INT J TUBERC LUNG DIS 7(7):665–672

Extensive transmission of *Mycobacterium tuberculosis* among congregated, HIV-infected prison inmates in South Carolina, United States

S. I. McLaughlin,* P. Spradling,† D. Drociuk,‡ R. Ridzon,§ C. J. Pozsik,‡ I. Onorato¶

*** National Immunization Program, Global Immunization Division, † National Center for HIV, STD and TB Prevention, Division of Tuberculosis Elimination, Centers for Disease Control and Prevention, Atlanta, Georgia; ‡ Department of TB Control, South Carolina Department of Health and Environmental Control, Columbia, South Carolina; § HIV, TB and Reproductive Health, Bill and Melinda Gates Foundation, Seattle, Washington; ¶ National Center for HIV, STD and TB Prevention, Division of HIV/AIDS Prevention, Centers for Disease Control and Prevention, Atlanta, Georgia, USA**

SUMMARY

BACKGROUND: In August 1999, a prison inmate infected with the human immunodeficiency virus (HIV) was diagnosed with pulmonary tuberculosis (TB). This source patient lived in a prison dormitory housing over 300 HIV-infected men, and was symptomatic for at least 2 months prior to diagnosis. We report a large outbreak of TB in HIV-infected prison inmates with subsequent transmission of *Mycobacterium tuberculosis* outside the prison.

METHODS: Exposed inmates were screened by symptom review, chest radiograph and tuberculin skin test (TST) in September and December 1999. We recorded CD4 cell counts, viral loads and receipt of highly active antiretroviral therapy (HAART).

RESULTS: The source patient lived on the right side of a two-sided dormitory exclusively housing HIV-infected men. Of 114 men tested from the right side, 75 (66%) had documented TST conversions. Of 96 converters overall, 82 (85%) had TSTs measuring ⩾15 mm. Within 6 months of diagnosis of TB in the source patient, 30 additional inmates and a healthcare worker who cared for the source patient developed TB disease. Two other inmates developed TB disease in spring of 2001.

CONCLUSIONS: We describe extensive transmission of *M. tuberculosis* in a group of HIV-infected prison inmates with high TST conversion rates and subsequent transmission in the community. In settings where HIV-infected persons are congregated, the consequences of TB outbreaks are magnified.

KEY WORDS: tuberculosis; outbreak; HIV; prison; congregation

SINCE 1980, the number of incarcerated persons in state and federal correctional facilities in the United States has increased dramatically. From 1980 through 1999, the number of prisoners increased by 380%, from 329 821 to 1 254 577.[1] With rising numbers of inmates, correctional facilities have become important foci of tuberculosis (TB) and human immunodeficiency virus (HIV) morbidity. Incarcerated populations have disproportionately high rates of both TB and HIV infection and are a group requiring the highest priority for TB screening and control.[2–6] Prison systems have reported TB rates as high as 100 to 2283 cases per 100 000 persons[2,3,7] compared to 5.8/100 000 in the general US population in 2000.[8] In 1999, US prisoners had more than five times the rate of AIDS compared to the US population.[9,10] Persons infected with HIV are at high risk for rapid development of TB disease once infected or reinfected with *Mycobacterium tuberculosis*.[11–13] Congregation of HIV-infected prisoners magnifies the risk of *M. tuberculosis* transmission, which can spread within the prison and into the community. TB outbreaks have been reported in US prisons,[14–16] but few document outbreaks among HIV-infected prisoners.[17,18] We report on a large outbreak of TB in HIV-infected prison inmates, with subsequent transmission to the community. This outbreak is notable because it highlights the special risks of congregating HIV-infected prison inmates, and demonstrates strikingly high rates of tuberculin skin test (TST) conversion in an HIV-infected cohort.

METHODS

Definitions

A positive TST was defined as an induration of 5 mm or more and a conversion as an increase in induration

Correspondence to: Steven I McLaughlin, National Immunization Program, Global Immunization Division, Centers for Disease Control, 1600 Clifton Rd NE MS E-05, Atlanta, GA 30333, USA. Tel: (+1) 404-639-1970. Fax: (+1) 404-639-8573. e-mail: SWM4@cdc.gov

Article submitted 30 September 2002. Final version accepted 13 February 2003.

of 5 mm or more in a previously TST-negative person.[4,13] A case of active TB was defined by laboratory or clinical criteria. Laboratory-confirmed case patients were those in whom *M. tuberculosis* was isolated from a clinical specimen or by nucleic acid amplification test (Accuprobe Mtb complex, Genprobe, San Diego, CA). A clinical case was a case patient with signs and symptoms of TB (abnormal, unstable chest radiograph or clinical evidence of TB disease), a completed diagnostic evaluation and treatment with at least two anti-tuberculosis medications. Isolates from all culture-positive cases had DNA fingerprinting performed using IS*6110* restriction fragment length polymorphism analysis.[19] All case patients, regardless of TST status, and all TST converters were classified as infected. All persons with previously positive TSTs (i.e., $\geqslant$5 mm) were considered possibly reinfected. Exposed inmates were those who lived in dormitory A at any time between 1 May and 30 September 1999, excluding the source patient. This period encompassed the start of the source patient's infectious period, up to the last date a potentially infectious case patient left dormitory A. Highly active antiretroviral therapy (HAART) was defined as combination antiretroviral therapy containing at least one protease inhibitor, or one non-nucleoside reverse transcriptase inhibitor plus one or more nucleoside reverse transcriptase inhibitors, or a combination of abacavir, lamivudine and zidovudine, or any combination of antiretroviral therapy with protease inhibitors. Inmates were classified as receiving HAART if they were on therapy in September 1999.

Screening

All eligible inmates at the prison underwent routine annual TST screening in May 1999. Inmates entering after May 1999 had TST performed during the intake process. Because of the diagnosis of TB in the source patient in August 1999, all inmates at the prison with a prior negative TST were skin tested again in September and December 1999. TSTs were performed with 5 TU of purified protein derivative (PPD) (Tubersol, Connaught Laboratories, Swiftwater, PA) by experienced nurses using the Mantoux method. Techniques for administration and reading skin tests were observed by State Health Department staff. All inmates exposed in dormitory A, regardless of TST status, received a medical evaluation, medical record review, chest radiograph and treatment for latent TB infection (LTBI). For those with clinical signs, symptoms or an abnormal chest radiograph, sputum smears and cultures were obtained.

Statistical analysis

We used the Kruskal-Wallis *t*-test and the Mantel-Haenszel χ^2 test to evaluate for differences between inmate characteristics on each side. Bivariate statistical analyses were performed with Epi Info software.[20] Multivariate logistic regression was performed using LOGISTIC software.[21]

RESULTS

Source patient

The source patient was a US-born 34-year-old African American male diagnosed with HIV infection in 1989 and incarcerated for 13 years between 1983 and 1999. He had a documented 15 mm TST in March 1984, but did not complete either of two attempted courses of treatment for LTBI in 1984 and 1987. This information was not clearly documented in his medical record. In July 1999, he presented at a community hospital with complaints of fever and abdominal pain. He had a 3-week history of productive cough, shortness of breath and weight loss. His TB infection status was not documented on his transfer record and chest radiographs taken during this hospitalization were normal. He was not in respiratory isolation during his 1-week hospitalization, and was discharged back to prison with a presumed diagnosis *Pneumocystis carinii* pneumonia. He was readmitted to the community hospital in late August 1999 with fever, productive cough and weakness and a 31-pound weight loss since June 1999. Follow-up chest radiographs were normal. Smears of sputum specimens demonstrated numerous acid-fast bacilli (AFB) (10–90/high power field) and a blood culture drawn in July grew *M. tuberculosis* susceptible to all first-line anti-tuberculosis medications. At the time of diagnosis he had a CD4 cell count of 17 cells/μL and had never received antiretroviral medication due to refusal. He had been symptomatic for approximately 9 weeks prior to diagnosis and isolation.

The source patient lived on the right side of dormitory A, one of three dormitories where only HIV-infected inmates are housed. Each dormitory has two sides, right and left. Inmates are not permitted to cross sides, but may socialize on their side of residence. Inmates from different sides may have contact at meals and recreation. The cells in each dormitory house two men. Air enters a cell from a vent over a solid door, leaves by flowing out under the door and exits the building via large ducts near the ceiling in a central common air space. Evaluations of airflow in 1998 and 1999 measured air circulation rates of 50 to 100 cubic feet per minute, two to five times the recommended rate.[22] Approximately 10% of dormitory air was estimated to recirculate through the air-cooling system at the time of the outbreak.

Exposed inmates

The exposed cohort of inmates was comprised of 323 men who lived in dormitory A during the exposure period: 161 lived on the right side at any time and 162 lived only on the left side. Characteristics of exposed men are listed in Table 1. Men from the right and left

Table 1 Characteristics of HIV-infected prison inmates exposed to *M. tuberculosis* 1 May–30 September 1999 ($n = 323$)

Characteristic	
Race, *n* (%)	
Black	289 (89)
White	33 (10)
Other	1 (1)
Age (years)	
Median	36
Range	21–57
Days exposed in dormitory A*	
Median	136
Range	1–152
CD4 cell count*	
Median (cells/μL)	420
Range	2–1505
CD4 <200 cells/μL, *n* (%)	39 (12)
Viral load (VL)*	
Median (copies/ml)	1129
Range	50–750 000
VL ⩽400 copies/ml, *n* (%)	128 (40)
On HAART in Sept. 1999, *n* (%)	185 (57)
Tuberculin skin test results, *n* (%)	
Converters	96 (30)
Nonconverters	132 (41)
Previous positive	66 (20)
Not screened	29 (9)
Converter data ($n = 96$)	
TST size range, mm	8–30
TST ⩾15 mm, *n* (%)	82 (85)
CD4 <200 cells/μL, *n* (%)	13 (14)
Total screened—*n* (%)	294 (91)

* Days exposed, CD4 and VL values not available for 1.

sides did not differ with respect to median age, median days exposed, median CD4 cell count, median viral load or receipt of HAART during September 1999. Of 323 exposed inmates, 294 (91%) completed a medical evaluation and TST screening (Table 1). Seventy-four were released prior to screening at the prison and required location and evaluation in the community; of these, 45 (61%) completed screening and 29 were lost to follow-up. These 29 unscreened men did not differ from screened men with respect to median age, median CD4 cell count, median viral load, or receipt of HAART. Sixty-six exposed inmates (43 right, 23 left) had previously positive TSTs and were not retested, but did undergo medical screening for TB.

Of 114 men evaluated from the right side of dormitory A with documented prior negative TSTs, 75 (66%) converted to positive TSTs. Of 114 men evaluated from the left, 21 (18%) converted to positive TSTs.

TST size ranged from 8 to 30 mm. Of 96 total converters, 82 (85%) had a TST ⩾15 mm. Thirteen converters had CD4 cell counts <200 cells/μL, and 12 (94%) had TSTs ⩾15 mm. TST size was not significantly associated with CD4 cell count, viral load or receipt of HAART. In addition to TST conversions, there was evidence of transmission of *M. tuberculosis* to another eight men with active TB disease (seven right, one left) who had either a previously positive ($n = 5$) or negative TST ($n = 3$).

Because annual skin testing had previously been performed in all six dormitories for all eligible inmates in May 1999, all men who subsequently developed positive TSTs during September and December 1999 were TST converters.

Among those exposed in dormitory A, infected men (TST converters and all case patients) had resided in dormitory A longer than the uninfected men (median days 148 vs. 132; $P = 0.00009$). Men exposed on the right side were more likely to convert to positive TSTs than men exposed only on the left side (RR = 3.6: 95% confidence interval [CI] 2.4–5.4). CD4 cell count, viral load and receipt of HAART were not significantly associated with infection (Table 2).

Table 2 Risk factors for *M. tuberculosis* infection in HIV-infected prison inmates who completed screening ($n = 233$)

Characteristic value	Infected* ($n = 104$)	Not infected† ($n = 129$)	RR	95%CI	*P* value
Median days exposed	148	132	—	—	0.00009
Median CD4 cell count	420	456	—	—	0.3
Side of dormitory					
Right	82	36	3.6	2.4–5.4	0.00001
Left	22	93			
CD4 cell count (cells/μL)					
CD4 <200	15	11	1.3	0.9–1.9	0.2
CD4 ⩾200	89	118			
Viral load (copies/ml)					
Viral load ⩽400	36	52	0.9	0.6–1.2	0.4
Viral load >400	68	77			
HAART‡					
HAART	60	67	1.1	0.8–1.5	0.5
No HAART	42	57			

* Infected = case patients plus converters.
† Not infected = non-converters, excluding patients with previously positive TST.
‡ On HAART September 1999. Analysis excludes 7 men for whom HAART status could not be confirmed.
RR = risk ratio; CI = confidence interval; HAART = highly active antiretroviral therapy.

Because of the complex and interactive nature of HIV and TB co-infection, we developed logistic regression models to examine the relative contributions of multiple risk factors to the risk of infection. The risk factor variables included viral load ≤400, CD4 cell count ≤200, HAART, right side residence, and ≥median days exposure. The best-fit logistic regression model for infection risk factors included only right side residence in dormitory A (adjusted odds ratio [AOR] 11.9: 95%CI 6.1–23.0) and greater than median days residence in dormitory A (AOR 3.3: 95%CI 1.7–6.4).

Case patients

Thirty additional cases were diagnosed in current or former inmates within 6 months of diagnosis of TB in the source patient in August 1999: two further cases were diagnosed by May 2001. Twenty-six case patients were diagnosed in the prison; six were diagnosed after release. Of the six men diagnosed outside the prison, five were released prior to detection of the outbreak and had not received treatment for LTBI. Twenty-eight case patients (88%) lived on the right side of dormitory A; four lived on the left (Figure). All case patients were HIV-positive males born in the US. Of 33 total inmate case patients (including the source patient), 21 were culture-positive for *M. tuberculosis* and 12 were diagnosed clinically (Table 3). Twenty-four had TST conversions from negative to positive. The source patient and five others were previously TST-positive; five were culture-confirmed. Three case patients had a negative TST. Of these, one had a CD4 cell count of 6 cells/μL and one had metastatic adenocarcinoma of the colon; both were culture-confirmed. The third case patient with a negative TST was culture-negative, but met the clinical case definition. All 21 available isolates had identical DNA fingerprints with nine matching bands.

One case patient, a former inmate diagnosed outside the prison in May 2001, had cavitary TB disease.

Table 3 Characteristics of prison inmates diagnosed with tuberculosis 1 August 1999–31 January 2000 (n = 33)

Characteristic	
Race, *n* (%)	
Black	33 (100)
Age, years	
Median	38
Range	23–53
Days exposed in Dormitory A*	
Median	131
Range	1–152
Cases by side in dormitory A, *n* (%)	
Right side	29 (87)
Left side	4 (13)
CD4 cell count	
Median (cells/μL)	360
Range	6–784
CD4 <200 cells/μL, *n* (%)	9 (29)
Viral load (VL)	
Median (copies/ml)	7000
Range	50–750 000
VL ≤400 copies/ml, *n* (%)	9 (29)
On HAART in Sept. 1999, *n* (%)	17 (52)
Tuberculin skin test results, *n* (%)	
Converters	24 (71)
Non-converters	3 (10)
Previous positive	6 (19)
Laboratory results, *n* (%)	
Sputum smear-positive†	14 (45)
Culture-positive	21 (61)
DNA fingerpring match	21 (100)
Drug resistant organism	0 (0)
Findings on chest radiography	
Normal	4 (12)
Adenopathy	17 (52)
Adenopathy + infiltrates	8 (24)
Infiltrates	3 (9)
Cavitary	1 (3)

* Excluding source case-patient.
† Acid-fast bacteria detected from bronchial lavagein one case-patient
HAART = highly active antiretorival therapy.

There were four case patients with normal chest radiographs; all had positive cultures for *M. tuberculosis* and three had sputum smears demonstrating

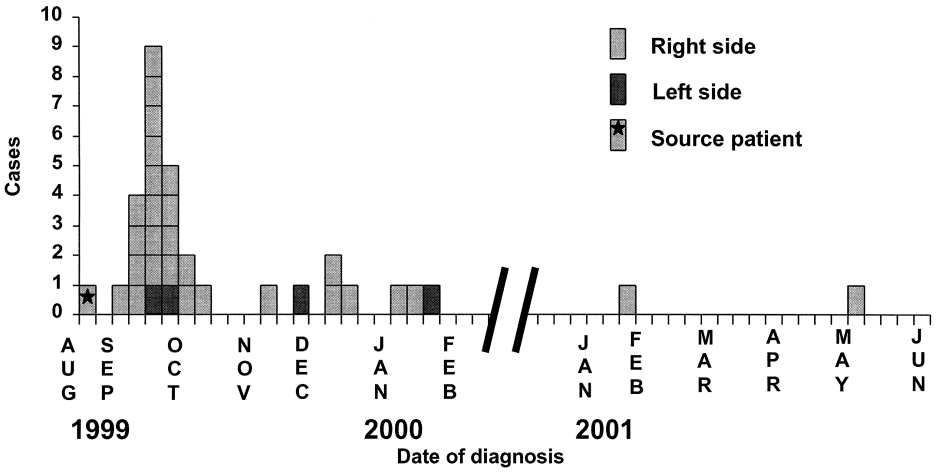

Figure Epidemic curve of tuberculosis cases in Prison Dormitory A by date of diagnosis, South Carolina 1999–2001.

Table 4 Risk factors for TB disease in HIV-infected prison inmates who completed screening ($n = 294$)

Characteristic	Value		RR	CI	*P* value
	Cases* ($n = 32$)	Non-cases ($n = 262$)			
Median days exposed†	131	150	—	—	0.06
Median CD4 cell count	360	438	—	—	0.02
Side of dormitory					
Right	28	129	6.1	2.2–17.0	0.00004
Left	4	133			
CD4 cell count (cells/μL)					
CD4 <200	8	25	2.6	1.3–5.4	0.009
CD4 ⩾200	24	237			
Viral load (copies/ml)					
Viral load ⩽400	9	106	0.6	0.3–1.3	0.2
Viral load >400	23	156			
HAART‡					
HAART	17	144	0.9	0.56–1.7	0.7
No HAART	15	111			

* Excludes source case.
† Days exposed not available for one.
‡ HAART September 1999. Analysis excludes 7 men for whom HAART status could not be confirmed.
RR = relative risk; CI = confidence interval; HAART = highly active antiretroviral therapy.

AFB. One of these case patients was asymptomatic at diagnosis, with a CD4 cell count of 28 cells/μL and numerous AFB on smear of a sputum specimen.

Case patients resided in dormitory A for a median of 131 days, compared to 150 days for non case patients ($P = 0.06$) (Table 4). Upon diagnosis, new case patients were removed from the dormitory and placed in respiratory isolation, shortening their exposure time. Men exposed on the right side of dormitory A were more likely to develop TB disease than men exposed only on the left side (RR 6.1; 95%CI 2.2–17.0). Case patients had a lower median CD4 cell count than non case patients (360 vs. 438 cells/μL; $P = 0.02$) and inmates with CD4 cell counts <200 cells/μL were more likely to become case patients compared to inmates with CD4 cell counts ⩾200 cells/μL (RR = 2.6; 95%CI 1.3–5.4). Viral load and receipt of HAART were not significantly associated with development of TB disease.

Other contact investigations

There were six prison dormitories in total, from which all 126 TST-eligible men from dormitory A and all 578 TST-eligible men from the other five dormitories were skin-tested in May. This testing was not a part of the investigation of this outbreak, but was the routine annual skin testing performed at the prison as per the current guidelines of the US Centers for Disease Control and Prevention (CDC). As this testing occurred before there was transmission at the prison, the results of this screening were used for a measure of the 'baseline' rate of positive skin tests and skin test conversions. Skin test conversion rates in dormitory A in May (7/126, 6%) did not differ significantly from skin test conversion rates in the other five dormitories (29/578, 5%). The subsequent rate of positive TST for inmates in the five unexposed dormitories was 2.6% (16/615) in September and 2.6% (14/537) in December 1999.

Contact investigations were conducted for prison employees, visitors to case patients, and contacts of released case patients. Of 398 previously TST-negative prison employees potentially exposed to infectious case patients, 307 (77%) completed TST screening. Sixteen (4%) employees had positive TSTs, of which seven were documented conversions. Of 22 visitors to culture-positive inmate case patients, one 11-year-old child had a positive TST. A previous TST result was not available. Community contact investigations around the three released case patients who were culture-positive revealed 12 persons with close contact: two had negative TSTs, one had a previous positive TST and nine had positive TSTs. Of those with positive TSTs, three were children under 12 years old. Previous TST results were not available.

There were 95 exposed employees at the community hospital where the source patient was hospitalized in July 1999. Overall, there were seven TST converters, 76 with negative TSTs, seven with previously positive TSTs and five who did not complete screening. Of 12 hospital employees exposed only during the source patient's 7-hour stay in the emergency department, three were converters. Of 83 employees exposed in other parts of the hospital, four were converters. Three converters were physicians who examined the source patient, and one was a student diagnosed with cavitary pulmonary TB in December 1999. The source case patient was not in respiratory isolation when the student examined him on four occasions. The DNA fingerprint of the student's isolate matched the outbreak strain.

DISCUSSION

The diagnosis of active TB disease in HIV-infected persons remains a challenge because presentation of TB can be non-specific or resemble other opportunistic respiratory infections.[23] The source patient initially presented with complaints of fever, abdominal pain, history of cough and weight loss; his chest radiograph was read as negative and he was treated presumptively for *P. carinii* pneumonia. At least two other outbreaks of TB in HIV-infected prison inmates have been traced to source patients with histories of fever and cough who were initially treated for *P. carinii* infection.[17,18] Lack of clear documentation of the source patient's LTBI in the prison medical record and the transfer record may have delayed the diagnosis of TB. If records had displayed the source cases's *M. tuberculosis* infection and therapy status, the spread of *M. tuberculosis* in this outbreak might have been limited. Routine treatment for LTBI might have prevented this outbreak. Despite a diagnosis of LTBI in 1984, the source patient never completed a course of treatment and subsequently developed infectious pulmonary TB, contributing to an outbreak involving 33 other cases, of which 31 (94%) were detected within 6 months of diagnosis in the source case. This outcome demonstrates the importance of treatment for all persons with LTBI, especially those at high risk for developing active disease.

HIV-infected persons with TB disease may have atypical chest radiographs,[23–26] and some may not develop a TST reaction in the presence of LTBI.[27–29] In an outbreak among HIV-infected prison inmates in California, seven secondary case patients had sputum smears positive for AFB but normal chest radiographs; five of the seven were asymptomatic at diagnosis.[17] In this report we describe a secondary case patient with no symptoms, a normal chest radiograph and sputum smears positive with numerous AFB. This demonstrates how an asymptomatic but sputum smear-positive case may escape detection until significant transmission has occurred in a congregated high-risk population.

Decreased reactivity to tuberculin among HIV-infected patients has been observed even in patients with CD4 cell counts of >400 cells/μL.[30–32] However, the number and size of TST conversions observed in this outbreak are striking: 85% of the converters had TST ⩾15 mm. Among converters with CD4 cell counts <200 cells/μL, all but one had a TST ⩾15 mm. These findings support recommendations of the American Thoracic Society and the CDC to perform TSTs on all persons exposed to *M. tuberculosis*, regardless of their immune status.[13,33]

The findings of this investigation regarding the effects of HAART on TB infection status should be interpreted in light of the complex but beneficial effects of HAART seen in other studies on patients co-infected with both *M. tuberculosis* and HIV. The use of HAART has been associated with immune reconstitution and subsequent decreases in rates of TB disease and death.[34–36] In one prospective study of HIV-infected TB patients, six of seven previously TST-negative patients with CD4 cell counts <200 cells/μL had positive TST results after combination antiretroviral therapy was started.[37] In our study, the receipt of HAART by 57% of skin-tested men may have contributed to the surprisingly large proportion of men with TST results ⩾15 mm. This finding is limited by the fact that, although the exposure period extended from May through September 1999, we are only able to present data on use of HAART in September 1999. In this report, four secondary case patients who had previously positive skin tests subsequently developed TB disease with isolates matching the outbreak strain. These data highlight the occurrence of exogenous reinfection and support the findings of other studies that suggest that in high-incidence populations, exogenous reinfection contributes significantly to the burden of TB.[38,39]

States vary by HIV testing policy and whether they congregate or segregate HIV-infected prison inmates. In states that congregate, HIV-infected inmates live in a designated area but may have contact with non-HIV-infected inmates during activities such as work, school, and recreation. In states that segregate, HIV-infected inmates are strictly separated from contact with other inmates.[5] According to a 1999 Bureau of Justice report, 78% (19 081/24 607) of known HIV-infected prison inmates in the US were incarcerated in 11 states (New York, Florida, Texas, California, Pennsylvania, New Jersey, Georgia, Maryland, Illinois, Connecticut and South Carolina).[9] Among these, only South Carolina congregates all HIV-infected inmates. Reasons for congregation cited by the South Carolina Department of Corrections included improvement of medical care and reduction of transmission of HIV to uninfected inmates. In Texas, the majority of HIV-infected inmates are congregated in prisons located close to a specialized medical facility. Some California facilities congregate HIV-infected prisoners under varying medical criteria. Currently, only Alabama maintains a policy of segregation of all HIV-infected inmates. In a January 2000 case, the Supreme Court upheld a lower court ruling permitting Alabama prisons to continue to segregate HIV-infected inmates.[40]

The outbreak in South Carolina occurred in a prison where precautions were taken to screen inmates for TB, including symptom review and physical examination upon entry to the prison. HIV-infected inmates also received a chest radiograph upon entry. Initial and annual skin testing was performed in those without a documented history of a positive TST. HIV-infected inmates received a medical review every 6 months; for those taking HAART, this review occurred every 3 months. Despite these measures, a large out-

break of TB occurred in this congregated, HIV-infected population, with subsequent transmission into the community. The results of this investigation suggest that even in a setting with surveillance systems in place, outbreaks of TB among congregated HIV-infected prison inmates may occur. Control of TB in prisons, especially among HIV-infected populations, is essential to protect the inmates, employees and the communities into which former inmates may be released. TB control is especially critical in settings where HIV-infected persons are congregated or segregated.

Acknowledgements

We thank the following persons for their participation in this investigation: Sallie Patterson and Jay Prashad (South Carolina Department of Corrections); Shea Rabley, Arthur Wozniak and Eric Brenner (South Carolina Department of Health and Environmental Control) and Patricia Simone, Beverly Metchock, Charles Woodley, Kim Newell and Scot Jones (Centers for Disease Control and Prevention).

Preliminary results of this investigation were presented at the Epidemic Intelligence Service Conference, Atlanta 10 April 2000 and in the Morbidity and Mortality Weekly Report 2000; 49: 1041–1044.

References

1 Beck A J. Bureau of Justice Statistics: Prison and Jail Inmates at Midyear 1999. Washington, DC: Department of Justice, Bureau of Justice Statistics, National Center for Justice Statistics, April 2000.
2 Braun M M, Truman B I, Maguire B, et al. Increasing incidence of tuberculosis in a prison inmate population. Association with HIV infection. JAMA 1989; 261: 393–397.
3 Centers for Disease Control and Prevention. Prevention and control of tuberculosis in correctional facilities: recommendations of the Advisory Council for the Elimination of Tuberculosis. MMWR 1996; 45(RR-8): 1–26.
4 Centers for Disease Control and Prevention. Targeted tuberculin testing and treatment of latent tuberculosis infection. MMWR 2000; 49(RR-06): 1–54.
5 Hammett T M, Harmon P, Maruschak L M. 1996–1997 Update: HIV/AIDS and TB in correctional facilities: Issues and Practices in Criminal Justice: US Department of Justice, 1999.
6 Jones T F, Craig A S, Valway S E, Woodley C L, Schaffner W. Transmission of tuberculosis in a jail. Ann Intern Med 1999; 131: 557–563.
7 Chaves F, Dronda F, Cave M D, et al. A longitudinal study of transmission of tuberculosis in a large prison population. Am J Respir Crit Care Med 1997; 155: 719–725.
8 Centers for Disease Control and Prevention. Reported Tuberculosis in the United States 2000. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, August 2001.
9 Maruschak L M. Bureau of Justice Statistics Bulletin: HIV in Prisons and Jails 1999. Washington, DC: Department of Justice, Bureau of Justice Statistics, National Center for Justice Statistics, July 2001.
10 Dean-Gaitor H D, Fleming P L. Epidemiology of AIDS in incarcerated persons in the United States, 1994–1996. AIDS 1999; 13: 2429–2435.
11 Selwyn P A, Hartel D, Lewis V A, et al. A prospective study of the risk of tuberculosis among intravenous drug users with human immunodeficiency virus infection. N Engl J Med 1989; 320: 545–550.
12 Daley C L, Small P M, Schecter G F, et al. An outbreak of tuberculosis with accelerated progression among persons infected with the human immunodeficiency virus. An analysis using restriction-fragment-length polymorphisms. N Engl J Med 1992; 326: 231–235.
13 American Thoracic Society. Diagnostic standards and classification of tuberculosis in adults and children. Am J Respir Crit Care Med 2000; 161: 1376–1395.
14 Bergmire-Sweat D, Barnett B J, Harris S L, Taylor J P, Mazurek G H, Reddy V. Tuberculosis outbreak in a Texas prison, 1994. Epidemiol Infect 1996; 117: 485–492.
15 Koo D T, Baron R C, Rutherford G W. Transmission of *Mycobacterium tuberculosis* in a California State Prison, 1991. Am J Public Health 1997; 87: 279–282.
16 Centers for Disease Control and Prevention. Tuberculosis transmission in a state correctional institution—California, 1990–1991. MMWR 1992; 41: 927–929.
17 Centers for Disease Control and Prevention. Tuberculosis outbreaks in prison housing units for HIV-infected inmates—California, 1995–1996. MMWR 1999; 48: 79–82.
18 Valway S E, Greifinger R B, Papania M, et al. Multidrug-resistant tuberculosis in the New York State prison system, 1990–1991. J Infect Dis 1994; 170: 151–156.
19 van Embden J D, Cave M D, Crawford J T, et al. Strain identification of *Mycobacterium tuberculosis* by DNA fingerprinting: recommendations for a standardized methodology. J Clin Microbiol 1993; 31: 406–409.
20 Epi-Info, Version 6.04B: A word processing, database and statistics program for public health on IBM-compatible microcomputers [computer program]. Version Version 6.04B. Atlanta, GA: Centers for Disease Control and Prevention, 1999.
21 LOGISTIC version 3.11Ef: A logistic regression program for the IBM PC [computer program]. Version 3.11Ef. Andover, MA: 1994.
22 ASHRAE Standard ventilation for Acceptable Indoor Air Quality 62. Atlanta, GA: American Society of Refrigerating and Air Conditioning Engineers, 1999.
23 Havlir D V, Barnes P F. Tuberculosis in patients with human immunodeficiency virus infection. N Engl J Med 1999; 340: 367–373.
24 Pitchenik A E, Rubinson H A. The radiographic appearance of tuberculosis in patients with the acquired immune deficiency syndrome (AIDS) and pre-AIDS. Am Rev Respir Dis 1985; 131: 393–396.
25 Perlman D C, el-Sadr W M, Nelson E T, et al. Variation of chest radiographic patterns in pulmonary tuberculosis by degree of human immunodeficiency virus-related immunosuppression. The Terry Beirn Community Programs for Clinical Research on AIDS (CPCRA). The AIDS Clinical Trials Group (ACTG). Clin Infect Dis 1997; 25: 242–246.
26 Markowitz N, Hansen N I, Hopewell P C, et al. Incidence of tuberculosis in the United States among HIV-infected persons. The Pulmonary Complications of HIV Infection Study Group. Ann Intern Med 1997; 126: 123–132.
27 Markowitz N, Hansen N I, Wilcosky T C, et al. Tuberculin and anergy testing in HIV-seropositive and HIV-seronegative persons. Pulmonary complications of HIV Infection Study Group. Ann Intern Med 1993; 119: 185–193.
28 Graham N M, Nelson K E, Solomon L, et al. Prevalence of tuberculin positivity and skin test anergy in HIV-1-seropositive and -seronegative intravenous drug users. JAMA 1992; 267: 369–373.
29 Jones B E, Young S M, Antoniskis D, Davidson P T, Kramer F, Barnes P F. Relationship of the manifestations of tuberculosis to CD4 cell counts in patients with human immunodeficiency virus infection. Am Rev Respir Dis. 1993; 148: 1292–1297.
30 Yanai H, Uthaivoravit W, Mastro T D, et al. Utility of tuberculin and anergy skin testing in predicting tuberculosis infection

in human immunodeficiency virus-infected persons in Thailand. Int J Tuberc Lung Dis 1997; 1: 427–434.
31 Khoo S H, Wilkins E G, Fraser I S, Hamour A A, Stanford J L. Lack of skin test reactivity to common mycobacterial antigens in human immunodeficiency virus infected individuals with high CD4 counts. Thorax 1996; 51: 932–935.
32 Garcia-Garcia M L, Valdespino-Gomez J L, Garcia-Sancho C, et al. Underestimation of *Mycobacterium tuberculosis* infection in HIV-infected subjects using reactivity to tuberculin and anergy panel. Int J Epidemiol 2000; 29: 369–375.
33 Centers for Disease Control and Prevention. Screening for tuberculosis and tuberculosis infection in high-risk populations. Recommendations of the Advisory Council for the Elimination of Tuberculosis. MMWR 1995; 44(RR-11): 19–34.
34 Girardi E, Palmieri F, Cingolani A, et al. Changing clinical presentation and survival in HIV-associated tuberculosis after highly active antiretroviral therapy. J AIDS 2001; 26: 326–331.
35 Kaplan J E, Hanson D, Dworkin M S, et al. Epidemiology of human immunodeficiency virus-associated opportunistic infections in the United States in the era of highly active antiretroviral therapy. Clin Infect Dis 2000; 30 (Suppl 1): S5–S14.
36 Leonard M K, Larsen N, Drechsler H, et al. Increased survival of persons with tuberculosis and human immunodeficiency virus infection, 1991–2000. Clin Infect Dis 2002; 34: 1002–1007.
37 Narita M, Ashkin D, Hollender E S, Pitchenick A E. Paradoxical worsening of tuberculosis following antiretroviral therapy in patients with AIDS. Am J Respir Crit Care Med 1998; 158: 157–161.
38 Van Rie A, Warren R, Richardson M, et al. Exogenous reinfection as a cause of recurrent tuberculosis after curative treatment. N Engl J Med 1999; 341: 1174–1179.
39 Small P M, Shafer R W, Hopewell P C, et al. Exogenous reinfection with multidrug-resistant *Mycobacterium tuberculosis* in patients with advanced HIV infection. N Engl J Med 1993; 328: 1137–1144.
40 Davis v. Hopper (98-9663): Federal Court of Appeals 11th Circuit; 1999.

RÉSUMÉ

CONTEXTE : En août 1999, le diagnostic de tuberculose (TB) pulmonaire est porté chez un pensionnaire de prison infecté par le virus de l'immunodéficience humaine (VIH). Ce patient-index vivait dans un dortoir de prison abritant plus de 300 hommes infectés par le VIH et accusait des symptômes depuis au moins 2 mois avant le diagnostic. Nous faisons état d'une large épidémie de TB chez les pensionnaires de prison infectés par le VH avec transmission ultérieure de *Mycobacterium tuberculosis* à l'extérieur de la prison.

MÉTHODES : Les prisonniers exposés ont été dépistés par recherche des symptômes, cliché thoracique et test cutané tuberculinique (TST) en septembre et décembre 1999. Nous avons enregistré les décomptes de cellules CD4, les charges virales ainsi que l'existence d'un traitement antirétroviral puissant (HAART).

RÉSULTATS : Le patient-index vivait du côté droit d'un dortoir à deux côtés, abritant exclusivement des hommes infectés par le VIH. Sur les 114 hommes du côté droit qui ont été testés, des virages du TST ont été documentés chez 75 (66%). Sur l'ensemble des 96 virages, 82 (85%) avaient un TST ⩾15mm. Au cours des 6 mois qui ont suivi le diagnostic de TB chez le patient-index, 30 prisonniers supplémentaires et un travailleur de soins de santé qui soignait le patient-index ont développé une maladie TB. Deux prisonniers supplémentaires ont développé une maladie TB au cours du printemps 2001.

CONCLUSIONS : Nous décrivons une transmission massive de *M. tuberculosis* dans un groupe de pensionnaires de prison infectés par le VIH avec des taux élevés de virage du TST et une transmission ultérieure de la TB dans la collectivité. Dans les situations où sont regroupées les personnes infectées par le VIH, les conséquences des épidémies de TB sont amplifiées.

RESUMEN

MARCO DE REFERENCIA : En agosto de 1999 se diagnosticó una tuberculosis (TB) en un prisionero infectado con el virus de la inmunodeficiencia humana (VIH). Este paciente índice vivía en un dormitorio de prisión que albergaba más de 300 hombres infectados con el VIH y presentaba síntomas por lo menos dos meses antes del diagnóstico. Se informa sobre un gran brote epidémico de TB en prisioneros infectados con VIH con transmisión subsecuente de *Mycobacterium tuberculosis* fuera de la prisión.

MÉTODO : Los prisioneros expuestos fueron detectados por medio de la búsqueda de síntomas, radiografía de tórax y prueba cutánea de tuberculina (TST) en septiembre y diciembre de 1999. Se registró el recuento de células CD4, las cargas virales y la administración de una terapia antirretroviral altamente activa (HAART).

RESULTADOS : El paciente índice vivía en el ala derecha de un dormitorio de dos alas que albergaba exclusivamente hombres infectados con el VIH. De 114 hombres del ala derecha que fueron examinados, 75 (66%) tuvieron una conversión documentada de la TST. Del total de 96 conversiones, 82 (85%) tenían reacciones TST con diámetro ⩾15 mm. Durante los 6 meses que siguieron al diagnóstico de TB en el paciente índice, 30 prisioneros adicionales y un trabajador de atención médica que atendía al paciente índice, desarrollaron una enfermedad TB. Dos prisioneros adicionales desarrollaron una enfermedad TB en la primavera de 2001.

CONCLUSIÓN : Se describe una extensa transmisión de *M. tuberculosis* en un grupo de prisioneros infectados con el VIH, con altas tasas de conversión de la TST y una transmisión subsecuente a la comunidad. En los sitios donde se encuentran agrupadas personas infectadas con el VIH, las consecuencias de los brotes epidémicos de TB son magnificadas.