

Tourette's Syndrome and Mycoplasma Pneumoniae Infection

The observance of streptococcal infection and Lyme disease in patients with Tourette's Syndrome led to the suggestion that either infection or a postinfectious immune process plays a role in the pathogenesis of Tourette's Syndrome.

Although Mycoplasma pneumoniae encephalitis can cause basal ganglia lesions and movement disorder, infection with Mycoplasma pneumoniae manifesting as Tourette's Syndrome has not yet been reported to our knowledge. We describe two patients with Tourette's Syndrome involving Mycoplasma pneumoniae infection.

Allen, a 7-year-old-boy, had suffered from slight motor (blinking and head shaking) and vocal (gurgling) tics since age 5. Three months before admission his tics increased in intensity and frequency and were accompanied by movement of his legs and arms. The results of a laboratory examination on admission showed normal routine blood values. The results of a lumbar puncture examination revealed CSF with 45 leukocytes/ μ l, 56 mg/dl glucose and 47 mg/ml protein. The results of all other bacterial, serological and viral tests were negative. The serum titer of antibodies to Mycoplasma pneumoniae tested by microparticle agglutination assay was found to be 1:80,000. The results of an immunoblot revealed positive immunoglobulin IgA, IgM, and IgG responses. Mycoplasma pneumoniae DNA was detected by a polymerase chain reaction assay of nasopharyngeal aspirate and a CSF examination. The authenticity of the DNA was confirmed by sequential analysis. In addition, Mycoplasma pneumoniae was cultured from Allen's nasopharynx and throat. Since Mycoplasma pneumoniae-induced deterioration of Tourette's Syndrome was suggested, treatment with erythromycin was begun (900 mg/day) and continued 4 weeks.

Allen's tics disappeared and the antibody titer to Mycoplasma pneumoniae decreased to 1:1,280.

Bailey, a 13-year-old girl, had had motor and vocal tics and an attention deficit disorder since age 4. At admission her motor tics included shaking of her head, arms and legs. Vocal tics comprised crying and coprolalia. The results of a blood examination revealed serum antibody titers to Mycoplasma pneumoniae (1:5,120) and positive IgA, IgM and IgG responses. The results of a CSF examination showed slight inflammatory signs (15 leukocytes/ μ l, 45 mg/dl protein, 49 mg/dl glucose) and a positive nested polymerase chain reaction for Mycoplasma pneumoniae DNA. In addition, Mycoplasma pneumoniae was cultured from Bailey's oropharynx. Therapy with erythromycin was begun (1600 mg/day). After 4 weeks of treatment, Bailey's antibody titer decreased (1:640) and there was marked clinical improvement of motor and vocal tics; however, signs of attention deficit disorder persisted.

Mycoplasma pneumoniae is known to cause extrapulmonary infections, including central nervous system (CNS) manifestations. CNS complications often occur without preceding or associated pulmonary symptoms. Reports of successful cultivation and a positive polymerase chain reaction with CSF indicate that Mycoplasma pneumoniae is able to penetrate the blood-CSF barrier. The inflammatory signs, immunological response, detection of Mycoplasma pneumoniae DNA in the CSF and prompt improvement of the symptoms of Tourette's Syndrome during antibiotic therapy for our two patients all point to encephalitis caused by Mycoplasma pneumoniae.

Tourette's Syndrome is believed to be the final common pathway of various disorders that have different etiologies. Infection with various agents, including Mycoplasma pneumoniae, should be considered a possible etiological factor in predisposed individuals. Immunological mechanisms have also been proposed for both Tourette's Syndrome and disorders appearing after infection with Mycoplasma pneumoniae. However, the underlying mechanisms remain unclear.

REFERENCES

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Norbert Müller, M.D. PH.D.
Michael Riedel, M.D.
Stefanie Förderreuther, M.D.
Christa Blendinger, M.D.
Marianne Abele-Horn, M.D.

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