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Endocrine and cytokine responses in humans with pulmonary tuberculosis.

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Source

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Abstract

Endocrine responses during chronic infections such as lung tuberculosis are poorly characterized. Hormonal changes are likely to occur since some of the cytokines produced during this disease could affect endocrine mechanisms that, in turn, influence the course of infectious/inflammatory processes. A main purpose of this work was to study endocrine responses involving pituitary, adrenal, gonadal, and thyroid hormones in parallel to IFN-gamma, IL-10, and IL-6 levels in tuberculosis patients with different degree of pulmonary involvement. We have also studied whether products derived from peripheral immune cells obtained from the patients can affect the in vitro production of adrenal steroids. The population studied comprised HIV-negative newly diagnosed, untreated male patients with mild, moderate, and advanced lung tuberculosis, and matched, healthy controls. IFN-gamma, IL-10, and IL-6 levels were elevated in patients with tuberculosis. Dehydroepiandrosterone and testosterone levels were profoundly decreased and growth hormone levels were markedly elevated in patients, in parallel to modest increases in cortisol, estradiol, prolactin, and thyroid hormone concentrations. Supernatants of peripheral blood mononuclear cells obtained from the patients and stimulated in vitro with Mycobacterium tuberculosis antigens significantly inhibited dehydroepiandrosterone secretion by the human adrenal cell line NCI-H295-R. These results support the hypothesis that at least some of the endocrine changes observed in the patients may be mediated by endogenous cytokines. The endocrine profile of tuberculosis patients would favor a reduction of protective cell-mediated immunity and an exacerbation of inflammation leading to perpetuation of the lung injury and to the hypercatabolic condition that characterizes this disease.

Comment in

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