

Case Report

Cutaneous vasculitis in a child with community acquired pneumonia

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ABSTRACT

Mycoplasma pneumoniae is an important etiological agent in community acquired pneumonia (CAP) in children aged 3 to 15 years. *Mycoplasma pneumoniae* may present with varied extra pulmonary manifestations. A 5 year old child presented with cough and fever and was initially managed as CAP due to *Streptococcus pneumoniae*. Child continued to have fever spikes and worsening distress and developed pleural effusion. Mycoplasma immunoglobulin M (IgM) was raised and child was treated with azithromycin. After 10 days of admission, the child developed fissuring of lips and discoloration of extremities. Direct Coombs test, cold agglutination test, antinuclear antibody (ANA) and anticardiolipin antibody were positive. Suspecting small vessel vasculitis, she was started on enoxaparin and aspirin and improved well. This case of CAP due to *Mycoplasma pneumoniae* is presented for the rare extrapulmonary manifestation of cutaneous vasculitis.

Keywords: *Mycoplasma pneumoniae*, Small vessel vasculitis, Macrolides

INTRODUCTION

Mycoplasma pneumoniae is a frequent cause of community acquired pneumonia in children between 3 to 15 years of age (7-40%).¹ Extra pulmonary manifestations can be seen in 25% of patients.² The exact pathogenesis of extra pulmonary manifestations is not clear.³ There are not many reports on mycoplasma induced vasculitis.

CASE REPORT

A 5 year old female child presented with fever and cough for 6 days. On examination, she was febrile, tachypneic (respiratory rate 48/min), room air saturation was 93% with mild sub costal and intercostal retractions. Palpatory findings were unequal chest expansion and there was dull note on percussion over the left infrascapular region. She also had reduced air entry in the left infrascapular region and bilateral crepitations. Clinical diagnosis of left lower lobe pneumonia was made. Complete blood count showed normal total count, hemoglobin of 8.4 gm/dL and peripheral smear showed microcytic hypochromic anemia mainly nutritional.

Chest x-ray revealed opacity left mid and lower zones. She was started on intravenous ampicillin. On day 3 of admission, the child persisted to have fever with no clinical improvement. There was no rash. Repeat chest x-ray revealed left lower lobe opacity with left sided pleural effusion (Figure 1). Chest computed tomography (CT) was done for better delineation of pulmonary tissue and showed left lobar consolidation and bilateral pleural effusion more on left side. Antibiotics were escalated to linezolid, piperacillin tazobactam and clindamycin in view of oxygen requirement and non-resolution of pneumonia and child was shifted to intensive care. Pleural fluid analysis showed increased white blood cells (WBC) count (1.850×10³/ul) and lymphocyte predominant, increased protein (4.2 gm), normal sugar (51 mg/dL) suggestive of exudative pleural effusion and culture showed no growth. Tuberculosis workup was negative. Scrub typhus and dengue serology were negative. Blood culture was negative. Mycoplasma serology immunoglobulin M (IgM) was increased (>27), ratio of more than 1.1 being positive as per lab reference and child was started on oral azithromycin (10 mg/kg/dose). Child improved clinically, tachypnea settled, oxygen support was weaned off and

antibiotics were deescalated from linezolid and piperacillin tazobactam to amoxicillin-clavulanic acid and azithromycin course was completed for 10 days as advised by infectious disease specialist only for pneumonia.

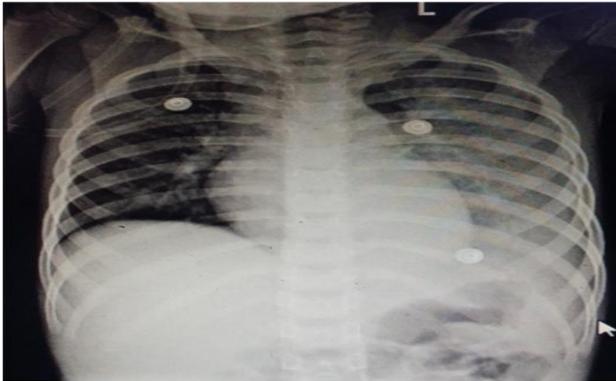


Figure 1: Chest X-ray showing left lobe opacity with left sided pleural effusion after worsening.

On day 10 of admission, she developed erythema and fissuring of lips with discoloration of toes (Figure 2). Direct Coombs test, cold agglutinin test (1:256), antinuclear antibody (ANA) and anti-cardiolipin antibody were positive. Complement levels were within normal limits. Antineutrophil cytoplasmic antibodies (cANCA and pANCA) were negative. Paediatric rheumatologist opinion was obtained and advised spot urine protein creatinine ratio which was normal, erythrocyte sedimentation rate (ESR) and c-reactive protein (CRP) was 124 mm/hour and 1.2 mg/dl respectively, platelets were 2.31 and coagulation profile was prothrombin time (PT)-14.6, partial thromboplastin time (PTT)-24.6, and international normalized ratio (INR-1.21). Skin biopsy was not done.



Figure 2: Discoloration of toe tips.

Small vessel vasculitis with cutaneous ischemia was suspected and vascular surgeon's advice was obtained to check flow of blood in arteries and veins with hand held Doppler and it was normal. She was started on enoxaparin prophylactically as advised by pediatric rheumatologist. Discoloration of toes improved within a couple of days. Enoxaparin was given for 5 days, and changed to aspirin

for 4 weeks in view of transient antiphospholipid antibodies (APLA) positivity. She received azithromycin for 10 days because the child had low grade fever spikes. Repeat antinuclear antibody (ANA), direct Coombs test (DCT), cold agglutinin and anticardiolipin IgM after a month were negative. A second serum sample to estimate the rise in mycoplasma titre could not be done because the child was on review with pediatric rheumatologist.

DISCUSSION

Cutaneous vasculitis due to *Mycoplasma pneumoniae* is rare and very few cases have been reported in pediatric age group, commonly in form of purpuric lesions. Van Bever et al described a 13 year old boy who developed severe respiratory distress syndrome, biochemical pancreatitis and skin vasculitis after an acute respiratory infection due to *Mycoplasma pneumoniae*.⁴

Fillipo reported a 7 year-old male affected with cutaneous and retinal vasculitis due to *M. pneumoniae* infection without pulmonary detection.⁵

This child presented with discoloration of tip of toes which made us suspect small vessel vasculitis. Though she had positive DCT and cold agglutinin, she did not have any clinical features of hemolysis such as pallor, jaundice or dark urine. She also had positive anticardiolipin IgM which in association with cutaneous changes was suggestive of small vessel vasculitis and hence was treated with enoxaparin. Treatment for vascular complications following mycoplasma infection includes corticosteroids and immunomodulator.³ This child did not receive steroids. Anticoagulation therapy with enoxaparin and aspirin was started for small vessel vasculitis and child improved well. The reason for ANA positivity in first episode followed by ANA turning negative in follow up is unclear.

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