CHAPTER 3

IMMUNE-PATHOGENESIS AND DIAGNOSIS OF LATENT TUBERCULOSIS INFECTION

Deniz GAZEL[1]

Tuberculosis (TB) is one of the oldest diseases of history. *Mycobacterium tuberculosis* complex is formed by a group of bacilli (*M. tuberculosis, M. bovis, M. africanum, M. microti, M. canetti and M. caprae*). The disease progresses by the host's inflammatory response due to the development of chronic granulomatous infection. Infection usually begins with inhalation of 1-3 microns of particles containing 1-3 bacilli within the inhalation. The bacteria pass the physical barriers of the upper airway and reach the alveoli (1).

TUBERCULOSIS INFECTION

Primary infection with positive skin test (PPD+) develops at about 30% of PPD (-) patients who were exposed to TB cases. Only 10% of primary infections develop to primary tuberculosis. The existence of infection or disease depends on the balance between the resistance of the host and the virulence of the bacilli. Both natural and acquired immunity play a role in the response of the host to tuberculosis. *M. tuberculosis* bacilli reaching the alveoli can be eliminated at the beginning or can be controlled by the immune response against the bacilli, or the bacilli can multiply to form a primary tuberculosis disease following the primary infection. The bacilli, which were dormant during the primary infection, may start to multiply after years and may cause secondary tuberculosis by reactivation. The primary infection may be activated at any age after a latent period (years or decades) and may cause secondary tuberculosis in other organs, most commonly in the upper regions of the lung. The immunological responses (cellular immunity and delayed of type hypersensitivity reaction) of the host against bacillus antigens determine the type of disease. Immuno-pathogenesis of pulmonary tuberculosis is staged from initial infection to cavity formation. Weeks after the invasion of alveolar macrophages by TB bacilli, the bacilli antigens are also transported to the regional lymph nodes by the infected cells. *M.tuberculosis*-infected macrophages and dendritic cells develop a specific inflammatory response; by macrophage ac-

[1] Asst. Prof. Dr., Gaziantep Üniversitesi Tıp Fak. Tıbbi Mikrobiyoloji AD. denizgazel@yahoo.com

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