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CASE REPORT

A Case of a 14-Year-Old Girl Who Developed Dermatomyositis Associated with *Mycoplasma pneumoniae* Infection

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Extrapulmonary manifestations of *Mycoplasma pneumoniae* infection are not uncommon and involvement of every organ system has been reported. However, association of inflammatory myositis with *M. pneumoniae* infection is rare. Here, we describe a patient who developed polymyositis associated with mycoplasma infection, who was treated successfully with glucocorticoid, intravenous immunoglobulin, and methotrexate.

Keywords: Dermatomyositis; *Mycoplasma pneumoniae;* Polymyositis

INTRODUCTION

Dermatomyositis (DM) is characterized by progressive proximal muscle weakness and distinctive skin manifestations. DM is a humorally mediated vasculopathy, which shows more B cells, complement deposition in perivascular regions on muscle biopsy. Although the etiology of DM is not fully understood, infection is supposed to be associated with initiation of inflammation, particularly in juvenile dermatomyositis (JDM) [1,2]. Association of various infectious organisms (streptococcus pyogenes, human immunodeficiency virus, coxsackievirus, and more) with DM has been reported, but the association with *Mycoplasma pneumoniae* infection is rare. Here, we report on a case of JDM after mycoplasma infection.

CASE REPORT

A 14-year-old girl presented with a 2-month history of progressive weakness and myalgia, and a 2-week history of fever in March 2014. She was ill-looking and complained of fatigue and myalgia. On admission, her vital signs were as follows: blood pressure was 100/60 mm Hg, pulse was 80 beats/min, respiratory rate was 18 rate/min, and body temperature was 38.0°C. Laboratory examination showed hemoglobin 10.2 g/dL, white cell count 8,810/mm^3 (neutrophil 76.1%, monocyte 2.0%, lymphocyte 20.0%, and eosinophil 1.9%), platelet count 526,000/mm^3, erythrocyte sedimentation rate 73 mm/hr (reference range, 0 to 30 mm/hr), increased serum C-reactive protein 16.3 mg/L (reference range, 0.01 to 3.0 mg/L), abnormal liver function test (serum aspartate aminotransferase 69 IU/L and alanine aminotransferase 47 IU/L), markedly increased creatinine kinase (CK) 1,578 IU/L (reference range, 50 to 200 IU/L) and myoglobin levels 432.2 mg/L (reference range, 25 to 75 mg/L), and serum lactic dehydrogenase 376 IU/L (reference range, 0 to 250 IU/L). Antinuclear antibody was positive with both a speckled and cytoplasmic pattern (1:80). Further extractable nuclear antibody testing was negative except anti-Ro antibody (>600 EU). Urine analysis was normal *M. pneumoniae* immunoglobulin M (IgM) antibodies 1.8 index (reference range, 0 to 0.9 index) and immunoglobulin G (IgG) antibodies >100 AU/mL (reference range, 0 to 12 AU/mL) were also elevated. Clarithromycin was started at a dose of 500 mg, and her myalgia and fever were improved the next day with decreased serum CK levels (1,108 IU/L). However, one week later, her myalgia and weakness were reappeared, and muscle enzymes was increased again (CK, 1,793 IU/L), albeit *M. peumoniae* IgM and IgG antibodies were decreased (1.4 index, 25.6 AU/mL).

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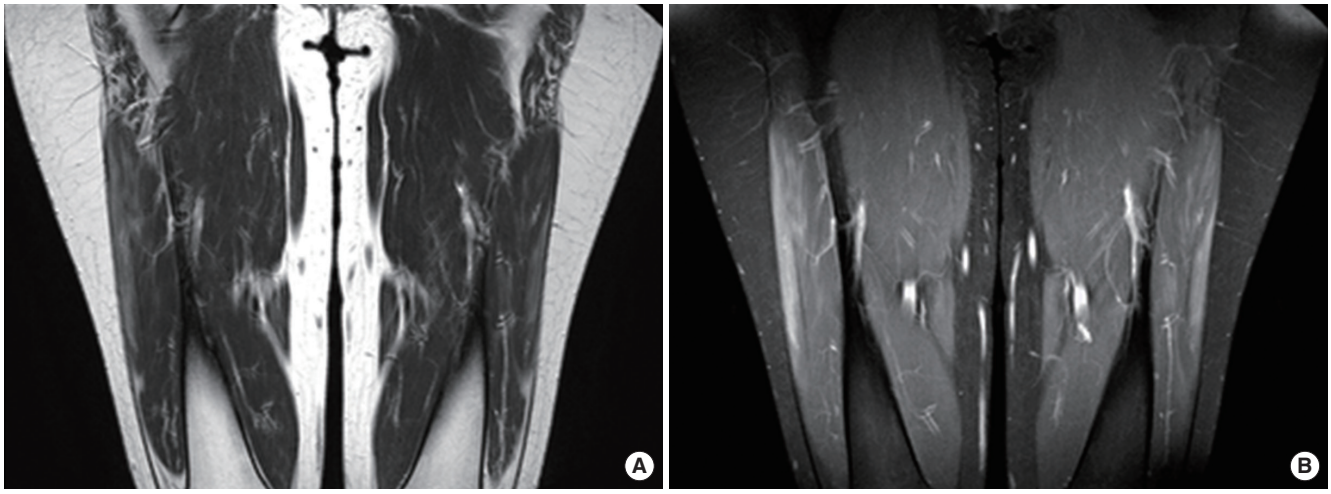

Fig. 1. Coronal section of both thigh magnetic resonance imaging showed symmetrical increased signal intensity on both vastus lateralis muscles; more prominently observed in (B) fat suppressed T1 weighted image than (A) T1 weighted image.

Her proximal muscle strength was decreased with grade III for both upper extremities and grade IV for both lower extremities. She also complained of a pruritic rash on both thighs, upper arm, and posterior scalp with a prominent heliotrope rash. On magnetic resonance image scans, increased signal enhancement was detected in both vastus muscle groups, rectus femoris, and gracilis muscle on fat suppressed T2-weighted images (Fig. 1). Electromyogram from the right thigh showed a myogenic pattern of polyphasic and short, small motor unit potentials. Muscle biopsy obtained from the left vastus lateralis muscle showed endomysial lymphocytic infiltration with muscle degenerations/regenerations and perivascular lymphocyte infiltrations (Fig. 2). On the 9th day of antibiotic treatment, she complained of resting dyspnea and her chest X-ray showed increased right pleural effusion. The chest CT scan showed subpleural and peribronchial consolidation and ground-glass opacity (GGO) in both lungs and bilateral pleural effusions (Fig. 3A). Treatment with intravenous immunoglobulin (1 g/kg/day for 2 days) was started, followed by high dose prednisolone 1 mg/kg. Her myalgia and proximal muscle weakness recovered gradually (grade V for all four extremities). Prednisolone was successfully decreased to 15 mg/day with methotrexate 15 mg/wk without symptom recurrence. Follow-up chest CT scan showed slightly improved subpleural and peribronchial consolidation and GGO without pleural effusion (Fig. 3B). However, methotrexate was switched to mycophenolate mofetil 1,000 mg/day after 3 months due to complaint of hair loss. With mycophenolate mofetil, she tapered her prednisolone to 5 mg qod (every other day) without recurrence.

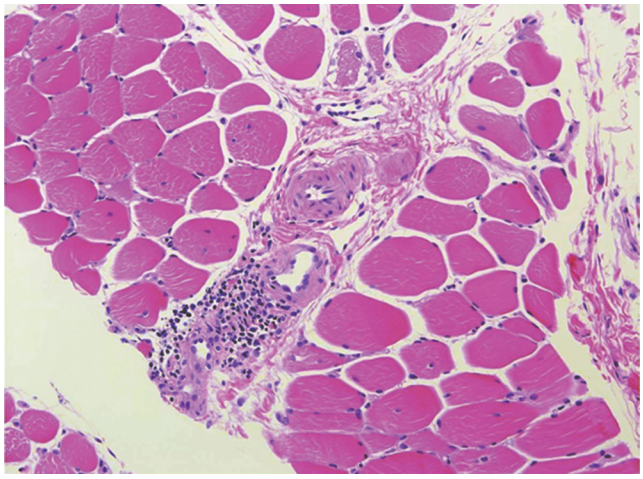

Fig. 2. Muscle biopsied from right vastus rateralis muscle showed endomysial lymphocytic infiltration with muscle degenerations/regenerations and perivascular lymphocyte infiltrations (H&E, ×100).

DISCUSSION

Idiopathic inflammatory myositis is a group of diseases characterized by progressive muscle weakness, which can be classified on the basis of distinct clinicopathologic features: DM, polymyositis, necrotizing autoimmune myositis, and inclusion-body myositis [3]. DM is the most common idiopathic inflammatory myositis with bimodal age distribution in both children (5-14 years of age) and adults (45-64 years of age). JDM has several clinical characteristics that are different from those of adult DM; vasculopathy, calcinosis, periungual and gingival telangiectasias, and ulceration are observed more frequently among JDM patients while co-existence

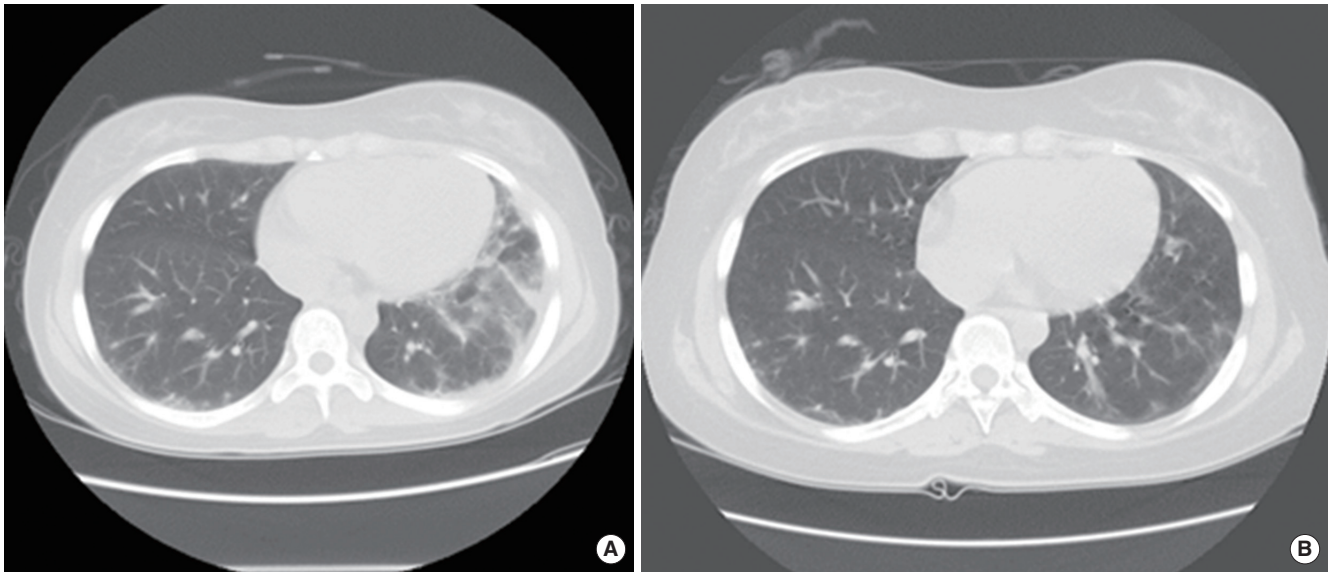

Fig. 3. (A) Initial chest CT scan showed subpleural and peribronchial consolidation and ground-glass opacity in both lungs and (B) bilateral pleural effusions, which had improved on follow-up CT scan approximately 3 months later. CT, computed tomography.

of malignancy, existence of myositis-specific antibodies, and interstitial lung disease are observed more frequently among adult DM patients. In addition, the prognosis of JDM is better than that of adult DM [4].

Although the etiology of DM has not been determined, infectious agents are supposed to be associated with the initiation of JDM. According to a previous study, more than half of children who developed JDM experienced respiratory symptoms including cough, sore throat, earache, rhinorrhea, cold, flu, otitis media, or lower respiratory illness (pneumonia, asthma, and bronchitis) [1]. Among those with constitutional and respiratory complaints, antibiotics were prescribed in 63% (100 of 159). Similarly, Manlhiot et al. [2] reported that more than one-third of patients had probable infection in the 3 months prior to JDM onset, and among them, clinically, respiratory infection was common, although pathogens were identified in only seven cases. Many cases of DM subsequent to various infections have been reported, including coxsackievirus [5-7], echorovirus [8], group A beta hemolytic streptococcus [9], human immunodeficiency virus [10], etc. One study reported that epitopes derived from homologous sequences shared between human skeletal myosin and Streptococcus M5 protein can stimulate T cells from patients with active JDM and induce various cytokines [11].

M. pneumoniae, one etiology of pneumonia among children, causes various extra-pulmonary manifestations including pericarditis, arthritis, and encephalitis [12,13]. These extrapulmonary manifestations have been explained by three pathomechanisms: direct effect, indirect effect, and vascular occlusion [12]. First, *M. pneumoniae* directly infects various organs and local cytokine induction results in extrapulmonary symptoms such as arthritis, otitis media, and pericarditis. Second, the components of *M. pneumoniae* such as lipoproteins, glycolipids, and glycoproteins, which resemble human cellular components, induce macrophage activation. Immune modulation by these activated macrophages results in indirect type of extrapulmonary manifestations such as autoimmune hemolytic anemia and erythema multiforme. Finally, cytokine and chemokine induction from the vessel wall by *M. pneumoniae* may cause vasculitic or thrombotic complications such as disseminated intravascular coagulation, stroke, and splenic infarction.

Development of inflammatory myositis following *M. pneumoniae* infection can occur by above mentioned pathogenesis. A few case reports written in English were found, but all cases were polymyositis [14-17]. Our case report suggests that not only polymyositis, but also JDM can develop as a result of *M. pneumoniae* infection. Thus, clinicians should put JDM on the list of differential diagnosis when a patient infected with *M. pneumoniae* complains of muscular weakness and rash.

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