A 25-Year-Old Woman with an Ulcerative Earlobe Lesion

(See pages 552–3 for the Photo Quiz.)

**Figure 1.** A, Hematoxylin-eosin stain of a skin wedge biopsy specimen obtained from the patient (original magnification, ×40) showing pseudo-epitheliomatous hyperplasia and granulomas involving the skin and cartilage. B, Hematoxylin-eosin stain of the skin wedge biopsy specimen (original magnification, ×400) showing a noncaseating granuloma. C and D, Photographs of the patient’s ear, which demonstrated significant clinical improvement after 3 months of antituberculosis therapy.
Diagnosis: cutaneous *Mycobacterium tuberculosis* infection.

A deep-wedge biopsy was performed, and the histopathological evaluation of the skin specimen showed pseudoepitheliomatous hyperplasia. A significant number of noncaseating granulomas were seen in the cutaneous and subcutaneous tissues (Figure 1A and 1B). Gram stain, fungal stain, and acid-fast bacilli stains had negative findings. Because of the presence of granulomas, a tuberculin skin test (TST) was performed, which resulted in a 30-mm induration. Tissue samples were cultured in Lowenstein-Jensen Middlebrook media, as well as in the liquid culture automated system. Because all of the stains had negative findings and the patient was asymptomatic except for the auricular lesion, antimicrobial drugs were withheld. Four weeks after the third biopsy was performed, the mycobacterial cultures were reported to be positive for *M. tuberculosis*. Afterwards, in vitro susceptibility studies determined that the organism was susceptible to isoniazid (minimum inhibitory concentration [MIC], 0.1 μg/mL), rifampin (MIC, 2.0 μg/mL), ethambutol (MIC, 2.5 μg/mL), and pyrazinamide (MIC, 100 μg/mL). Therapy was initiated with all 4 drugs. Three months after initiation of therapy, the patient was again seen in the clinic. This time, the lesion had already demonstrated significant clinical improvement and resolution of discharge, tenderness, and erythema (Figure 1C and 1D).

Extrapulmonary tuberculosis constitutes 10%–12% of all cases of tuberculosis, whereas cutaneous tuberculosis is seen in ~1.5% of all cases [1–3]. Because cutaneous tuberculosis has a wide spectrum of initial presentations, it is not unusual for the time from presentation to diagnosis to be prolonged. A high index of suspicion among patients who are at high risk can help to direct the appropriate investigations, thereby leading to an early diagnosis and initiation of appropriate treatment.

In developed countries, cutaneous *M. tuberculosis* infection tends to occur among patients who are immunosuppressed because of malignancy, chronic corticosteroid use, or immunosuppressive therapy, whereas in developing countries, cutaneous *M. tuberculosis* infection occurs more often in the general population [4]. In recent years, however, there has been an increase in the incidence of cutaneous *M. tuberculosis* infection, especially in regions with higher rates of human immunodeficiency virus infection and in regions with multidrug-resistant pulmonary tuberculosis [5].

According to the differentiation scheme proposed by Tappeiner and Wolff [6], cutaneous *M. tuberculosis* infection can be classified as either exogenously acquired (tuberculous chancre, tuberculosis verrucosa cutis, and lupus vulgaris) or endogenously acquired (lupus vulgaris, scrofuloderma, miliary tuberculosis, and orificial tuberculosis). Our patient appeared to have a lupus vulgaris type of cutaneous tuberculosis. In Western countries, lupus vulgaris is typically seen in the region of the head and neck and generally tends to occur in individuals with a variable degree of immune dysfunction who demonstrate marked tuberculin sensitivity [4]. For unknown reasons, it is more commonly seen in female patients. Cutaneous lesions from hematogenous spread are frequently found on the face, whereas lesions located on extremities tend to occur as a result of exogenous inoculation [7]. Lupus vulgaris often runs an extremely chronic course and, as in this case, can lead to severe tissue destruction before the diagnosis is made (lupus vulgaris mutilans). The morphology of lupus vulgaris is quite variable (lupus planus, lupus exfoliativus, lupus hypertrophicus, lupus tumidus, and lupus ulcerosus) [8]. Occasionally, squamous cell carcinoma may develop in the residual scars of some patients with chronic lupus vulgaris [9].

Diagnosis is generally made by means of a positive TST result and histological findings (including smear for demonstration of the presence of acid-fast bacilli), but a definitive diagnosis can only be made by isolating *M. tuberculosis* on culture. When few viable mycobacteria are present in the tissue, culture and subsequent identification of isolates may take 6–8 weeks or longer. No correlation has been identified between TST reactivity and the extent of cutaneous *M. tuberculosis* infection (localized or disseminated) [3].

In addition, newer tests are able to detect specific *M. tuberculosis* DNA sequences that can be amplified in vitro and require only small amounts of tissue. The various polymerase chain reaction and DNA probe assays allow the different mycobacterial species to be correctly identified and differentiated with a high degree of sensitivity and specificity.

Conventional antimicrobial regimens that are recommended for the treatment of pulmonary *M. tuberculosis* infection are also adequate for treating cutaneous *M. tuberculosis* infection, because the bacillary load in cutaneous infections tends to be lower than the bacillary load in pulmonary *M. tuberculosis* infections. It is important to note that it is not unusual for patients with cutaneous *M. tuberculosis* infection to simultaneously have clinically unapparent visceral involvement [4]. The adequate duration of antimicrobial therapy has not been adequately studied, but it is currently recommended that therapy be continued for at least 2 months after the complete involution of the lesions, because several reports have demonstrated that specimens from clinically healed lesions have yielded positive culture results.

In our patient, therapy was initiated with 4 drugs and was continued for a period of 2 months. After in vitro susceptibility results were finalized and it was determined that the organism was susceptible to all 4 drugs, therapy with isoniazid and rifampin was continued for an additional 4 months, with close clinical follow up. After only 3 months of therapy, the lesion demonstrated significant clinical improvement (Figures 1C and 1D).
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