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Mycoplasma pneumonia – an unusual cause of acute myocarditis in childhood

Gouder M Formosa, M Bailey, C Barbara, C Muscat, and V Grech
Paediatric Department, St. Luke's Hospital, Guardamangia, Malta
Contact information: Dr. Victor Grech, Paediatric Department, St. Luke's Hospital, Guardamangia - Malta ; Email: victor.e.grech@gov.mt

Abstract

Mycoplasma pneumoniae is primarily a respiratory pathogen but may affect exhibit a diverse range of presentations from asymptomatic infection to life threatening conditions. Myocarditis of varying severity is an unusual complication. We report a 6 year old with mycoplasma myocarditis, a rare age for such a presentation, and who responded well to treatment with no sequelae. Serological testing for *Mycoplasma pneumoniae* should be part of the routine work-up for myocarditis.

MeSH: Mycoplasma Infections, Myocarditis

Introduction

Mycoplasma is the smallest free-living organism and is unique among prokaryotes in that it lacks a cell wall. This feature is largely responsible for particular biologic properties including lack of a Gram stain reaction and nonsusceptibility to many commonly prescribed antimicrobial agents, including beta lactams. *Mycoplasma pneumoniae* is a common pathogen that first linked to respiratory infections in 1898 when Roux and Nocard isolated the organisms from bovine pleuropneumonia specimens. Initially Mycoplasma species were thought to be viral in nature because of their size, and this was disproved by the presence of both mycoplasma DNA and RNA. *Mycoplasma pneumoniae* has an unusually small genome (800Kb)[1] and is an obligatory parasite. *Mycoplasma pneumoniae* can be communicated through close personal contact via respiratory droplets and has an increased prevalence in autumn and winter.[2]

Mycoplasma pneumoniae is predominantly a respiratory pathogen but may also involve other systems. The incidence of non-respiratory manifestations of *Mycoplasma. pneumoniae* varies greatly. Exanthems and gastrointestinal symptoms are quite common,[3] whereas *Mycoplasma*-associated carditis (myo- or pericarditis) is an uncommon complication, occurring in only 1-5% of patients. We report a child with acute myocarditis due to *M. pneumoniae* and analyse all documented cases of Mycoplasma infection in our hospital over a one year period.

Case report

Our patient, a 6 year old boy, was admitted to hospital in July 2004 with a 4 day history of pyrexia up to 104°F, headache, loose stools and lethargy together with a one day history of shortness of breath. He had been started on miocamycin (a newer macrolide) by his general practitioner one day prior to admission. He is a known asthmatic on prophylactic inhaled steroids.

On admission he was very pale, sweaty and tachypnoiec with a temperature of 99.8°F and a regular heart rate of 155bpm. The first and second heart sounds were normal and there was a loud third sound. Blood pressure was 78/52mmHg. The chest was clear and there was mild hepatomegaly and no rashes. Oxygen saturation was 97% in air. His leukocyte count was normal and his C-reactive protein was significantly increased (223mg/L). A chest X-Ray showed cardiomegaly and pulmonary oedema and an echocardiogram showed decreased fractional shortening (25%) and mitral incompetence. There were no effusions and no vegetations.

A working diagnosis of myocarditis with decompensated shock was made and he was started on peripheral dobutamine and transferred to intensive care where he was ventilated, intravenous ceftriaxone was added and oral miocamycin continued. He developed a persistent maculopapular rash, ceftriaxone was changed to meropenem and the rash subsided. Blood, urine and CSF cultures were negative and an ELISA test for *Mycoplasma Pneumoniae* IgM was positive. Coxsackie A, Coxsackie B, and echovirus antibodies were negative.

Over the next few days, the child's condition improved, the shortening fraction increased from 25% to 40% over a ten day period and mitral regurgitation disappeared. He was extubated after 6 days and dobutamine was stopped after 4 days. Miocamycin was stopped after fourteen days of treatment when the C-reactive protein had returned to normal. He was discharged on enalapril, low dose frusemide and regular inhaled budesonide. Six months later, he was off all treatment other than inhaled steriods for asthma.

Mycoplasma infections in St. Luke's Hospital

A review of all cases of documented Mycoplasma infections in our hospital in the year 2004 showed no other cases of carditis, 29 cases of respiratory infections, 13 cases investigated for pyrexia of unknown origin and 5 miscellaneous cases.

Discussion

Mycoplasma carditis is an uncommon entity, especially in the paediatric age group. Mycoplasma infection affects mostly children and adolescents but the mean age of patients with Mycoplasma carditis in the literature is higher (32 years).[4,5] It is thought that pneumonia with associated myocarditis is commoner in adults while extrapulmonary manifestations, such as arthritis and central nervous system complications, are commoner in the paediatric age group.[6]

The clinical spectrum of respiratory infections caused by *M. pneumoniae* ranges from a complete absence of symptoms to frank pneumonia.[3] Only 7-11% of patients develop pneumonia, while 5-20% may develop pleural effusion.[7,8] However, in patients with mycoplasma carditis, the rate of pneumonia and pleural effusion is higher (43% and 19% respectively).[5] Alternatively, some of the cases of Mycoplasma-carditis manifesting without pneumonia may remain undiagnosed and classified as idiopathic carditis.[9]

The ECG is a sensitive and inexpensive tool for the initial evaluation of suspected cases since the diagnosis of carditis may be unclear due to lack of specific cardiac symptoms. For example, tachycardia was found in only 43%, and chest pain in 38% of the patients with carditis. In contrast, ECG abnormalities were found in 100% of reported ECGs.[5] Complete heart block due to Mycoplasma carditis has also been reported.[10] Echocardiography is naturally useful to quantify cardiac function and monitor progress.

ELISA (enzyme linked immunosorbent assay) based methods are the laboratory methods of choice and utilize an enzyme immunoassay for *Mycoplasma pneumoniae* specific IgM. This IgM is found in 80% of *Mycoplasma pneumoniae* cases within 1 week of infection. Culture of *Mycoplasma* species usually requires 1-2 weeks[3] and is successful in only 40-90% of cases.[6]

Successful treatment of Mycoplasma carditis hinges on promptly considering Mycoplasma as a potential etiologic agent, obtaining proper diagnostic tests for its detection, and providing appropriate antimicrobial coverage. The lack of a cell wall renders B-lactam antibiotics useless. Macrolides are the agents of choice as they inhibit mycoplasma growth, possibly by blocking dissociation of peptidyl tRNA from ribosomes, causing RNA-dependent protein synthesis to arrest. Tetracyclines and fluoroquinolones may be considered in older patients.

Long-term sequelae are not infrequent in *Mycoplasma* carditis. A significant reduction in cardiac sequela has been observed. In the earlier review done by Ponka et al in 1979, 43% of the patients had cardiac sequelae, whereas Potasman et al in 2003 described a complication rate of less than 30% and this may have been due to better antibiotic coverage.[5]

Conclusion

M. pneumoniae is a ubiquitous and protean infection. Respiratory syndromes other than pneumonia caused by this organism are reported to be 10-20 times more frequent than pneumonia. Since most patients with *Mycoplasma* carditis present solely with respiratory symptoms, an ECG may be a useful screening test in suspected cases of mycarditis. Appropriate and timely treatment is essential and therefore serological testing for *Mycoplasma pneumoniae* should be part of the routine work-up of myocarditis.

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