Correspondence

Spondylitis and Arthritis Due to *Mycoplasma hominis:* The Case for Awareness in Undefined Pleuropneumonia

SIR—*Mycoplasma hominis* rarely causes pneumonia [1]. Because special culture media are required to detect *M. hominis* and are not often requested, the lack of clinical suspicion and/or detection may result in inappropriate treatment. As a consequence, the risk of bacterial seeding is increased, as is shown in the case we report.

A 48-year-old woman with a history of primary hypogammaglobulinemia was hospitalized because of osteoporotic fracture of the T12 vertebra. Her medical history included recurrent pneumonia, polyarthritis, and chondrocalcinosis of the right knee. Five weeks after admission to the hospital, she developed bilateral pneumonia (figure 1), which deteriorated to ventilator-dependent respiratory failure, despite treatment with amoxicillin-clavulanate followed by treatment with ceftriaxone plus clarithromycin. Results of cultures of blood, urine, and pleural effusion specimens were negative for pathogens; results of a urinary antigen test for *Legionella pneumophila* and a PCR assay for *Mycoplasma pneumoniae* were negative. *Citrobacter* species and *Candida albicans* grew from samples of tracheal aspirate. Hence, treatment was switched to imipenem-cilastatin plus fluconazole, and immunoglobulins (200 mg/kg) were substituted once a week. Pansinusitis caused by *Citrobacter* species was confirmed, and fluid was drained. However, the patient's condition did not improve, and pulmonary infiltrates persisted. Results of serological tests (for *M. pneumoniae, Chla-*

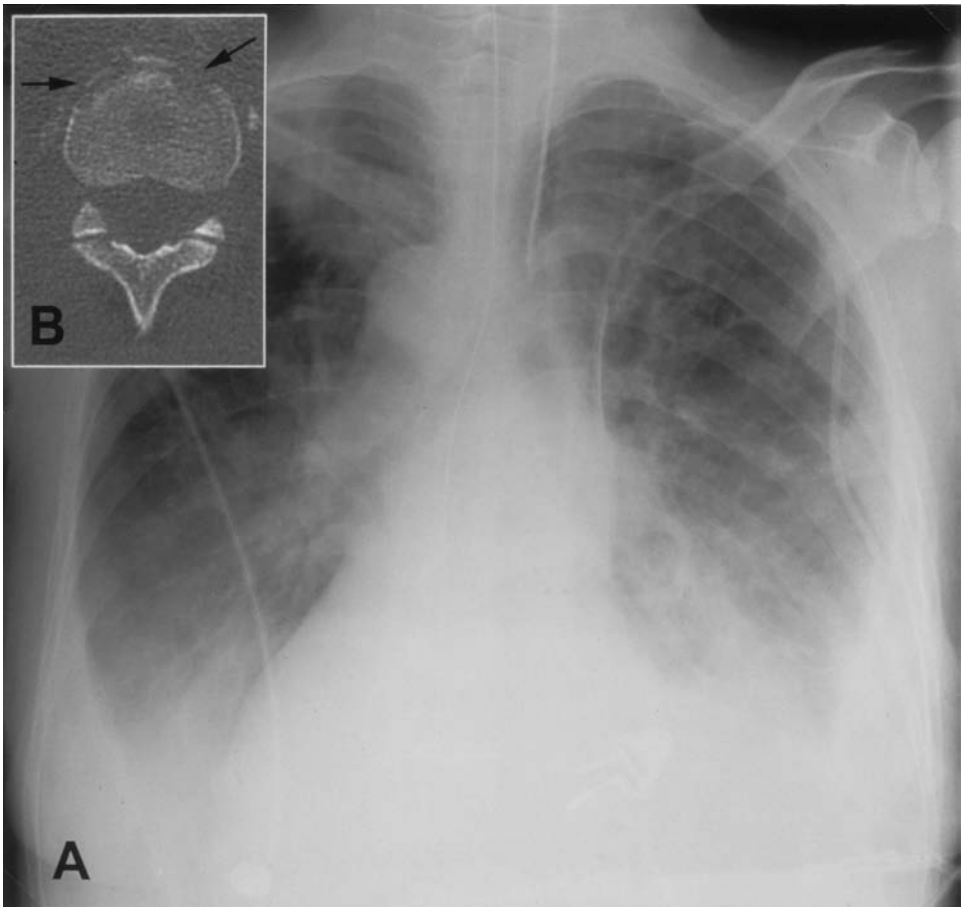

Figure 1. *A,* Conventional radiograph of the thorax showing bilateral pneumonia and pleural effusion. *B,* CT scan of the T12 vertebra showing erosions caused by spondylitis *(arrows)* and severe decalcification caused by osteoporosis.

mydia pneumoniae, Chlamydia trachomatis, Chlamydia psittaci, parvovirus B19, rubella, *Borrelia burgdorferi,* and HIV) were negative. Eight days after the patient underwent intubation, skin inflammation was observed at the level of the vertebral fracture. A CT scan showed erosions and severe decalcification (figure 1). Two weeks later, swelling of the right knee occurred. Arthrocentesis yielded 30 mL of cloudy yellow synovial fluid with 24,300 cells/μL (92% neutrophils) and calcium pyrophosphate crystals. Results of additional standard cultures and PCR for *B. burgdorferi* and *M. pneumoniae* were negative. Needle aspiration of the T12 vertebra was performed, and *M. hominis* was identified using broad-spectrum eubacterial PCR. In the meantime, *M. hominis* grew in cultures of samples of synovial fluid. Thus, systemic *M. hominis* infection with arthritis and spondylitis was diagnosed. Clinical and laboratory findings rapidly improved during treatment with doxycycline (200 mg/day), and pulmonary infiltrates regressed.

M. hominis is a commensal of humans and mainly causes genitourinary tract infection [2]. The patient described here had neither a recent infection nor recent manipulation of the genitourinary tract. Extragenitourinary infections caused by *M. hominis,* such as arthritis, spondylitis, brain abscess, meningitis, and respiratory tract infections have rarely been reported [3]. Most of these cases, much like the case we report, occurred in an immunocompromised host [1, 4]. In the case that we report, it is likely that the primary infection was pleuropneumonia due to *M. hominis,* because of the sequence of manifestations of infection and because of the favorable course of pneumonia only after administration of doxycycline. It is interesting that *M. hominis* was seeding to 2 foci that had previous pathologic findings—namely, the fractured vertebra and the knee that had active crystal synovitis—illustrating that these sites of inflammation represented a locus minoris resistentiae.

Antibiotic therapy for *M. hominis* is not standardized and depends on the type of infection. Tetracyclines are given mainly for bone and joint infections. However, resistant strains of *M. hominis* have been reported [5]. Clindamycin, erythromycin, and ciprofloxacin are documented alternatives but are not used as first-line therapy [1, 4]. In the case of culture-negative pleuropneumonia, clinical awareness of *M. hominis* is important, so that specific culture media can be requested and organ failure and bacterial seeding can be prevented.

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Parham Sendi,[1,2] Werner Zimmerli,[1] and Marc Michot[2]

[1]Basel University Medical Clinic, Liestal, and [2]Medical Intensive Care Unit, Kantonsspital, Aarau, Switzerland

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Reprints or correspondence: Dr. Parham Sendi, Basel University Medical Clinic, Rheinstrasse 26, CH-4410 Liestal, Switzerland (sendi-pa@magnet.ch).

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Chlamydia pneumoniae and Asthma in Children: Diagnostic Issues

SIR—We refer to the recent article by Biscardi et al. [1] on the association between atypical respiratory pathogens and asthma in children, specifically the methods used for determining *Chlamydia pneumoniae* infection. Although the study was primarily concerned with the role of *Mycoplasma pneumoniae,* PCR and serologic testing with an EIA were also performed for diagnosis of *C. pneumoniae* infection. But these tests have significant limitations. PCR testing for *C. pneumoniae* is not standardized. Although there are more than 18 such in-house tests described in the literature [2], none, including the assay used by Biscardi et al. [1], has been adequately validated by comparison with culture for respiratory-tract specimens. Recent data also suggest major problems with both inter- and intralaboratory reproducibility, especially with nested assays [3, 4]. The EIA used by Biscardi et al. [1] has also not been validated in comparison with culture or validated PCR for respiratory infection [2]. The Centers for Disease Control has recommended the microimmunofluorescence (MIF) assay as the serologic method of choice, although this assay is also subject to interlaboratory variability [5].

One issue not addressed at all is the poor correlation between *C. pneumoniae* serologic test results and results of culture for children. Emre et al. [6] and several other studies not cited [7–9] have documented that the majority of children with culture-documented *C. pneumoniae* infection are seronegative by MIF assay. This doesn't mean that these children do not have anti–*C. pneumoniae* antibody, but that the antibodies they do make are not detected by the MIF [9]. Because the EIA used by Biscardi et al. [1] (ELISA-Chlamydia, Savyon) uses whole *C. pneumoniae* elementary bodies as the antigen, one would expect results similar to the MIF assay, which also uses whole elementary bodies. As expected, results of EIA sero-