Disseminated Infection Due to Mycobacterium avium-intracellulare Complex

To the Editor:

The letter by Savage and Dellinger entitled "Disseminated tuberculosis caused by M intracellularre" (Chest 1982, 82:900-01) describes a woman whose care in her terminal illness was directed by us. We would like to clarify their report and comment on the diagnosis of the acquired immune deficiency syndrome (AIDS) which was retrospectively made by others. Omitted from their letter was that on January 1, 1982, three days prior to discharge from Keesler Air Force Base Hospital, she was started on therapy with prednisone, 40 mg daily. This dose of prednisone was continued for six weeks, accompanied by the use of isoniazid and rifampin. When the dose was reduced to 30 mg daily, about February 26, 1982, fever recurred. She tapered the prednisone. Because of severe febrile and systemic symptoms, she sought care and was admitted to Schumpert Hospital (Shreveport, LA) on March 22, 1982 by one of us (AJT), two days after stopping prednisone. The positive culture for Mycobacterium intracellulare from the transbronchial lung biopsy and bronchial washings done in December, 1981 at Keesler Air Force Base Hospital was not known to the patient until after her admission to Schumpert Hospital.

During her final admission, bone marrow and liver biopsies showed granulomas with definite acid-fast bacilli. Some of the bone marrow granulomas were caseous. These specimens, as well as lung tissue cultured post-mortem, grew M avium-intracellulare complex. Her hospital course was inexorably downhill, despite therapy with isoniazid, rifampin, amikacin, cycloserine, pyrazinamide, ethambutol, and ethionamide, instituted shortly after admission. In addition, she received indomethacin, levamisole, and lymphocyte-rich leukocyte transfusions, most from donors with stable or resolved M avium-intracellulare complex infections, in an attempt to improve her cell-mediated immunity. None of these measures had a clinically significant effect upon her course and she died April 7, 1982. The post-mortem examination showed acid-fast bacilli in the lungs, lymph nodes, liver, and bone marrow. Dr. Albert Hand, pathologist, reported the granulomatous disease to be in a pattern of largely nonreactive, rapidly disseminated mycobacteriosis.

It is of note that there was no history of either bisexual behavior or drug abuse in her husband or the patient, as has been described for some other cases of disseminated M avium-intracellulare complex.\(^1\) This patient's illness might have ended fatally without therapy with steroids, but there is no doubt that the steroids are implicated in the progressive dissemination of her disease. She represents case 2543 of AIDS, registered at the Center for Disease Control (Atlanta, GA). While this case meets the general definition of a case of AIDS,\(^4\) it causes us to reflect that a fatal infection in a person who is otherwise normal with regard to cellular immunity should not be adequate for the diagnosis of AIDS, and that using this definition will call all cases of certain serious or disseminated infections of a variety of causes as AIDS, not leaving any without the AIDS designation. The result of this practice is predictable: the number of cases of AIDS will increase until all such cases are included, at which point the rise in the number of new cases will plateau, thus terminating the portion of the "epidemic" created in part by the case definition. The other result will be a loss of regard for the natural course of certain disseminated infections, the diagnosis of which automatically equates with AIDS. Accepting the present case as one of AIDS seriously challenges the conception of the case definition.

Burton C. West, M.D., Associate Professor of Medicine and Chief, Section of Infectious Diseases; and A. Judson Tillinghast, M.D., F.C.C.P., Clinical Assistant Professor of Medicine, Pulmonary Section, Louisiana State University Medical Center, Shreveport

Reprint requests: Dr. West, Louisiana State University Medical Center, PO Box 33932, Shreveport 71130

REFERENCES
2 CDC. Update on acquired immune deficiency syndrome (AIDS)—United States. MMWR 1982; 31:507-14

To the Editor:

Drs. West and Tillinghast make several points that deserve response. It is often difficult to understand reasons for therapeutic decisions without all the facts leading to those decisions. For the sake of brevity in our brief communication, many significant aspects of the patient's care were omitted. She was discharged 4 January 1982 after an extensive multisystem evaluation. Transbronchoscopic and open lung biopsies showed marked acute inflammation compatible with hypersensitivity pneumonitis. We never had any bacteriologic evidence of tuberculosis from these biopsies, sputum or other materials. A therapeutic trial of steroids was initiated. However, because of her positive PPD, radiographic findings, and one non-caseating granuloma demonstrated on bone marrow biopsy, we felt obligated to cover for tuberculosis with isoniazid and rifampin, at least until results of all cultures were negative. Even if cultures were negative, as long as significant steroid dosages were given, we recommended continuing isoniazid. There was never any reason to suspect disease caused by atypical mycobacteria. The suggestion that steroid therapy was continued despite disseminated disease with Mycobacterium intracellulare is inaccurate, since culture results were not available at the time of her discharge. We agree that a decision to add steroids to an antituberculosis regimen for disseminated tuberculosis would be controversial.

All culture material for tuberculosis is sent to our state laboratory for identification. We were notified of positive identification of M intracellulare in mid-March 1982, and our patient's referring military physician was contacted. Subsequently, when we learned from that physician that she was again hospitalized, we contacted her civilian physicians who informed us of her terminal stage of illness. We did