumonia justified timely Doxycycline use, which is appropriate in older children and supported by observational data.[1-3,11]

PE is an increasingly recognized complication of severe M. pneumoniae infection, with several reports in school-aged children and adolescents with markedly elevated D-dimer.[1,4-7,9,10] Proposed mechanisms include infection-induced endothelial injury, hypercoagulability, and immune-mediated thrombosis associated with antiphospholipid antibodies.[4-8] Many children with *M. pneumoniae*-related thrombosis show transient anticardiolipin, beta-2 glycoprotein or lupus anticoagulant positivity, reverting to negative on follow-up, as in this child.

Corticosteroid therapy is widely recommended for severe or refractory *M. pneumoniae* pneumonia with marked systemic inflammation, with observational studies showing faster defervescence and radiographic improvement.[1–3,5,9] The rapid response to methylprednisolone here supports a major immunopathologic component, although steroids should be reserved for carefully evaluated patients after excluding empyema or uncontrolled bacterial sepsis.[1-3]

Management of *M. pneumoniae*-associated PE follows general pediatric venous thromboembolism principles, usually involving weight-adjusted LMWH followed by oral anticoagulation, with duration individualized by thrombus burden and risk factors. Most pediatric cases improve with timely anticoagulation, although fatalities have been reported in critical illness. The absence of deep vein thrombosis in this patient, as in some prior reports, raises the possibility of in situ pulmonary thrombosis on inflamed vascular endothelium rather than classic embolism from limb veins.

Pleural effusion in *M. pneumoniae* pneumonia is less frequent than in typical bacterial pneumonia but is increasingly recognized in severe disease, usually exudative and sterile; the non-empyematous effusion and its resolution with medical therapy plus thoracentesis in this child mirror prior descriptions. Transaminitis in severe *M. pneumoniae* infection is often attributed to sepsis-related hepatic injury or drugs and generally improves with supportive care and control of inflammation.

This case emphasizes the need for high suspicion of PE in severe *M. pneumoniae* pneumonia with marked D-dimer elevation and supports an approach combining second-line antibiotics, corticosteroids, early CT pulmonary angiography, anticoagulation, and thrombophilia evaluation.[1-7,9-11]

CONCLUSION

RMPP in children may be complicated by PE and transient antiphospholipid antibodies, even without an underlying condition. Elevated D-dimer should prompt early CT pulmonary angiography, and timely second-line antibiotics, steroids, and anticoagulation improves outcome.

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