

Bronchiectasis after mycoplasma pneumonia

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Infection by *Mycoplasma pneumoniae* is common and usually mild. A few cases are severe and deaths have occurred. Long term sequelae are rare. We describe the case of a 23 year old man who now has cylindrical bronchiectasis. His symptoms date from an episode of mycoplasma pneumonia, complicated by Stevens-Johnson syndrome, seven years previously. He is also unusual in that he has had a second episode of illness probably caused by *M pneumoniae*.

Case report

In February 1976 a 15 year old schoolboy presented with a one week history of headache, anorexia, and productive cough. There was no history of respiratory disease, pertussis, or croup, although as a child he had had uncomplicated measles. Two days before admission to hospital he had been prescribed flucloxacillin and ampicillin. Within 12 hours of the first dose he developed a sore mouth, swollen eyelids, and a generalised rash, initially red but later blistering.

On admission he was febrile (37.5°C) and delirious. Considerable ulceration of lips, mouth, and penile meatus was present. A vesicular rash, with a few "target" lesions, affected limbs and trunk. There was severe conjunctivitis. He had copious mucopurulent sputum but no adventitious sounds on chest auscultation. A diagnosis of Stevens-Johnson syndrome was made, secondary to a presumed viral respiratory illness. A chest radiograph showed left mid-zone inflammatory changes and an initial sputum culture grew *Haemophilus influenzae*, although over the next four days heavy growths of pneumococci and group B meningococci were also obtained. No antibiotics were given and his temperature settled spontaneously. Five days after his admission his serum mycoplasma antibody titre from the day of admission was reported as 128. This had risen to 2048 by day 6. The cold agglutinins titre was 1240. On day 5 treatment with oxytetracycline was started. He slowly improved, although fever returned on day 8 and was intermittently present over the next week. On day 12 crepitations and rhonchi were noted at the left lung base and a chest radiograph now showed consolidation in the left lower lobe and inflammatory changes in the right lower zone. Treatment was changed to amoxycillin, improvement continued, and he was discharged from hospital after a stay of five weeks. At review two weeks later he still had a

productive cough, although a chest radiograph showed appreciable resolution with only minor changes at the left base.

In December 1976 the boy was readmitted with fever and pleuritic chest pain. He was now smoking 15 cigarettes a day and stated that his cough had remained productive since his first admission. There was clinical and radiological evidence of left mid-zone consolidation and this responded to ampicillin. *Klebsiella aerogenes*, *Enterobacter cloacae*, and *Serratia marcescens* were cultured from sputum. Mycoplasma titre in both acute and convalescent sera was 64. He did not attend for review. When next admitted in 1980 with left lower lobe pneumonia he confirmed that an increasingly productive cough had persisted since his last stay. When the pneumonia resolved after treatment with erythromycin, changes suggestive of bronchiectasis were seen on the chest radiograph.

In June 1982 he noted a further increase in sputum volume, malaise, fever, pleuritic pain, and painful swelling of knees, wrists, and ankles. This did not respond to cephadrine. On admission he was febrile (38°C) and had enlarged cervical lymph nodes and gross finger clubbing. Signs of a right pleural effusion were present and coarse crepitations were heard over the left lower zone. The chest radiograph confirmed the effusion and again showed appearances suggestive of left lower lobe bronchiectasis. There was symmetrical tenderness and swelling of the wrists, knees, and ankles. The erythrocyte sedimentation rate was 101 mm in one hour, and sputum culture grew *H influenzae*. The antistreptolysin O titre was 100 units/ml and the results of both rheumatoid and antinuclear factor tests were negative. IgA and IgM concentrations were raised. He was treated with erythromycin and paracetamol. His arthropathy resolved rapidly but the respiratory features improved more slowly. The mycoplasma antibody titre rose from 256 on admission to 1024 after two weeks and fell to 32 after three months. After discharge he noted exertional dyspnoea. Cylindrical bronchiectasis at both bases was shown by bronchography. Concentrations of α_1 antitrypsin and sweat sodium were normal.

Discussion

We believe that bronchiectasis developed in this patient as a result of his *M pneumoniae* infection. The part played by the three secondary bacterial invaders (rare in mycoplasma pneumonias) remains uncertain. These pathogens or mucosal changes from the Stevens-Johnson syndrome may have been essential contributory factors leading to damage in this case. The Stevens-Johnson syndrome was itself probably induced by the mycoplasma infection rather than

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from the prescribed penicillins, as amoxycillin was given later in the same illness without any side effect. The exact mechanism of damage following mucosal attachment of *M pneumoniae* is uncertain. The inflammatory changes are submucosal and peribronchial.¹ Ciliary motility is impaired in test systems.²

Although extrapulmonary complications, such as rashes and arthralgia, are common pulmonary complications, other than small effusions, are rare. Severe interstitial pneumonia leading to an adult respiratory distress syndrome may occur³ and fatal cases have been reviewed.⁴ Lung abscesses may develop but these usually resolve rapidly.⁵ Long term respiratory sequelae are few. There has been one report of diffuse interstitial fibrosis,⁶ one of Swyer-James (Macleod's) syndrome (unilateral hyperlucent lung),⁷ and one of obliterative bronchitis (Stevens-Johnson syndrome was also present in the initial illness).⁸ Two previous reports^{9, 10} have implicated *M pneumoniae* in the pathogenesis of bronchiectasis, the most recent¹⁰ presenting a similar picture of mycoplasma pneumonia and Stevens-Johnson syndrome in a young man.

The second rise and fall of mycoplasma titres might have been an anamnestic response but the clinical illness and height of titre would support reinfection. Interestingly, Stevens-Johnson syndrome was not again provoked, as in Stevens-Johnson syndrome associated with recurrent herpes simplex infection. Polyarthrits (unlike arthralgia) occurs rarely with *M pneumoniae* infection, affects the larger joints, and mostly resolves rapidly. It is thought to be due to immune complex deposition or an autoimmune reaction, although the organism has been isolated from acutely inflamed joints.

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