

Table 1. Peak elevations of transaminases in person who received alternative preventive therapy with pyrazinamide and ofloxacin.

Group	Peak AST				Peak ALT			
	1–45 U/L (normal)	46–225 U/L (1–5× ULN)	226–450 U/L (>5–10× ULN)	>450 U/L (>10× ULN)	1–45 U/L (normal)	46–225 U/L (1–5× ULN)	226–450 U/L (>5–10× ULN)	>450 U/L (>10× ULN)
APT completed (n = 9)	6	3	0	0	6	3	0	0
APT not completed (n = 13)	4	5	2	2	4	2	4	3

NOTE. ALT = alanine aminotransferase; APT = alternative preventive therapy; AST = aspartate aminotransferase; and ULN = upper limit of normal value.

received preventive therapy with pyrazinamide and a fluoroquinolone, a large proportion reported adverse events, all of which were mild (Centers for Disease Control and Prevention [CDC], unpublished data). Serious adverse effects were seen in three of the 22 contacts discussed herein. Other contacts had mild-to-moderate elevations of aminotransferases, but because medications were stopped, it is unknown what the outcome would have been if the drugs had been continued.

In Public Health Service Trials that examined the efficacy of isoniazid as preventive therapy for tuberculosis and the ability to tolerate the drug, mild aminotransferase elevations were noted but were determined to be of little clinical significance [3, 4]. However, several individuals in our group of 22 contacts who had mild aminotransferase elevations had a progressive rise in serum aminotransferase levels when medications were continued. High rates of intolerance, including elevated aminotransferase levels, were recently reported in adult health care workers who received pyrazinamide and ofloxacin as APT [5].

Decisions to initiate APT for persons who are likely to be infected with isoniazid- and rifampin-resistant *M. tuberculosis* should be based on the likelihood of progression from infection to active tuberculosis and on knowledge of the drug susceptibility pattern of the infecting organism. Whenever infection with isoniazid- and rifampin-resistant *M. tuberculosis* occurs and APT is to be initiated, a regimen better tolerated than pyrazinamide and ofloxacin (e.g., pyrazinamide and ethambutol) should be considered, as long as the infecting organism is susceptible to these medications.

Some physicians have used ofloxacin alone to treat persons with isoniazid- and rifampin-resistant *M. tuberculosis* infection (CDC, unpublished data). Persons who receive APT with pyrazinamide and ofloxacin should be closely monitored for adverse effects. Ultimately, the best measures for preventing the emergence of isoniazid- and rifampin-resistant *M. tuberculosis* infection include the prompt recognition and appropriate isolation and treatment of persons with tuberculosis.

**Renée Ridzon, Jody Meador, Roberta Maxwell,
Kathy Higgins, Penny Weismuller, and Ida M. Onorato**

Division of Tuberculosis Elimination, Centers for Disease Control and Prevention, Atlanta, Georgia; and the Orange County Health Care Agency, Santa Ana, California

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Pneumocystis carinii Pneumonia in Asians and Pacific Islanders

With use of U.S. surveillance data for adolescents and adults whose condition was diagnosed as AIDS from 1988 through 1992,

Hu and coauthors [1] showed that the risk for *Pneumocystis carinii* pneumonia (PCP) was higher for blacks and Hispanics than for whites. They excluded data on Asians and Pacific Islanders and on Native Americans from their analyses. Despite growing evidence that the incidence of AIDS among Asians and Pacific Islanders in the United States—the fastest-growing racial group in this country—will rapidly increase, the circumstances of Asians and Pacific Islanders with AIDS are routinely ignored. The risk of PCP is of particular concern for many clinicians because PCP is preventable with the use of commonly available prophylactic agents, and thus the appearance of PCP may be indicative of barriers to receiving or maintaining appropriate care [2–4] or to group differences in microbe strain or exposure history [5, 6].

Reprints or correspondence: Dr. Haftan Eckholdt, Department of Psychiatry, UMDNJ, ADMC 1404E, 30 Bergen Street, Newark, New Jersey 07107-3000.

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To assess the existence of a racial/ethnic trend with regard to the risk for PCP, we analyzed data on the frequency and proportion of confirmed PCP diagnoses as the presenting opportunistic infection among adolescents and adults diagnosed with AIDS in the United States from January 1984 through December 1994; these data were obtained directly from the Centers for Disease Control and Prevention's Public Information Data Set (AIDSPIDS) [7] and were the same type that were used by Hu and coauthors [1]. Logistic regression analysis was conducted with use of SAS software (SAS Institute, Cary, NC). Models were designed after those used by Hu and coauthors [1].

The outcome was a confirmed PCP diagnosis as the presenting opportunistic infection, and racial group was dummy coded, contrasting each racial group against "Asian and Pacific Islander." Covariates were the year of diagnosis (entered as a continuous variable [84 through 94]), transmission exposure group (with use of the AIDSPIDS exposure categories, these groups were as follows: injection drug use, heterosexual contact with a person with or at increased risk for HIV infection, and other risks factors such as hemophilia and having undergone a blood transfusion—data for these groups were contrasted with data for men who have sex with men), age at diagnosis (with use of the AIDSPIDS age category, age was entered as one of the following continuous variables: 13–19 years, 20–24 years, 25–29 years, 30–34 years, 35–39 years, 40–44 years, 45–49 years, 50–54 years, 55–59 years, 60–64 years, and ≥ 65 years), geographic region of residence in the United States (the following AIDSPIDS categories were used: central, western, southern, mid-Atlantic, and smaller metropolitan statistical areas [population of 50,000–1,000,000]—these regions were contrasted with the northern region), gender, and birthplace (born in the United States vs. born outside the United States).

Inspection of these data showed that, compared with all other racial groups, a higher proportion of Asians and Pacific Islanders with AIDS were diagnosed with PCP as the presenting opportunistic infection each year from 1984 through 1994 in spite of the overall decrease in the incidence of PCP as the presenting opportunistic infection that was diagnosed during that decade (β for year when race was controlled for = -0.289 , $\chi^2 = 39224$, $P < .0001$). For example, PCP was diagnosed as the presenting AIDS-defining condition for 82.61% of Asians and Pacific Islanders in 1984, when the overall average was 62.5%. Similarly, PCP was diagnosed as the presenting AIDS-defining condition for 23.51% of Asians and Pacific Islanders in 1994, when the overall average was 13.1%.

Table 1 describes the results of a logistic regression analysis that was done to assess the strength and magnitude of the difference in risk for PCP diagnoses between Asians and Pacific Islanders and other racial groups for the 10-year period from 1984 to 1994; adjusted odds ratios showed that Asians and Pacific Islanders were at increased risk for PCP compared with all other racial groups ($P < .0001$) when controlling for year of diagnosis, transmission exposure group, geographic region of residence in the United States, gender, and birthplace.

Asians and Pacific Islanders are at increased risk for PCP compared with all racial groups when controlling for relevant temporal, demographic, and HIV transmission/exposure factors. The consistently high proportion of PCP diagnoses among Asians and Pacific Islanders with AIDS is thought, in part, to

Table 1. Adjusted odds ratios for AIDS-defining PCP diagnoses among Asians and Pacific Islanders in the United States vs. those for other racial groups.

Race	OR*	95% CI
A & PI vs. Native American	1.367	(1.16, 1.60)
A & PI vs. Latino	1.919	(1.78, 2.07)
A & PI vs. African American	1.689	(1.56, 1.81)
A & PI vs. White	1.533	(1.42, 1.65)

NOTE. Odds ratios were determined by controlling for year of AIDS diagnosis, transmission exposure risk group, gender, country of birth, region of residence, and age at diagnosis. A & PI = Asians and Pacific Islanders.

* Each adjusted OR was significant at $P < .0001$.

be the result of barriers to adequate health care [2–4]. Hu et al. [1] concluded that differences in rates of AIDS-defining conditions by race/ethnicity are also likely due to differences in underlying exposure or reporting of conditions [5, 6]. Other researchers believe that the barriers to proper care are the result of the lack of culturally relevant and language-appropriate educational materials, denial, distrust of institutions, fear of being identified as a person with AIDS, and poor knowledge of the disease and its sequelae [8, 9].

Before any firm conclusions can be made as to the nature of racial/ethnic differences in AIDS-defining conditions, readers should be cautioned that it is necessary to acknowledge both methodologically and statistically that there is a great deal of diversity and variance within each racial/ethnic group [10]. It is crucial for researchers, clinicians, and policy makers to understand that barriers may be preventing Asians and Pacific Islanders, like other minority groups, from receiving adequate prophylaxis or from maintaining prophylaxis for an easily preventable AIDS-defining condition.

Haftan Eckholdt and John Chin

Department of Psychiatry, University of Medicine and Dentistry of New Jersey, Newark, New Jersey; and Asian and Pacific Islander Coalition on HIV/AIDS, New York, New York

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***Tsukamurella incheonensis* Bacteremia in a Patient Who Ingested Hydrochloric Acid**

Gordona, *Nocardia*, *Rhodococcus*, and *Tsukamurella* are mycolic acid-containing bacteria that are classified as nocardioform *Actinomycetes* [1, 2]. The infections due to these organisms have been well reported in the literature, except for those due to *Tsukamurella*. [3, 4]. We noted only three cases of catheter-associated *tsukamurella* bacteremia in the English-language literature [5]. In Korea as well as in other countries, caustic soda has been the most frequently ingested corrosive material, accidentally or for suicidal purposes, while ingestion of hydrochloric acid has been rare [6].

We isolated gram-positive, weakly acid-fast bacilli from blood cultures performed for a patient who ingested hydrochloric acid. Yassin et al. [7] showed that the isolate was distinct from any other previously described species and named it *Tsukamurella incheonensis* (*sic*; Shinchon, not Incheon, is the name of the city where the patient was treated, and the strain was isolated in Shinchon Severance Hospital). We were not able to find any other cases of *T. incheonensis* bacteremia in a patient who had ingested hydrochloric acid in the English-language literature. In the only other case in which *T. incheonensis* was isolated, the isolate originated from the lung tissue of a patient who had a necrotic lung tumor [7].

A 30-year-old man was transferred to the emergency department at Shinchon Severance Hospital in Seoul, South Korea, on 17 March 1991 with complaints of hematemesis, abdominal pain, and severe irritability. Because of family problems, he had drunk liquor, after which he ingested ~150 mL of 20% hydrochloric acid. On physical examination, chemical burns were noted on his lips and oral mucosa. His abdomen was flat but slightly rigid and tender. Coarse crackles were noted in both lung fields. His blood pressure was 130/80 mm Hg, and his pulse was 100/min.

His stomach was irrigated with ice water, and mechanical ventilation was started. On the second hospital day, analysis of arterial blood gases revealed a pH of 7.05, a PCO₂ of 58 mm Hg, a PO₂ of 68 mm Hg, and an O₂ saturation of 83%. His severe acidosis was corrected by the mechanical ventilation. On hospital day 4, the results of blood chemistry tests were unremarkable except for the level of electrolytes, which were as follows: sodium, 137 mmol/L; potassium, 2.9 mmol/L; chloride, 102 mmol/L; and CO₂,

25 mmol/L. On hospital day 7, swallowing became difficult. Total parenteral nutrition was started through a subclavian catheter on hospital day 12.

Between hospital day 25 and 34, a fever (temperature, 38.0°C–39.4°C) was noted, and the peripheral blood leukocyte count rose from 5,900/μL to 18,400/μL. Five blood cultures performed during hospital days 25–28 yielded gram-positive bacilli (strain YMC 91-4-5931). The organism was considered to be a contaminant until it was isolated repeatedly.

The isolate formed tiny colonies on common bacteriological media after 24 hours of incubation at 37°C in room air. The colonies were 2–3 mm in diameter after 3 days and were tan. The isolate stained weakly acid fast, but it was not identified as *Nocardia* species as it did not produce aerial hyphae. It was suspected to be either *Rhodococcus*, *Gordona*, or *Tsukamurella*. With oxidation-fermentation medium [8], acid was produced from glucose, mannitol, sucrose, and maltose. The organism hydrolyzed esculin and urea, it grew in the presence of 6% NaCl, and it grew at 42°C. Gas-liquid chromatography (kindly tested by Dr. Hee Kyung Seong, Inje University Hospital, Pusan, South Korea) with Microbial Identification System (HP 5890, Hewlett-Packard, Palo Alto, CA) showed that the major methylated fatty acids of the isolate were C14:0 (4.6%), C15:0 (15.8%), C16:0 (32.2%), and C18:1 (30.5%); the organism was identified as *Nocardia amarae* (probability, 57%), *Rhodococcus chubuensis* (44%) and *Gordona rubropertincta* (35%). However, Yassin et al. found that the chemotaxonomic characteristics and genomic 16S ribosomal DNA sequences of our isolate were distinct from those of other *Tsukamurella* species [7].

The natural habitat of *T. incheonensis* is unknown, but it is probably ubiquitous in the natural environment, as are other acid-fast organisms such as *Nocardia*, *Rhodococcus*, *Gordona*, and many of the *Mycobacterium* species. *Tsukamurella paurometabolum* has been isolated from human sputum, from mycetoma, from ovaries of bed bugs, and from pseudoinfection due to laboratory contamination [9]. Infections due to *Mycobacterium* other than *M. tuberculosis* and to *Nocardia* species occur frequently, unlike infections due to *Rhodococcus*, *Gordona*, and *Tsukamurella* species [3, 10]. Bloodstream infections due to *Gordona*, *Rhodococcus*, and *Tsukamurella* species are usually associated with indwelling catheters. Shapiro et al. reported catheter-associated *T. paurometabolum* sepsis in three oncology patients [5].

It is often difficult to determine the etiologic role of saprophytic organisms. However, based on the repeated isolation of *T. incheonensis* from five blood cultures during a 5-day period, together with the patient's fever and leukocytosis, we believe that the isolate was the causative agent. It is possible that after the patient ingested hydrochloric acid the organism remained alive on the damaged mucous membrane because of the high fatty acid content of the

Reprints or correspondence: Dr. Yunsop Chong, Department of Clinical Pathology, Yonsei University College of Medicine, C.P.O. Box 8044, Seoul, South Korea.