36 Tuberculosis of the Skin

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36.1 Tuberculous and Nontuberculous Infections

36.1.1 History

Tuberculosis has been a recognized ailment of mankind from ancient history. The most ancient documentation is from the Pharaonic dynasties, 2000–3000 years B.C. It is one of the most important human infections because of its prevalence and its being one which causes more suffering for humans than all bacterial genera combined (Tapeiner and Wolff 1999; Sehgal 1994). DNA of Mycobacterium tuberculosis has been detected in Peruvian mummies (Gawkrodger 1998). Cutaneous tuberculosis was first documented by Laennec where he described his own prospector’s wart (Laennec 1962) in 1826.

36.1.2 Epidemiology

Tuberculosis of the skin has a worldwide distribution. It constitutes a small portion of all cases of extrapulmonary tuberculosis which in itself is a small fraction (10%) of all tuberculous infections. It has been more prevalent in regions with cold and humid climates but is recognized in the tropics (Anonymous 1991; Markov et al. 1992). The improvement in socioeconomic standards and living conditions in European and North American countries, immunization programs, and the use of three or four drug regimens of treatment have caused a steady decline in its incidence over the last decades; it parallels the decline in incidence of pulmonary tuberculosis. Resurgence and increased incidence of cutaneous tuberculosis in the United States is mainly due to the AIDS epidemic, and this increase could be explained either by endogenous reactivation or increased susceptibility to reinfection with M. tuberculosis or low virulence mycobacteria (atypical) because of impaired cellular immunity. In the United States, until the 1980s, skin tuberculosis was a rare disease; in Europe the incidence now is less...
than 0.5%. In Germany only 300–400 new cases are estimated to occur every year. In India the incidence is 0.15% (Tapeiner and Wolff 1999). In our experience over the last 10 years (1993–2002) a total of 23 cases of cutaneous tuberculosis were seen among a total of 20,000 new dermatologic cases attended to by our hospital for the same period (0.12%).

### 36.1.3 Classification of Mycobacteria

Mycobacteria are slender, unencapsulated, nonmotile, nonsporulating, weakly positive acid-fast anaerobic rods. The tubercle bacillus takes the form of slender, curved, delicate, beaded or banded rods, 1–4 μm long and about 0.5 μm in diameter (Sehgal and Wagh 1990a). They occur either singly, in pairs, or in clumps. Their highly complex waxy coating determines the mycobacterial physiologic properties (e.g., resistance to degradation following phagocytosis) and may influence the pattern of host response (Citron and Girling 1987; Higuchi et al. 1981). They are difficult to stain, also due to their cell wall high lipid content, but once stained with basic dyes (e.g., carbolfuchsin), they retain the color and resist decolorization with acid and alcohol, thus the name “acid-fast bacilli.” Runyon (1965, 1974) classified the mycobacteria other than tuberculosis (MOTT) according to their rate of growth and pigment-production ability with or without light. Since then more studies have been made and the genus Mycobacterium stands among the best classified bacterial genera. Table 36.4 shows the classification of the atypical mycobacteria (Runyon 1965, 1974). Slow growers are divided according to their pigment-forming properties in culture: group I photochromogens capable of pigment production on light exposure, group II scotochromogens which are able to produce pigment without exposure to light, group III nonchromogens, non-pigment producers, and group IV including all rapid growers. A different classification could be used for clinical purposes which divides mycobacteria into obligate and facultative human pathogens and non-pathogens (Tapeiner and Wolff 1999).

### 36.2 Tuberculosis of the Skin

Cutaneous tuberculosis has been described long before Robert Koch identified *M. tuberculosis* (see Laennec 1962). It is caused by *M. tuberculosis*, *M. bovis*, or under certain circumstances by the bacille Calmette-Guérin (BCG). Various classifications of the clinical variants exist but none of them has been entirely satisfactory. These classifications were based on the mode of the infection, immunologic state of the host, and whether mycobacteria have been cultured and identified from the skin lesions or not. This resulted in a number of conditions with no culturable connection to tuberculosis but some have shown therapeutic response to the use of antituberculous treatment, while others show spontaneous resolution, response to other antibiotics, or to nonspecific measures such as bed rest. These are called tuberculids (Table 36.2). In this chapter an attempt to take into consideration variables that govern the outcome of the infection as well as the route of infection will be discussed, clinico-immunologic classification of the various cutaneous manifestation of the tuberculous process is seen in Table 36.1.

### 36.2.1 Etiopathogenesis

The clinical presentation is determined by the outcome of the host immune responses, mycobacterial virulence, and the route of entry of the bacilli. The host has inherent or acquired ability to control infection. Susceptibility is decreased in populations where there is contact with TB for many generations. Such contact results in partially immunized individuals through natural infections. Blacks as well as carriers of HLA-B15 major histocompatibility antigen are more susceptible to the disease. The age, an intact immune system, as well as socioeconomic and environmental factors are also of importance (Tapeiner and Wolff 1999).

Once the host is invaded by the mycobacterium, the interaction between host immune responses and mycobacterial factors such as its virulence and the size of the inoculum will determine the outcome. Either there is unchecked multiplication, with the disease becoming apparent, or this process is arrested. Antigen-presenting cells engulf the bacteria and through the antigens displayed on their surface, they activate the T lymphocytes which release cytokines. These in turn activate macrophages that engulf the mycobacteria and kill them. Few organisms are killed in each cycle, and in the very early stages resistance depends on rapidly repeated cycles. Cytotoxic T cells lyse infected phagocytic cells with the subsequent release of surviving organisms which are phagocytosed again,