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decreases in HIV-1 replication that would reduce the level of *tat* gene expression, and this reduced level of expression could induce neither angiogenesis nor malignant transformation of endothelial cells by *tat*-induced cytokines [3]. Third, the use of antiretroviral agents could be associated with an improvement in the immune response, enabling control of tumor progression.

Further studies are needed to better determine the therapeutic effects of antiretroviral therapy on the progression of KS in HIV-infected patients. Combinations of these drugs could become alternatives to the conventional treatment of AIDS-related KS.

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Three Episodes of Tuberculosis—To Multidrug Resistance and Back to Susceptibility

We report the case of a patient who had three episodes of tuberculosis; two of these episodes were due to resistant strains of *Mycobacterium tuberculosis,* and one was due to a susceptible strain.

A 31-year-old Tibetan woman who was born in India developed smear-positive tuberculosis (TB) of the lung in 1987, 1990, and 1995. Upon immigration to Switzerland in 1984, findings on her chest radiograph were normal. Three years later, progressive, cavernous TB of the left upper lobe was diagnosed (episode 1). *M. tuberculosis* was isolated from sputum and gastric fluid specimens, and primary resistance to isoniazid was detected. The patient was hospitalized and treated with isoniazid, rifampin, and pyrazinamide for 2 months, followed by a regimen of rifampin and ethambutol for 4 months. After 14 weeks of treatment, cultures became negative.

After the patient recovered clinically, she traveled to India and Nepal in 1989; however, upon her return in 1990, she developed a cough and night sweats, and cavernous TB of the left upper lobe, with laryngeal and endobronchial involvement, was diagnosed (episode 2). The *M. tuberculosis* isolate proved to be resistant to isoniazid, rifampin, and streptomycin. Treatment with isoniazid, rifampin, ethambutol, pyrazinamide, and ciprofloxacin was started; therapy with the latter three drugs was continued for 12 months. After she recovered, she traveled several times to India between 1990 and 1993 and subsequently worked as an assistant nurse at the University Hospital in Zurich. In the fall of 1995, she complained of pain under the right scapula and developed a productive cough, malaise, fever, and night sweats. An infiltrate was observed on a chest radiograph, and pulmonary TB with right-sided cervical lymphadenitis was diagnosed (episode 3). For the third time, cultures were positive, but surprisingly, the *M. tuberculosis* isolate was fully susceptible to all first-line drugs.

This finding prompted us to study all isolates at the molecular level to better understand this case. By using *Pvu*II as the restriction enzyme and IS*6110* as a probe [1], we found two different patterns of restriction fragment length polymorphism (RFLP): the isolates recovered during episode 1 and episode 2 had identical DNA fingerprints, whereas those recovered during episode 3 differed considerably from the initial strains. On probing *Alu*I-digested DNA with the polymorphic GC-rich repetitive sequence [2], banding patterns for the isolates from episodes 1 and 2 were again found to differ from those of episode 3.

On the basis of this case, three major conclusions can be drawn: (1) Of the two episodes of TB in 1990 and 1995, only one was a true relapse, probably caused by inadequate drug therapy. Conversely, episode 3 was due to exogenous reinfection. (2) Treatment of TB after episode 2 was successful, as indicated by the significant improvement in the patient's condition and the numerous culture-negative follow-up specimens that were obtained. (3) A mixed-strain infection during episode 3 can be safely ruled out, not only on account of the DNA fingerprinting results but also because of the isolate's susceptibility pattern (fully susceptible), which

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excludes the presence of the previous strains (resistant to isoniazid and to multiple drugs).

Since the strain recovered from the patient's sister, who had active TB in 1984, was not available for typing, it is not clear whether the patient became infected via her sister or whether she was infected while living in Tibet. The latter scenario is quite likely, since the incidence of TB among Tibetan immigrants living in Switzerland is very high (384 cases per 100,000 persons; P. Helbling, Swiss Federal Office of Public Health, personal communication). In addition, the time of reinfection with the fully susceptible strain is not known.

Exogenous reinfection has been demonstrated conclusively in patients with advanced HIV infection [3], but such reinfection has also been reported among immunocompetent individuals with either alcoholism [4] or diabetes [5] as risk factors. The patient described herein had neither underlying disease nor other known risk factors. To our knowledge, this is the first well-documented case demonstrating that both relapse of TB, with progression to multidrug-resistant TB, followed by exogenous reinfection and development of active disease within a short period (8 years) is possible within the same patient. A history of frequent travel to areas where TB is highly prevalent supports the assumption that in such countries, exogenous reinfection may be a common mechanism by which clinical TB develops [6]. If so, global strategies for TB control must be aimed primarily at minimizing transmission, i.e., eliminating *M. tuberculosis* in such environments.

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Inflammatory Tinea Corporis Due to *Trichophyton verrucosum*

A recent report by Sabota et al. describes tinea barbae due to *Trichophyton verrucosum* in dairy farmers [1]. *T. verrucosum* may also cause other infections including tinea corporis [2, 3], and other animals, such as horses, may be the source of infection [4, 5]. We describe a patient, with horse contact, who developed inflammatory tinea corporis due to *T. verrucosum,* confirmed by histopathology and culture results.

A 54-year-old man, whose work involved riding horses in rural areas, presented with left leg tenderness and erythema; he had no fever or other systemic symptoms. There was no response to treatment with oral flucloxacillin and roxithromycin. A biopsy of the leg lesion was performed. A gram stain of the specimen was negative, but coagulase-negative staphylococci were isolated, and histopathology demonstrated nonspecific chronic inflammatory changes with folliculitis. Treatment with flucloxacillin (1 g q6h iv) and oral ciprofloxacin (750 mg b.i.d.) was begun, but there was no improvement in the patient's condition.

Seven weeks after his initial presentation, he was referred to our institution for consideration of hyperbaric oxygen therapy for ''cellulitis'' that was not responding to therapy with antibacterial agents. Examination of the medial aspect of the left leg revealed a 15 × 6-cm, erythematous, indurated plaque, containing pustular lesions with necrotic areas (figure 1). There was no fever or inguinal lymphadenopathy. Results of a complete blood count and blood chemistry evaluation were normal, and the erythrocyte sedimentation rate was 13 mm/h. Repeated skin biopsy was performed, and results of gram and acid-fast stains as well as routine bacterial and mycobacterial cultures were negative. Histopathologic evaluation demonstrated a mixed inflammatory cell infiltrate and a fungal spore (figure 1). After 2 weeks' incubation at 30°C, *T. verrucosum* was isolated. Oral itraconazole (200 mg b.i.d.) was administered for 12 weeks, and the leg lesion resolved completely.

Chronic tinea corporis is uncommon and is usually due to either *Trichophyton mentagrophytes* or *T. verrucosum* [4]. *T. verrucosum* is distributed globally [5], and human infection has been reported previously from Australasia [6, 7]. *T. verrucosum* usually infects cows, but it is also isolated frequently from horses [4]. In the current case, the lesion was on the patient's leg, where there had been direct contact with a common site of horse ringworm [4]. Zoophilic dermatophytes like *T. verrucosum* may produce inflammatory disease that can be mistaken for bacterial infection [1, 5, 8]. Tinea corporis due to zoophilic dermatophytes may resolve spontaneously, but systemic antifungal therapy is usually required [5, 9].

In cases of tinea corporis, biopsies may demonstrate cylindrical fungal filaments, or rounded arthroconidia [10], which are characteristically large in *T. verrucosum* infection [3]. In inflammatory tinea corporis caused by zoophilic dermatophytes such as *T. verrucosum,* hypersensitivity reactions may produce vesicles and papules accompanied by dermal infiltrates [10], or vasculitis characterized by small follicular papules with necrotic areas [3]. Histopathologic features of hypersensitivity reactions include infiltration of lymphocytes, plasma cells, neutrophils, and eosinophils into the dermis [10].

Tinea corporis due to *T. verrucosum* should be included in the differential diagnosis of chronic inflammatory skin lesions in patients with a history of contact with animals, including cows and horses.

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