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TUBERCULOSIS AND HIV INFECTION: MORE THAN THE SUM OF THE PARTS

TUBERCULOSIS increases the human immunodeficiency virus (HIV) viral burden, accelerates the clinical course of HIV infection, and increases mortality in HIV-infected persons.[1,2] TB is a leading cause of morbidity and mortality in individuals infected with HIV. Indeed, the diagnosis of TB in an HIV-infected person is AIDS-defining.

Until the mid 1980's the number of TB cases in the United States steadily declined. The number of reported TB cases then leveled out and even increased in some parts of the country. (In Oregon, rates varied little between 1984 and 1995; but did increase notably last year.) Many experts believe that progress against TB slowed because of the AIDS epidemic.

Coinfections with TB and HIV are common. Among 20 clinics in the U.S. the median clinic HIV seroprevalence rate among persons with recently diagnosed TB was 3% (range, 0.3-46%).[3] HIV seroprevalence was higher among clinics in the Northeast and Atlantic states as well as among U.S. born patients and those with extrapulmonary TB. In Oregon, 3% of newly diagnosed TB cases were also reported as HIV positive between 1988-1990. In this article we discuss how more recent and more complete surveillance data have given us an improved understanding of the interaction of these two diseases.

In 1993, questions about HIV infection status began to be asked routinely (and, most of the time, answered) of new TB cases. We reviewed surveillance data submitted to the Health Division (OHD) from 1993-96 to evaluate HIV screening of TB cases, to estimate the number of TB-HIV coinfections, and to determine the number of Oregonians whose HIV infection was found as a part of their TB evaluation.

Local health departments (LHD) report new TB cases to the OHD, including information about HIV infection status (as determined by serologic testing or client history). For each TB patient identified as being coinfected with HIV, we abstracted the AIDS-defining illness information and dates of first positive HIV tests from the OHD AIDS registry. HIV infection recognized for the first time at the time of TB diagnosis was defined as the first positive HIV test within one month of TB diagnosis. Rates were compared using simple rate ratios.

In addition to identifying coinfections from routine surveillance reports, we also cross-checked the TB registry with persons named in the AIDS case registry to identify persons who may not have been identified otherwise.

RESULTS

From January 1993 through December 1996, 664 cases (annualized incidence: 5.3/ 100,000) of TB were reported in Oregon. Of these, 535 (81%) were culture-confirmed, 115 (17%) were clinical diagnoses only, and 14 (2%) had tissue that appeared infected with *Mycobacterium* spp., a clinical course consistent with TB, but were not culture-confirmed. HIV status was determined for only 353 (53.2%) of these patients. Of the 311 cases for whom HIV status was not determined, 166 (53%) were not offered testing, 14 (5%) refused testing, 124 (40%) were stated to be "unknown," and for 7 (2%) the HIV question was unanswered.

Age was strongly associated with documentation of HIV status. New TB patients who were 20-59 years old were three times more likely to have had their HIV status documented than younger or older cases (71% vs. 24%). Other groups that were more likely to have their HIV status determined included: injection drug users, persons living in the metropolitan Portland area, persons using excessive alcohol, homeless persons, and men. Foreign-born patients or patients with extrapulmonary TB were not more likely to have had their HIV status determined.

Of 353 persons with TB for whom HIV status had been determined, 26 (7.4%) were HIV-infected. Risk factors for HIV infection among tested TB patients included: injection drug use, middle (!) age [20-59 years old], male sex,* being born in the U.S., and having extrapulmonary TB. No missed opportunities for HIV diagnosis were identified. Our cross-checking of TB and AIDS registries turned up no additional coinfections (i.e., the list of coinfected patients derived from routine surveillance was complete). Twelve of the 26 persons had AIDS prior to TB but, for four patients previously known to be HIV-infected, TB was the AIDS-defining illness. For these 16 persons the time between the first positive HIV test and the diagnosis of TB ranged from 3 to 130 months (median, 29 months).

Of 337 persons with TB without a previous history of HIV infection, 10 (3%) were newly recognized as being coinfected with HIV (nine had positive *M. tuberculosis* cultures). The median age was 33 (range, 24-67 years); all were male. Four were reported to use injected drugs and were homeless at the time of diagnosis. None of the others were known to be homeless or drug/alcohol abusers. Six were U.S.-born; three were from Mexico; one was from Central America.

DISCUSSION

Because TB may be one of the first infections seen in HIV-infected persons,[4] newly diagnosed TB can be an important sentinel event in the identification and treatment of HIV-infected persons. In Oregon from 1993-96, despite the fact that HIV status was not determined for nearly half of the new cases of TB, at least 10 persons—3% of those tested—had newly identified HIV infections. These figures should not be extrapolated to those not tested, however. Some physicians may be quite good at assessing the HIV infection status of their patients with newly diagnosed TB by history or even just by eye.

* as in having a Y chromosome.

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Others may prefer to rely on test results. In general, we recommend the latter.

Knowing the HIV status of your TB patients has important benefits. TB and HIV are strongly associated and can be thought of as interacting synergistically. HIV-infected patients often need longer TB treatment regimens. Most importantly, TB can be the "yellow flag" that leads to the discovery of an unsuspected HIV infection, opening the doors to the benefits of anti-retroviral treatment, preventive therapy for other opportunistic infections, and counseling to reduce HIV transmission. It is also worth noting that significant interactions have been noted between rifampin and protease inhibitors; an i.d. consult is recommended when devising a drug regimen for coinfected patients. All things considered, HIV screening is strongly recommended for every new case of TB—regardless of a provider's perception of risk.

Oregon is typical of many states where the prevalence of HIV infection and the incidence of TB are relatively low. Nonetheless, the rate of coinfection in Oregon in 1993-96 was higher than rates reported from most western TB clinics surveyed in 1988-89.[3] Furthermore, this rate may be increasing in Oregon. The rate of TB cases coinfected with HIV for 1993- 96 (7.6%) was more than twice the 1988-90 rate in Oregon. (Some of this increase may be due to changes in methodology.) In this setting, routine surveillance appeared as sensitive and specific as matching TB and AIDS registries to identify TB-HIV coinfected persons.

Unlike previous surveys at large TB clinics, reviewing population-based TB surveillance data that included "HIV Status" allowed us to assess the rates of HIV and TB coinfection in a low TB incidence area and among persons who were more likely to be seen in a physician's office or at local health departments rather than at tuberculosis clinics. Despite differences in the populations and the methods compared to previous surveys,[3] we noted similar trends among persons with recently diagnosed TB. Persons born in the U.S. were more likely to be coinfected with HIV. And, although most TB-HIV coinfected persons had pulmonary TB, HIV infection was more common in persons with extrapulmonary infections.

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Influenza Season to Open with Important Change

EFFECTIVE NOVEMBER 1, the Oregon State Public Health Laboratory will accept throat wash specimens for its seasonal influenza lab surveillance program. Last season the lab ran 684 specimens, with exactly 100 positives. A review of data from previous years suggests a simple way to reduce the number of specimens processed without significantly reducing our ability to track viral activity. Simply put, influenza recovery is unlikely in specimens collected ≥4 days after clinical onset. Overall, 584 (19.1%) of 3061 specimens tested in the 1991–97 seasons were positive for influenza. Only 7.4% of specimens collected ≥4 days after were positive, however, compared to 22.5% of fresher specimens (odds ratio 3.6; $p < 0.00001$).

Accordingly, specimens will only be accepted if they were collected within 3 days of clinical onset, and that fact is clearly indicated on the requisition slip. We regret the inconvenience this may cause some of you, but think this is the best way to ensure the overall quality of the data this program provides.

Interestingly, although Oregon-specific data are less complete, anecdotal information and a careful review of the literature suggest that the probability of viral recovery is also enhanced by obtaining specimens from patients who have so-called "influenza-like illness." The presence of this syndrome (temperature ≥38.3°C, cough, myalgia, and two or more of the following: headache, sore throat, rhinorrhea, malaise, chills, prostration) should be verified before specimens are collected.

Throat wash kits will be available from the OSPHL (osphl.ohd@state.or.us; 503/229-5882) or from your local health department. Lab slips should be marked "rule out influenza." Specimens should be kept cool and processed promptly. If transit time to the OSPHL will exceed 24 hours, specimens should be sent with a cold pack (but not frozen).