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CASE STUDY

RARE CASE REPORT OF MYCOPLASMA RELATED POST-MYOCARDITIC CARDIOMYOPATHY AND A BRIEF REVIEW OF LITERATURE

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ABSTRACT

We report second case of mycoplasma related postmyocarditic cardiomyopathy in adults which is perhaps the second case report in literature After the case report of by Kyung Rim Huh[43] 2008 in Korea . A 56 year old male presented with fever, running nose, sore throat and bodyache since 15 days and 2 days prior to admission he developed pedal oedema, progressive dyspnoea on exertion with palpitation. He recovered very well with antibiotics, diuretics, anti-hypertensives and short steroid therapy. Microbial myocarditis treatment is not clear, antibiotics and steroids can be of great help in reducing the sequelae of dilated cardiomyopathy.

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INTRODUCTION

Mycoplasma pnemoniae mainly causes respiratory tract infections in person 5-20 years of age. Mycoplasma pnemoniae is predominantly a respiratory pathogen but may also involve other systems. The incidence of non-respiratory manifestations of mycoplasma pnemoniae varies greatly. Mycoplasma-associated carditis (myo or pericarditis) is an uncommon complication, occurring in only 1-5% of patients. Myocarditis is an inflammatory disease of the frequently resulting from viral infections and/ or post-viral immune mediated responses. It is one of the important causes of dilated cardiomyopathy worldwide. There is some evidence that immunosuppressive and immunomodulating therapy are effective for chronic, virus-negative inflammatory cardiomyopathy.

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We report a rare case showing mycoplasma related post-myocarditic cardiomyopathy.

Case Report

A 56 year old male, not a known case of any major illness, farmer by occupation, presents with fever, running nose, sore throat, bodyache for which he got treatment from private practinior. After 15 days he developed pedal oedema, dyspnoea on exertion with palpitation. Palpitation were present since fever started. Patient was referred to us with above condition and ultimately he was sent to emergency department and admitted to intensive care unit. From there the patient was discharged as a case of ischemic heart disease with dilated cardiomyopathy with congestive cardiac failure with pneumonia. On clinical examination, the patient was averagely built with weight of 55 kgs and he had lost 5 kgs of weight in 15 days. Patient was conscious, oriented, febrile with pulse rate of 120/ min, blood pressure of 90/60 mm of Hg, respiratory rate 32/min, tempature was 39.2^0c.

Mild pallor and jaundice was observed, there was redness of throat. Chest auscultation showed left basal crackles. He had frank failure and dyspnoea at rest. We kept him with history of febrile illness followed by cardiac symptoms. We suspected of viral myocarditis going for cardiac failure. Laboratory evaluation revealed normal leucocyte count (8500/mm^3), haemoglobin of 9.5g/dl, platelet count was 1,60,000/ mm^3. Glucose in blood chemistry 110 mg/dl, total protein 7.2g/dl (6.5-8.5), albumin 3.8g/dl (2.5-4.5). Liver function tests, kidney functions were deranged and lipids were normal. As we suspected of viral myocarditis and his blood was send for mycoplasma pnemoniae specific IgM (ELISA) which turn to be positive. ECG showed heart rate 120 beats/min with PR depression. Chest radiographs of PA view showed left basal pnemonitis. USG Abdemen suggestive of hepatomegaly. 2DECHO showed evidence of dilated left ventricle with ejection fraction reduced to 20%.

Clinical suspicion of mycoplasma related post-myocarditic cardiomyopathy was confirmed. We treated him with Macrolides (Azithromycin or Clarothromycin or Doxycycline) for 3 weeks, standard heart failure regimen including Beta – blockers, diuretics, tab. Ivabradin 5mg twice a day, angiotensin receptors blockers (ARBs) and steroids. Intravenous methyl prednisolone was given for 3 days as pulse. After 3 days, symptoms improve markedly in the form of reduction in the heart rate and blood pressure normalisation (110/80 mm of Hg). He was put on oral steroids and gradually tapered in a course of 6 weeks. After improvement in blood pressure, he was put on minimal doses of beta blockers and ARBs. Patient improved in 1 week with a given treatment and was discharged and under regular follow-up. After 2 months, his coronary angiography was done at larger centre as advised by cardiologist and found to have absolutely normal coronaries.

At present, the patient is without any complaints and given low dose of beta blocker and ARBs. His 2DEHO was done recently which is suggestive of marked improvement in systolic function. We hereby describe a rare case report of mycoplasma related post-myocardotic cardiomyopathy.

DISCUSSION

Mycoplasma is the smallest free-living organism. Mycoplasma pnemoniae is a common pathogen that first linked to respiratory infections in 1898 when Raux and Nocard isolated the organisms from bovine pleuropnemonia specimens (Himmelreich *et al.,* 1996). Mycoplasma pneumonia can be communicated through close personal contact via respiratory droplets and has an increased prevalence in winter (Cimolai, 1998). Mycoplasma pneumonia infection is usually on, sometimes causing high incidence of infection is likely the highest incidence in age from 5 and 20 years of age increased in late childhood and youth (Baum, 2005). 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 (Koskiniemi, 1993). The clinical spectrum of respiratory infections caused by M. Pnemoniae ranges from a complete absence of symptoms to frank pneumonia (Baum, 1979). Only 7-11% of patients develop pneumonia, while 5-20% may develop pleural effusion (Fine *et al.,* 1970). However, in patients with mycoplasma carditis, the rate of pneumonia and pleural effusion is higher (43% and 19% respectively) (Pazs and Potasman, 2002). Alternatively, some of the cases of Mycoplasma-carditis manifesting without pneumonia may remain undiagnosed and classified as idiopathic carditis (Karjalainen, 1990). Myocarditis is an inflammatory disease of the myocardium caused by different infectious and noninfectious triggers.

Etiology	Subgroups Examples
Infectious	Bacterial: *Chlamydia, Corynebacterium diphtheria, Legionella, Mycobacterium tuberculosis, Mycoplasma, Staphylococcus, Streptococcus A, Streptoccocus pneumoniae*
	Fungal: *Actinomyces, Aspergillus, Candida, Cryptococcus*
	Helminthic: *Echinococcus granulosus, Trichinella spiralis*
	Protozoal: *Toxoplasma gondii, Trypanosoma cruzi*
	Viral: Adenoviruses, Echoviruses, Enteroviruses (e.g., Coxsackieviruses), Herpes Viruses (Human Cytomegalovirus, Epstein-Barr virus, Human Herpesvirus 6), Hepatitis C Virus, Human Immunodeficiency Virus (HIV), Influenza A virus, Parvovirus B19
	Rickettsial: *Coxiella burnetti, Rickettsia typhi*
	Spirochetal: *Borrelia burgdorferi, Leptospira, Treponema pallidum*
Autoimmune diseases	Celiac disease, Churg-Strauss syndrome, Crohn's disease, dermatomyositis, giant cell myocarditis, hypereosinophilic syndrome, Kawasaki disease, lupus erythematodes, lymphofollicular myocarditis, rheumatoid arthritis, sarcoidosis, scleroderma, ulcerative colitis
Hypersensitivity reactions to drugs	Penicillin, ampicillin, cephalosporins, tetracyclines, sulfonamids, antiphlogistics, benzodiazepines, clozapine, loop and thiazide diuretics, methyldopa, smallpox vaccine, tetanus toxoid, tricyclic antidepressants
Toxic reactions to drugs	Amphetamines, anthracyclines, catecholamines, cocaine, cyclophoshamide, 5-fluorouracil, phenytoin, trastuzumab
Toxic	Ethanol
Others	Arsenic, copper, iron, radiotherapy, thyreotoxicosis

Table 1. Etiology of Myocarditis

In 1995, myocarditis was defined by the World Health Organization (WHO)/International Society and Federation of Cardiology (ISFC) as an inflammatory disease of the heart muscle, diagnosed by established histological, immunological, and immunohistochemical criteria (Richardson *et al.,* 1995). Myocarditis often results from common viral infections and post-viral immune-mediated responses. In patients with human immunodeficiency virus infection, myocarditis was observed in <50% of performed autopsies (Anderson *et al.,* 1988). Furthermore, myocarditis can be triggered by nonviral infections, for example, with *Borrelia burgdorferi* (Lyme disease), *Corynebacterium diphtheriae*, or *Trypanosoma cruzi* (Chagas disease) (Hidron *et al.,* 2010). Numerous medications like antipsychotics (e.g., clozapine) (Kilian *et al.,* 1999) antibiotics (penicillin, ampicillin, sulfonamides, tetracyclines) (Stelts *et al.,* 2008) can induce hypersensitivity eosinophilic myocarditis, which commonly is reversible after withdrawal of the causative agent. Eosinophilic-lymphocytic myocarditis may also occur after smallpox vaccination (Murphy *et al.,* 2003). Systemic autoimmune diseases such as Churg-Strauss syndrome (Vinit *et al.,* 2010) or hypereosinophilic syndrome (Loeffler's disease) (Corssmit *et al.,* 1999) can be associated with eosinophilic myocarditis.

Myocarditis is regarded as a precursor of dilated cardiomyopathy (DCM), which is currently the most frequent reason for heart transplantation (Maron *et al.,* 2006). Murine models of enteroviral myocarditis suggests that the course of viral myocarditis is characterised by 3 phases (Kawai, 1999). First, the entry of the virus into the myocytes is mediated through a specific receptor.Second phase is characterized by activated virus-specific T lymphocyte, which may target the host's organs by molecular mimicry. Cytokine activation (tumour necrosis factor-alpha, interleukin (IL-1and-6) and antibodies to viral and cardiac proteins may aggravate cardiac damage and cause impairment of the contractile function. Last chronic phase, which is characterized by myocardial remodelling and development of DCM (Liu and Mason, 2001).

Polymerase chain reaction test is use by Bernet (Bernet *et al.,* 1989) for the first time in 1989 by detecting Mycoplasma pneumonia. Since been widely used because of its high sensitivity, but polymerase chain reaction test alone are not enough to diagnosis Mycoplasma pneumonia. Mycoplasma pneumonia are respiratory specimen can be detected without the antibody response to several months in some patients during convalescence.

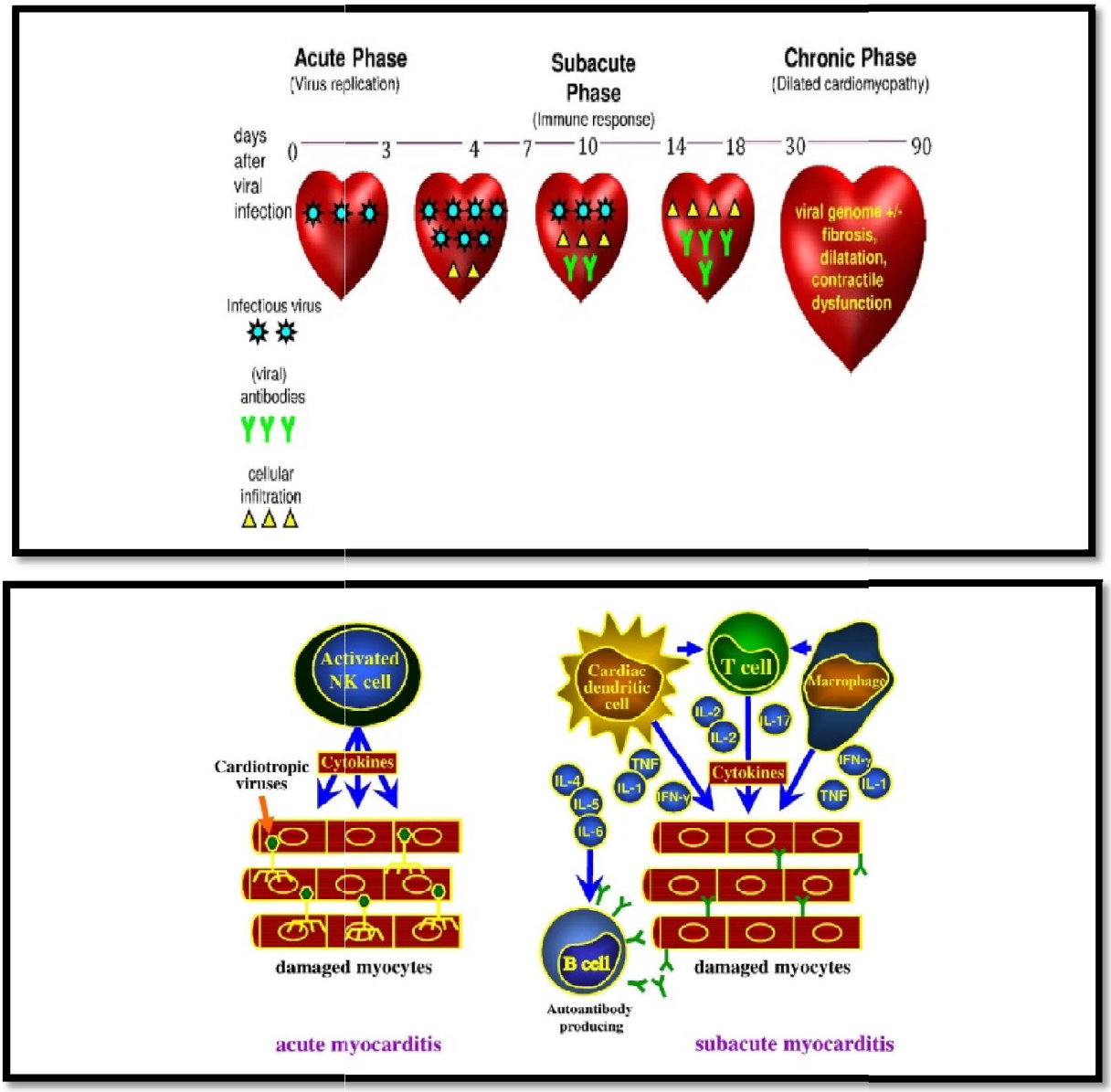

Fig 1 & 2: Showing Pathophysiology of Myocarditis

Thus pneumonia can be helpful in early diagnosis and treatment to consider with clinical evidence of infection with Mycoplasma polymerization chain reaction results (Daxboeck *et al.,* 2003). ELISA (enzyme linked immunosorbent assay) based method are the laboratory methods of choice and utilize an enzyme immunoassay for Mycoplasma pneumonia specific IgM. This IgM is found in 80% of Mycoplasma pneumonia within 1 week of infection. The electrocardiogram (ECG) is widely used as a screening tool despite low sensitivity (Morgera *et al.,* 1992). The ECG findings in patients with myocarditis vary from nonspecific T-wave and ST-segment changes to ST-segment elevation mimicking an acute myocardial infarction (Cooper, 2009). Also, atrial or ventricular conduction delays as well as supraventricular and ventricular arrhythmias can occur in patients with inflammatory heart disease (Pauschinger *et al.,* 2006). Echocardiography allows the evaluation of cardiac chamber sizes and wall thickness as well as systolic and diastolic function in patients with myocarditis. Especially before an EMB procedure, echocardiography is needed to exclude pericardial effusion and intracavitary thrombi, which have been noted in up to 25% of patients (Blauwet and Cooper 2010). Cardiovascular magnetic resonance (CMR) imaging has evolved as a noninvasive and valuable clinical tool for the diagnosis of myocarditis.

The gold standard in diagnosis of myocarditis is still the EMB. According to the Dallas criteria, acute myocarditis is defined by lymphocytic infiltrates in association with myocyte necrosis. Borderline myocarditis is characterized by inflammatory infiltrates without evidence of myocyte necrosis (Aretz, 1987). The Dallas criteria are limited by the high interobserver variability in interpreting biopsy specimens (in particular with regard to borderline myocarditis) and because noncellular inflammatory processes cannot be detected. Thus, immunohistochemistry is gaining further acceptance in the diagnosis of myocarditis. Monoclonal antibodies allow the characterization and localization of the mononuclear cell infiltrates (Baughman, 2006).

However, when performed by experienced interventionalists, left and right ventricular EMB are safe procedures, with a major complication rate of < 1%. Recent studies demonstrated not only the diagnostic but also the prognostic value of EMB in patients with suspected myocarditis (Kindermann *et al.,* 2008). The short duration of treatment is likely to recur in Mucoplasma pneumonia infection is to administer a macrolide, since there will be atleast two weeks. Macrolides are the agent of choice as they inhibit mycoplasma growth, possibly by blocking dissociation of peptidyl t RNA from ribosomes, causing RNA-dependant protein synthesis to arrest. Tetracycline and fluroquinolones may be considered in older patients (Radisic *et al.,* 2000). Specific types of myocarditis based on autoimmunity are treated with immunosuppression and combined treatment with immunosuppressants (cyclosporine and corticosteroids)with or without azathioprine may improve the poor prognosis, and yield a median survival time of 12 months compared with 3 months for untreated affected patients (Cooper, 2008). Withdrawal of immunosuppression can results in recurrent and sometimes fatal giant cell myocarditis.

In case of cardiac sarcoidosis, early immunosuppressive therapy with high-dose corticosteroids has been associated with improved cardiac function. The prognosis of patients with treatment is variable, with a 5 year survival ranging from 60% to 90%. Specific treatment options for viral myocarditis are not established yet (Kim *et al.,* 2009). By early initiation of rennin angiotensin blockade, chronic maladaptive cardiac remodelling can be attenuated, and the progression to dilated cardiomyopathy can be reduced (Godsel *et al.,* 2003).Diuretics are used to prevent or to treat fluid overload. Torsemide reduced the progression of myocarditis to DCM by decreasing fibrosis, myocyte sizes, and myocardial protein levels of transforming growth factor-beta-1, collagen III, and aldosterone synthase, beyond its renal effects (Veeraveedu *et al.,* 2008).

Beta-blocker treatment should be avoided in the acute phase of decompensated HF and in the very early treatment of fulminant myocarditis Beta-blocker may prevent arrhythmia and reduces cardiac work and improves ventricular function, reduces hospital admission for worsening HF, and increases survival. Experimental data suggest that the type of beta-blocker has an impact in inflammatory cardiomyopathy (Dickstein *et al.,* 2008). Administration of aldosterone antagonists is recommended for systolic HF patients with persistent NYHA functional class II to IV symptoms. Aldosterone antagonists reduced hospital admission for worsening HF and increased survival in addition to established HF therapy. Anti-inflammatory effects of eplerenone on murine viral myocarditis were shown by inhibition of mast cell-derived proteinases and resulted in an improvement of myocardial remodeling by suppressing fibrosis (Xiao *et al.,* 2009).

High doses of digoxin increased myocardial production of pro-inflammatory cytokines and worsened myocardial injury. Digoxin may limit the maximal tolerated dose of betablocker due to bradycardia or heart block. Therefore, digoxin should be avoided in patients suffering from acute HF induced by viral myocarditis (Matsumori *et al.,* 1999). Nonsteroidal anti-inflammatory drugs (NSAIDs) and colchicines are applied for anti-inflammatory treatment of pericarditis as a “nonspecific” anti-inflammatory therapy, whereas there is no indication for application in patients with myocarditis (Lotrionte *et al.,* 2010). Temporary pacemaker insertion is indicated for patients with acute myocarditis who present with symptomatic atrioventricular (AV) block II or III (Semmler *et al.,* 2010). Insertion of an implantable cardiac defibrillator (ICD) in patients with myocarditis is indicated after cardiac arrest due to ventricular fibrillation or after symptomatic ventricular tachycardia. Cardiac resynchronization therapy with defibrillator function is indicated for patients with impaired LV function (LV ejection fraction _35%) and left bundle branch block (Dickstein *et al.,* 2007). For patients with cardiogenic shock due to acute fulminant myocarditis who deteriorate despite optimal medical treatment, mechanical circulatory support or extracorporeal membrane oxygenation may be required to bridge the patient to recovery or heart transplantation (Mirabel *et al.,* 2011).

The target of immunoadsorption is the elimination of anticardiac antibodies against various cardiac cell proteins, which have been identified in patients with DCM and myocarditis (Felix *et al.,* 2002). In chronic DCM, azathioprine and prednisone resulted in an improvement of LV function (Wojnicz *et al.,* 2001). The prognosis of patients with myocarditis depends on clinical presentation, different clinical parameters, and EMB findings. Patients with acute myocarditis and preserved LV ejection fraction have a good prognosis with a high rate of spontaneous improvement without sequelae (Blauwet and Cooper, 2010). In a study by Kindermann *et al*. (Kindermann *et al.,* 2008) , the prognostic role of EMB, with detailed analysis of myocardial specimens including immunohistochemical staining for characterization of inflammation and molecular pathological analysis for detection of viral genome, was examined in 181 patients with suspected myocarditis. A risk stratification approach based on biopsy results, clinical findings, and drug treatment demonstrated that patients in NYHA functional class III or IV with positive immunohistology and without beta-blocker therapy have the poorest prognosis, with a 5-year transplantationfree survival rate of only 39%.

Conclusion

Mycoplasma pneumonia is an uncommon cause of myocarditis related cardiomyopathy, After the case report of by Kyung Rim Huh (Kyung Rim Huh *et al.,* 2008) 2008 in Korea, We report second case of mycoplasma related postmyocarditic cardiomyopathy in adults which is perhaps the second case report in literature. Myocarditis is an under-diagnosed cardiac disease resulting from a broad range of infectious, immune, and toxic causes. Affected patients may recover, develop DCM, or die. Immunomodulating and immunosuppressive therapy have been effective, only in chronic, virus negative inflammatory cardiomyopathy. Microbial myocarditis treatment is not clear, antibiotics and steroids can be of great help in reducing the sequelae of dilated cardiomyopathy.

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