

Henoch-Schönlein purpura associated with *Mycoplasma pneumoniae*

To the editor,

Mycoplasma pneumoniae is an important microorganism which causes respiratory infections in children, but it can also cause clinical pictures progressing with involvement of muscle-nerve, blood, heart, kidney, gastrointestinal system, bone-joint, skin and eye outside the respiratory system. Vasculitis is reported very rarely (1,2). Therefore, we aimed to present a case of Henoch-Schönlein purpura (HSP) associated with *M. pneumoniae* infection.

An eight year old female patient presented with nasal discharge, cough, vomiting, fever and skin eruption lasting for the last two weeks. It was learned that the patient had cough, fever and abdominal pain 2 weeks ago, a diagnosis of lobar pneumonia was made 8 days ago, ceftriaxone and clarithromycin treatment was started and the treatment was still continuing. Skin eruption appeared on the legs and trunk 10 days after the complaints started.

On physical examination, the body weight was found to be 32 kg (50-75th percentile), the height was found to be 137 cm (75-90th percentile), axillary temperature was found to be 39 °C and blood pressure was found to be 110/60 mmHg. Palpable purpura which did not wane by pressure was observed intensively in the lower extremities and gluteal region and less intensively in the upper extremities. Urticarial rash was observed in the proximal areas of the lower extremities and edema, erythema and increase in skin temperature were present in the right ankle. Crepitant rales were heard in the lower part of the right lung. Other system findings were normal.

Laboratory findings were as follows: hemoglobin:12 g/dl, white blood cells: 6300/mm³, platelets: 343 000/mm³, erythrocyte sedimentation rate: 66 mm/s, CRP: 53.1 mg/L, urea: 18 mg/dl, creatinine: 0.5 mg/dl, AST: 66 U/L, ALT: 31 U/L, urine pH: 6, urine density: 1015, urine sediment: 2 erythrocytes, one leucocyte. Involvement compatible with pneumonia in the right lower lobe was observed on postero-anterior lung graphy. Atypical pneumonia was considered in the patient and *M. pneumoniae* IgM was found to be positive.

The patient was hospitalized with a diagnosis of lobar pneumonia and HSP and clarithromycin treatment was continued. Abdominal pain started one day after hospitalization When bloody stool was observed severe gastrointestinal system involvement was considered and oral methylprednisolone treatment was started (2 mg/kg/day). Abdominal pain regressed on the second day of hospitalization and bloody stool regressed one week later. Oral steroid and clarithromycin treatments given for the diagnoses of *Mycoplasma pneumoniae* and HSP were continued. The patient had no health problem during follow-up.

M. pneumoniae is a microorganism displaying findings involving other systems in addition to the respiratory system. Narita (3) separated this effect of mycoplasma into three. The first is the direct effect of the microorganism, the second is the effect by immunologic mechanisms and the third is vasculitis and/or vascular obliteration. Vasculitis occurs as a result of both the second and third mechanisms.

Involvement of the systems other than the respiratory system by mycoplasma can lead to clinical pictures effecting many organs and systems. Main clinical findings include neurologic involvement (meningitidis, brain infarct, cerebellar ataxia, Guillian-Barre syndrome, transverse myelitis), joint involvement (polyarthralgia, monoarticular arthritis), hepatic involvement (anicteric hepatitis), hematologic involvement (hemolytic anemia, paroxysmal cold hemoglobinuria, hemolysis and renal failure), eye involvement (conjunctivitis, uveitis, papilla edema), cardiac involvement (pericarditis, endocarditis, arrhythmia, cardiac failure) and skin involvement (Stevens-Johnson syndrome, erythema multiforme, urticaria) (1,4). In addition, Kawasaki disease, microscopic polyangitis and Raynaud phenomenon may be observed (5-9).

M. pneumoniae associated vasculitis has been rarely reported. Despite Narita's (3) classification the pathogenesis of *M. pneumoniae*-related vasculitis is not known clearly. Some investigators think that the underlying pathology is type III hypersensitivity. It is proposed that circulating immune complexes accumulate in the vascular wall and effect specifically postcapillary venules (10). However, the extremely low number hinders scientific studies about pathogenesis.

Vasculitis related to mycoplasma has been reported to be observed with a low frequency and has been reported mostly as leucoclastic vasculitis in the literature (2,4,10-13). The first case of *M. pneumoniae* related vasculitis is a 13 year old boy reported by Van Bever et al. (11) in 1992. IgA, IgM and C3 accumulation was shown in the vascular wall in the skin biopsy in the patient who had acute respiratory failure syndrome (ARDS) secondary to diffuse lung involvement. Three of the other 5 cases are adults and two are children (2,4). The 3 adult cases include a 28 year old woman with polyarthritis and leucoclastic vasculitis reported by Perez et al.(12), a 75 year old man with encephalitis, pneumoniae, cutaneous leucoclastic vasculitis reported by Perez and Montes (10) in 2002 and a 51 year old woman with pneumoniae, cutaneous vasculitis and bilateral episcleritis reported by Jover et. al (13) in 2003. The pediatric cases include a 16 year old male patient with cutaneous vasculitis reported by Orlandini et al. (4) in 2004 and a 7 year old male patient with cutaneous vasculitis reported by Fillippo Greco et al.(2) in 2007. Our case is the fourth pediatric case in the literature.

Henoch-Schönlein vasculitis is the most common vasculitis observed in children and the exact cause is still not known. Main clinical findings include palpable purpura, abdominal pain, arthritis, gastrointestinal bleeding and nephritis. Various studies showed that it was related to infectious agents and especially beta-hemolytic streptococci (14-16). It is thought that a microorganism initiates vasculitis by triggering the immun system and leads to Henoch-Schönlein which is an immun complex vasculitis (16). In our case, vasculitis might have occurred by such a mechanism.

When searching for an etiologic agent in Henoch-Schönlein vasculitis, mycoplasma should also be considered.

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