A Case of Lupus Vulgaris Followed by Miliary Tuberculosis

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We report a case of lupus vulgaris followed by miliary tuberculosis. A 58-year-old male patient was admitted to the Department of Medicine because of general malaise and coughing of 20 days duration. He was referred to the Department of Dermatology for evaluation of skin lesions, which had begun as yellowish brown papules following a glass injury on the flexor aspect of the right forearm 2 years ago, and had developed into a single plaque. Chest X-ray examination revealed miliary mottling throughout both lung fields. Sputum culture produced growth of Mycobacterium tuberculosis. Skin biopsy showed lichenoid inflammatory cell infiltration and multiple minute granulomas with Langhans' giant cells with a few acid-fast bacilli. This association supports the hypothesis that a subsequent infection might stimulate the multiplication of tubercle bacilli of the dormant strain. (Ann Dermatol 2:(2) 125-127 1990)

Key Words: Lupus vulgaris, Miliary tuberculosis

Lupus vulgaris (LV) is an extremely chronic and progressive form of post-primary cutaneous tuberculosis occurring in individuals with a high degree of tuberculin sensitivity.[1]

The lesion usually appears on normal skin as a result of direct extension, lymphatic[2] or hematogenous spread,[3] primary inoculation[4] or BCG vaccination,[5] and in the scar of scrofuloderma.[6]

Miliary tuberculosis is an illness elicited by acute diffuse dissemination of tubercule bacilli via the blood stream.

This report describes a case of LV manifesting as a single plaque on the right forearm, followed by miliary tuberculosis 2 years later.

REPORT OF A CASE

A 58-year-old male patient was admitted to the Department of Medicine because of general malaise with productive coughing of 20 days duration. Associated constitutional symptoms of easy fatigability, weight loss, fever and chilling were reported.

He was referred to the Department of Dermatology for evaluation of the skin lesions, which had begun as yellowish brown papules following a glass injury and had developed into a single plaque on the flexor aspect of the right forearm. He was vaccinated with BCG in childhood. He denied any personal or family history of tuberculosis, or recent exposure to tuberculosis.

Pertinent findings on physical examination included a pulse rate of 82, respiratory rate of 20 and temperature of 37.8°C. Moist and dry rales were heard on the entire lung fields. No lymph node was palpable. A single, 4 cm in diameter, reddish brown colored, annular plaque with central clearing and peripheral soft papules was presented on the flexor surface of the right forearm (Fig. 1). The skin lesions showed apple jelly colored papules on diascopy.

Laboratory work-up revealed ESR of 40 mm/hour and Hb of 12.2 g/100ml. Liver function tests, stool examination, EKG, BUN, creatinine, ASLO and VDRL were all within normal limits. Chest x-ray revealed miliary mottling throughout both lung fields (Fig. 2). Sputum smear was positive for acid-

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fast bacilli and Mycobacterium tuberculosis grew in the culture. We could not find any other tuberculous lesion.

Examination of the skin biopsy showed hyperkeratosis, focal spongiosis and mild exocytosis in the epidermis and a lichenoid inflammatory cell infiltration composed of lymphocytes, histiocytes, epithelioid cells and occasional Langhans' giant cells in the upper dermis (Fig. 3). Fite stain revealed a few acid fast bacilli in the cytoplasm of Langhans' giant cells (Fig. 4).

A diagnosis of LV with miliary tuberculosis was made and the patient was treated with isoniazid (300mg/day), ethambutol (800 mg/day) and cycloserine (500mg/day). The skin lesions improved in 2 months (Fig. 5).

DISCUSSION

LV is the most common, the most serious and variable type of cutaneous tuberculosis.[7] The lesion usually appears in normal skin as a result of direct extension, lymphatic or hematogenous spread, primary inoculation or BCG vaccination, and in the scar of scrofuloderma.

Miliary tuberculosis is an illness produced by

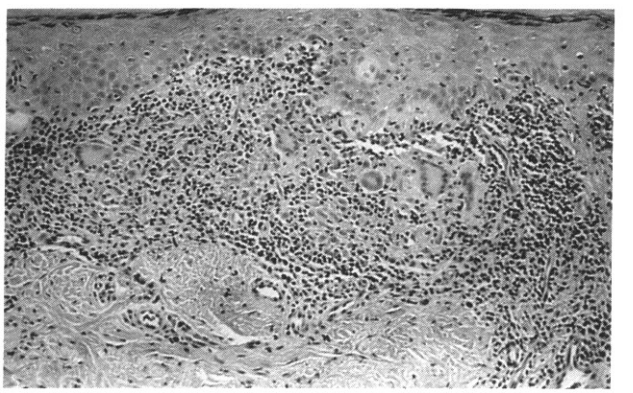

Fig. 3. Lichenoid inflammatory infiltrate composed of lymphocytes, epithelioid cells, and occasional Langhans' giant cells in the upper dermis (H & E statin, ×40).

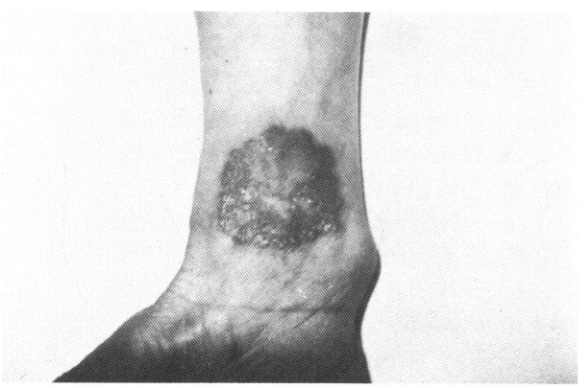

Fig. 1. A single, reddish brown annular plaque with central clearing and peripheral papules on the flexor aspect of the right forearm.

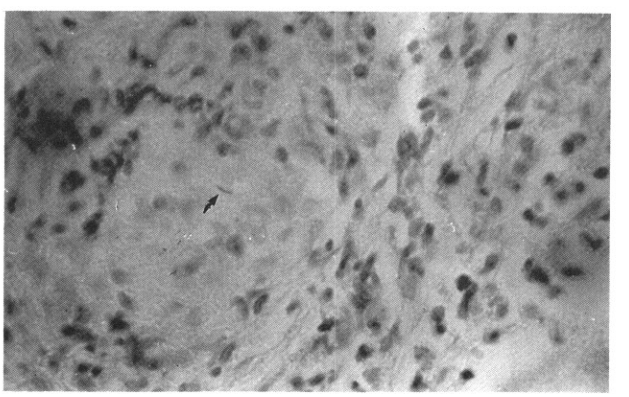

Fig. 4. Acid fast bacillus (arrow) in a Langhans' giant cell (AFB stain, ×200).

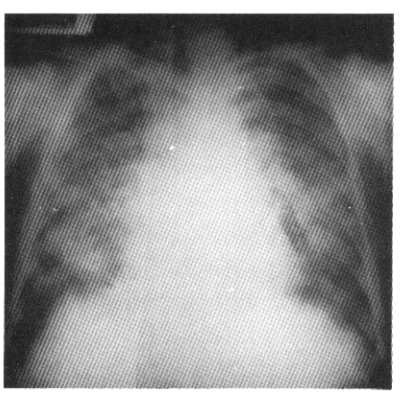

Fig. 2. Miliary mottling throughout both lung fields.

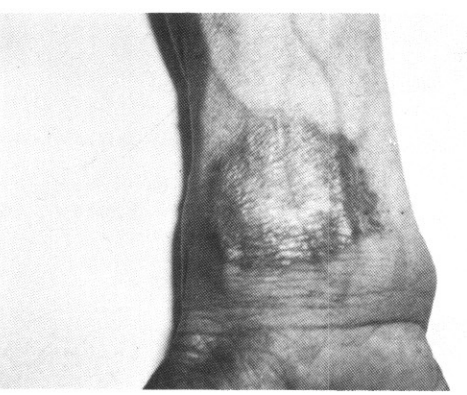

Fig. 5. The lesion improved in 2 months.

acute diffuse dissemination of tubercle bacilli via the blood stream. If follows primary pulmonary tuberculosis, recrudescence of an old primary lesion and hematogenous or lymphatic spread from pre-existing foci.[8,9]

The lesion of LV usually begins as an asymptomatic macule or soft papule. The papules or nodules characteristically show apple jelly colored lesions on diascopy. Larger patches are formed by peripheral enlargement and coalescence of papules and patches. Since the course of this disease is marked by ulceration and scarring, the clinical manifestations of LV are diverse.[7] The lesion is usually solitary and located in the head and neck region in 90 percent of the cases.[10] A small percent of the lesions affect the extremities and, rarely, the trunk.[10] In one review, only 6 percent of four thousand patients with LV grew Mycobacterium tuberculosis in Lowenstein-Jensen culture medium.[11]

The histological picture is characterized by tubercles formed by epithelioid cells, Langhans' giant cells and the peripheral zone of lymphocytes. Little, if any, caseation necrosis is present and tubercle bacilli are rarely seen.[12] It is interesting that miliary tuberculosis developed in this patient because LV is a post-primary tuberculosis showing a high degree of tuberculin sensitivity.

The underlying pathogenesis is unclear, but there are three possible pathogeneses: 1) reactivation of dormant tubercle bacilli, 2) hematogenous spread, and 3) exogenous superinfection.

Wingfield suggested[13] that the subsequent infection stimulates the multiplication of tubercle bacilli of the dormant strain. If protein moiety doses of the subsequent tubercle bacilli are large, some disturbance of the previous immune balance in one direction or another is inevitable. Therefore, an adverse disturbance, if of any great degree or of an appreciable duration of time, should influence the behavior of dormant tubercle bacilli and activate it clinically.[13] Late reactivation of primary infection can develop without obvious subsequent infection. But usually there are predisposing factors, such as measles, whooping cough, acute tonsillitis, alcoholism, pregnancy, neoplasm, cerebral vascular disease, corticosteroid therapy, etc.[8,9] There were no predisposing factors and no history of recent contact with tuberculosis in this patient.

We assume that he had dormant pulmomary foci and LV might have activated his disease. But we can not exclude the possibility of hematogenous spread from other pre-existing tuberculous foci although we could not find any other lesion.

In a patient with cutaneous tuberculosis with no evidence of systemic involvement, treatment with isoniazid alone gives a high cure rate. However, when there is any evidence of extracutaneous involvement, a combined drug regimen must be followed.[7]

Our patient was started on a triple drug regimen, consisting of isoniazid, ethambutol and cycloserine. The skin lesion improved in 2 months. After 3 months of therapy, ethambutol was discontinued. He is still presently treated to complete an 18-month course.

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