

Radiologic and clinical manifestations in active pulmonary tuberculosis Correlation of serum tumor necrosis factor

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Editorial

The precise clinical manifestations of tuberculosis are likely to result from a complex interaction between the host and the pathogen. We took serum samples from a group of patients with a variety of clinical and radiological stages of pulmonary tuberculosis in order to characterize tumor necrosis factor- α (TNF- α), interleukin-4 (IL-4) and soluble interleukin-2 receptor (sIL-2R) response. We further evaluated whether the levels of TNF- α , IL-4 and soluble IL-2R are related with each other, and also evaluated the levels of TNF- α , IL-4 and sIL-2R after anti-tuberculosis therapy and relation with radiologic scores. Forty-three inpatients with active pulmonary tuberculosis and 19 healthy controls participated in the study. Patients were divided into four categories radiologically on chest X-ray (minimal, moderate-advanced, far-advanced and with miliary infiltration). Concentrations of TNF- α (20.9 \pm 10/15.4 \pm 8 pg/ml) and sIL-2R (2569 \pm 842/1444 \pm 514 pg/ml) were statistically different between patients and controls ($p=0.02$ and $p=0.0001$, respectively). Before chemotherapy there was a positive correlation between TNF- α and sIL-2R ($r=0.34$), but there was no correlation between IL-4 and TNF- α , and between IL-4 and sIL-2R ($r=-0.23$ and $r=-0.22$). The TNF- α level was not statistically different in four groups before and after chemotherapy. Results of this study provided some evidence confirming the previously reported role of TNF- α , IL-4 and sIL-2R in the control of tuberculosis, but these cytokines were not found related with disease severity. Cytokines are primarily involved in host responses to disease or infection and any involvement with homeostatic mechanism has been less than dramatic. Many cytokines are produced during tuberculosis (TB),^{1,2} with a predominance of Th1 cytokines during the early stage^{3,4} and Th2 cytokines in the later stages of the infection.⁵ These cytokines exert important roles to limit or exacerbate the disease depending on their balance and combinations. An understanding of the basis of these associations and correlations during TB could be useful in elucidating protection/pathogenesis. Some cytokines promote inflammation and are called proinflammatory cytokines [tumor necrosis factor- α (TNF- α), interleukin (IL)-1, IL-6, IL-8], whereas other cytokines suppress the activity of proinflammatory cytokines and are called anti-inflammatory cytokines (IL-4, IL-10, IL-13).⁶ TNF- α has harmful effects, such as acute-phase

pathophysiologic events including fever and tissue necrosis. It also plays a protective role against mycobacterial infection.^{7,8} Soluble interleukin 2 receptor (sIL-2R) is a surrogate marker of T-lymphocyte activation and proliferation. A soluble fraction of the IL-2 receptor, released from the cell membrane, is detectable in serum and its concentration is known to be elevated in TB.⁹ In the literature, most reports on cytokines during TB are from studies on in vitro-stimulated lymphoid cells with few reports on in vivo plasma levels. We consider immunity of an individual to TB to sometimes be reflected in the plasma levels of some cytokines. In addition, the plasma is easily accessible, thus requiring simple procedures and equipment to process it. In the present study we therefore examined the levels of TNF- α , IL-4 and sIL-2R in the serum of pulmonary TB patients.

To understand systemic Tcell response in pulmonary tuberculosis to some degree, we analyzed TNF- α , IL-4 and sIL-2R in serum. Also, to evaluate the hypothesis that different clinical and radiological manifestations of active pulmonary tuberculosis are associated with different patterns of cellular immune response systemically, we took serum samples from a group of patients with a variety of clinical and radiological stages of pulmonary tuberculosis. We further evaluated whether the levels of TNF- α , IL-4 and sIL-2R are related with each other and also evaluated the levels of TNF- α , IL-4 and sIL-2R before and after anti-TB therapy. Forty-three inpatients (20 male and 23 female) with active pulmonary TB and 19 healthy controls (eight male and 11 female) participated in the study. All patients were recruited from the Tuberculosis Hospital and Erciyes University Medical Faculty, Clinic of Pulmonology in Kayseri, which is located in the central region of Turkey. Their mean age was 36.49/ 15.5 years (range 16/67 years). On entry, all patients had positive smear for acid-fast bacilli in sputum or bronchial lavage and subsequent cultures of these specimens yielded tubercle bacilli. None of the patients had any evidence of concomitant bacterial or viral infections as indicated by sputum and blood cultures and viral serologic study including HIV. Five patients had diabetes mellitus. All patients were administered anti-TB therapy in which isoniazid, rifampicin, pyrazinamide and streptomycin or ethambutol were used. We evaluated patients with physical examinations.