to accurately rule out the diagnosis of myocardial contusion. In contrast, because of its relatively high specificity, an elevated troponin T value may be of some importance in suspecting myocardial contusion.

Fifth, Dr. Radusky suggests that troponin I could be more useful than troponin T in the diagnosis of myocardial contusion. Since we did not measure it, we are unable to verify this point. Nevertheless, it should be pointed out that the sensitivity of troponin T was only 0.35. Thus, even if troponin I slightly increases this sensitivity, we are afraid that the improvement would not be of great enough magnitude to modify anything. This is the reason why, in the conclusion of our paper, we presented our concern for the unlikelihood of any biological diagnosis of myocardial contusion in the future. We would be happy to hear that our prediction is wrong.

Mustapha Ferjani, MD
Bruno Riou, MD, PhD
CHU Pitié-Salpêtrière
Paris

REFERENCE


Cytomegalovirus as a Pulmonary Pathogen

To the Editor:

I read with interest the article “Cytomegalovirus as a Primary Pulmonary Pathogen in AIDS” by Waxman and colleagues (January 1997). They reported that in some patients with AIDS, cytomegalovirus (CMV) represented a primary pulmonary pathogen. They also recommended that clinicians consider the diagnosis and the treatment with anti-CMV therapy. However, their findings didn’t completely establish an etiologic role of CMV as a primary pulmonary pathogen.

Generally, CMV infections are effectively controlled by the immune system without the ultimate clearance of the virus. It was reported that CMV evolved a unique mechanism for selectively limiting the presentation of the potentially immunogenic immediate-early (IE) protein, which might preclude IE-specific cytotoxic CD8+ lymphocytes from providing protective immunity to CMV infection.2 Van Laer et al3 suggested that during viremia, leukocyte populations could be infected with CMV and that polymorphonuclear leukocytes were a major target cell during active infection.

Waxman et al4 defined CMV pneumonitis as the universal presence of the following criteria: (1) positive CMV cultures from both BAL and transbronchial biopsy (TBB) specimens; (2) characteristic cytomegalic inclusion bodies from both BAL and TBB specimens; and (3) absence of any other pulmonary pathogen. However, as they reported, pulmonary CMV disease still remains a confusing disease in patients with AIDS because there are no definite criteria for diagnosis of the disease, and the number of CMV-specific inclusions on histologic study appears unrelated to clinical manifestations.4 A large prospective study is necessary to evaluate the diagnostic role of the criteria for CMV pneumonitis. It is also unknown how Waxman et al5 proved the absence of other viral infections.

The diagnosis of active CMV infection can be also established by isolating the virus from clinical specimens. Although the appearance of characteristic cytomegalic inclusion bodies is specific for CMV infection, generally it is very hard to detect from clinical specimens. Polymerase chain reaction (PCR) and other molecular biological assays for CMV provided a rapid, sensitive, and specific method to diagnose active CMV infection as viral DNA is present in the clinical specimens such as serum or BAL of patients infected with CMV.6 More sensitive and specific methods of diagnosis for active CMV infection may allow prospective longitudinal study.

Kei Numazaki, MD, PhD
Department of Pediatrics
Sapporo Medical University School of Medicine
Sapporo, Japan

Reprint requests: Kei Numazaki, MD, PhD, Dept of Pediatrics, Sapporo Medical University School of Medicine, S.I W.16 Chuokai, Sapporo 060, Japan
REFERENCES


To the Editor:

We appreciate the opportunity to address the questions raised by Dr. Numazaki. He points out that we did not completely establish the etiologic role of cytomegalovirus (CMV) in these nine cases of pneumonitis.1 We absolutely agree and point out that the retrospective nature of the study design does not allow for causality to be examined. In fact, one of the study objectives was to develop explicit criteria using information that would be available to most clinicians for this “clinical diagnosis,” so that it will be more feasible to collect prospective data. We agree that the establishment of diagnostic criteria for CMV pneumonitis in human immunodeficiency virus (HIV) infection represents a challenging task. However, we would claim that the strict criteria we used, “...the universal presence of: (1) positive CMV cultures from both BAL and transbronchial biopsy specimens; (2) cytopathological examination that documents the typical cytopathologic condition of CMV with characteristic enlarged cells with large pleomorphic nuclei and intranuclear and cytoplasmic inclusions; and (3) absence of any other pulmonary pathogen ...” in the clinical setting of dyspnea, hypoxemia, and interstitial infiltrates,2 distinguishes our study from previous studies, and represents the most rigorous approach to describing this clinical entity, within the limitations of available data.

Because CMV is frequently isolated from respiratory secretions of AIDS patients and is often found along with other pathogens such as Pneumocystis carinii,2 diagnosis is difficult. Moreover, without cytopathologic changes it is unclear if CMV represents a pathogen.2 All of our patients had cytopathologic changes documented, absence of any other pathogen documented, and the clinical criteria noted above. As detailed in our methods, all samples were cultured for all viral pathogens, not just CMV. We do agree, as we stated in our discussion, that to address more adequately causality, diagnosis, and outcome, a large prospective study is required.

Finally, Dr. Numazaki makes the point that the diagnosis of CMV infection can be established by isolating the virus from clinical specimens. We would argue that if the diagnosis of pneumonitis is being considered, that approach is not adequate. In bone marrow transplantation the culture of CMV from BAL fluid is fairly reliable in identifying infection.4 This has not been the case in HIV-infected patients in whom recovery of virus without cytopathologic evidence of invasion does not necessarily represent disease.5 He suggests polymerase chain reaction (PCR) as a more rapid, sensitive, and specific diagnostic test. Although PCR data were not available for our patients, we would argue that the specificity of PCR for clinical CMV pneumonitis might potentially be low. As mentioned above, CMV is frequently isolated from patients who do not have active pulmonary disease. The benefits of a very sensitive test would need to be weighed relative to the consequences of more false-positives resulting from the unavoidable tradeoff of lower specificity. Furthermore, it is unclear that we even know what the operating characteristics of PCR are for CMV pneumonitis. While the use of PCR has been studied in AIDS patients with retinitis6 and CNS disease,7 it has not been studied in patients with pulmonary disease. We suggest that the use of our diagnostic criteria may also help to prospectively study the performance of PCR and other diagnostic tests.

Sue J. Goldie, MD
Harvard School of Public Health
Department of Health Policy and Management
Boston
Aaron B. Waxman, MD, PhD
Department of Internal Medicine
Section of Pulmonary and Critical Care Medicine
Yale University School of Medicine
New Haven, Connecticut

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Community-Acquired Pneumonia

Need for a Cost-Effective Approach to Treatment

To the Editor:

Patients with mild community-acquired pneumonia (CAP) can be treated as outpatients, but more severe cases of pneumonia require inpatient treatment with parenteral antibiotics. Siegel and colleagues (October 1996)3 demonstrate the usefulness of short-term parenteral therapy, which would help reduce the period of hospitalization and cost of therapy for these patients. This need to cut costs is even more relevant in developing countries. Emergence of drug-resistant bacteria results in an additional financial burden.2,8 In countries like India, second- and third-generation antibiotics are unaffordable to a large number of patients attending state hospitals. We conducted a study of patients admitted to a community-based hospital in Bombay to assess, in particular, the effectiveness of penicillin and ampicillin in the treatment of inpatients with moderate to severe CAP. There were 20 patients, 15 male and 5 female, between 15 and 71 years of age. Immunocompromised, chronically debilitated, or otherwise bedridden patients were not included.