Detection of Mycoplasma pneumoniae DNA in Cerebrospinal Fluid and Local Immune Response

Str—I read with great interest the report by Abele-Horn et al. [1] that described a patient with transverse myelitis due to Mycoplasma pneumoniae infection for whom PCR analysis of CSF was positive. One point that must be emphasized and further discussed is that the patient lacked respiratory symptoms, which have been considered a hallmark in M. pneumoniae infection.

In our previous study [2], using a nested PCR method, we demonstrated that the rate of PCR positivity for M. pneumoniae DNA in blood (serum samples) was significantly higher for patients without pneumonia than for patients with pneumonia. In addition, the rate of PCR positivity apparently increased with the progression of the antibody response (i.e., 17% positivity for patients with a 4-fold rise in antibody titers that were measured principally by a microparticle agglutination test, 28% positivity for patients with sustained high titers, and 50% positivity for patients with a fall in titers). This finding indicated that the occurrence of mycoplasmal bacteremia, which predisposes patients to nonrespiratory complications, is independent of humoral immunity (IgM or IgG antibody response) and suggested that local immunity (IgA antibody response or cellular immunity) in the respiratory tract may play a pivotal role in dissemination.

Local immunity to M. pneumoniae must be studied to understand the pathogenesis of nonrespiratory complications.

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References


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Reply

Str—With regard to our recently published case of transverse myelitis due to Mycoplasma pneumoniae that was diagnosed by serological methods and by nested PCR [1], Dr. Narita, the author of a study about mycoplasmal CNS infections [2] assumed that our patient lacked respiratory tract symptoms. In his previous study he describes (1) a significantly higher rate of PCR-positivity of serum samples from patients with extrapulmonary mycoplasmal CNS infections than from patients with pulmonary mycoplasmal infections and (2) a significantly high rate of PCR-positivity of serum samples from patients with high titers of IgM and IgG antibodies to M. pneumoniae.

From the first observation, Dr. Narita concluded that mycoplasmal bacteremia occurs frequently without pneumonia and may be a risk factor for CNS infections. On the basis of the second observation, he suggested that extrapulmonary mycoplasmal CNS infections develop independently from humoral antibody response (with IgG or IgM antibodies) and that local immunity in the respiratory tract (IgA response or cellular immunity) may play a pivotal role in dissemination.

I agree with the first point. Regarding the second point, I have some comments. It is true that the patient’s humoral antibody response cannot prevent an extrapulmonary mycoplasmal infection or a CNS infection occurring as a complication following pulmonary infection.

In our case, the patient developed transverse myelitis without respiratory disease, although the focus of the CNS infection was the patient’s oropharynx, from which we obtained culture samples that yielded a high level of M. pneumoniae. Furthermore, the infection occurred despite an adequate antibody response with IgA and IgG antibodies, although the IgM antibody response was inadequate. We inferred that the local immune response with IgA antibodies could protect a patient from an infection of the respiratory tract but not from myelitis.

These findings in this case were underlined by the results of a pilot study in which patients with mycoplasmal CNS infections were examined (unpublished data); one-third of these patients developed CNS infection after a pulmonary infection. At the beginning of the course of CNS disease, all patients’ cultures were negative for M. pneumoniae, but all PCR results were positive. All patients produced antibodies, as determined by the microparticle agglutination assay (Serodia-Myc II Kit; Fujirebio, Tokyo): 90% of patients produced IgM: 70%, IgA; and 100%, IgG.

The other two-thirds of the patients who had extrapulmonary infections also harbored the microorganisms in their oropharynx. They did not show any signs of respiratory tract infection. Furthermore, although all patients revealed high titers of IgG and IgA antibodies (100% and 90%, respectively), few had an IgM response (10%). It is not clear whether a lack of IgM antibodies is associated with a higher risk for extrapulmonary infections.

The present discussion assumes that mycoplasmal CNS infections are caused by viable organisms, but this has not yet been proved. It is unclear whether mycoplasmal CNS infections are caused by viable microorganisms, by an immune immune response, or by an autoimmune reaction.