

IMMUNOMODULATING EFFECT OF LOW-ENERGY LASER RADIATION IN THERAPY OF BRONCHIAL ASTHMA

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Asthma is a chronic disease underlied by respiratory tract inflammation mediated by a variety of cells, especially eosinophils and mast cells. In predisposed subjects, this process is associated with enhancement of the respiratory response to various stimuli and results in symptoms of bronchial obstruction which is typically self-resolving or reversible by therapy.

Mast cells and eosinophils, which can release several inflammatory mediators, have a key role in inflammation associated with both allergic and nonallergic asthma.

Inflammation severity as been shown to be related to participation of T lymphocytes, fibroblasts, epithelial and other cells.

A great role of immune mechanisms in the onset and progression of bronchial asthma has been documented (2).

Impairment of T cell-mediated immunity is seen as a major pathogenetic determinant of bronchial asthma. The impairment occurs as T suppressor deficiency which results in the release of B lymphocytes and autoantibody formation and T helper deficiency which eventually causes antibody production.

Other immune mechanisms can intervene, with resultant tissue damage.

Pathogenesis of bronchial asthma remains far from elucidated, and an adequate, pathogenetically relevant therapy is not available.

Patients with bronchial asthma need a prolonged intake of drugs whose efficacy is often low.

With this in mind, we used low-intensive laser radiation (LILR) to improve the effectiveness of bronchial asthma therapy.

Goals of the Study

- (1) Identification of an immune imbalance in patients with bronchial asthma.
- (2) Evaluation of laser therapy effects on the immune status of patients with bronchial asthma.
- (3) Assessment of LILR efficacy in bronchial asthma.

Materials and Methods

Immunoassays were carried out in patients who sought pulmonary care for bronchial asthma: determination of blood counts of T and B lymphocytes, T helper and T suppressor subpopulations, circulating immune complexes, neutrophil phagocytic activity, and immunoglobulins A, G and M.

T lymphocyte counts were obtained using a rosette test with ram erythrocytes, and T helpers and T suppressors were measured with a theophylline rosette test. The neutrophil phagocytic activity was determined through the phagocytic index (percentage of phagocytes) and the phagocyte number (a mean number of digested particles per phagocyte). Serum immunoglobulins A, M and G were assayed with a Mancini immunodiffusion test.

A total of 48 patients with moderate-to-severe asthma were examined. The patients ranged in age from 35 to 58 years.

Bronchial asthma was diagnosed on the basis of medical histories, clinical and X-ray findings, and external respiration tests. All patients were followed up by a pulmonologists and received a basic therapy which included corticosteroids in eight cases.

No clinical improvement was seen, and LILR was added to treatment of 28 patients (group 1). Twenty other patients remained on the basic therapy (group 2). Twenty-four normal subjects who underwent blood immunoassays made a control group.

Laser therapy used a semiconductor low frequency infrared laser with a wavelength of 0.89 μm .

Laser irradiation was applied to the chest skin, an adrenal projection (a lumbar area at the level of T12-L2), a thymus projection (a chest area at the level of the second rib) and a vascular bundle projection (a left supraclavicular area). A total dosage was 2.62 J/sq.cm.

Blood immunoassays were carried out before drug treatment, at two weeks and before and after LILR.

Clinical Results

Blood immunoassays were performed in 48 patients with bronchial asthma. The patients had a 43 percent higher helper activity of T lymphocytes as compared to controls and showed B lymphocytosis, a 39.8 percent lower T lymphocyte suppressor function and a 1.9-fold higher immunoregulation T helper/T suppressor index.

Immunological evaluation of group 1 patients after LILR showed a 24 percent lower T lymphocyte helper function, a 29 percent higher suppressor function, a 18 percent decrease in B lymphocyte counts and a decrease in T helper/T suppressor ratio from 5.7 to 3.1. Patients who remained on drug therapy alone showed no significant improvement of the immune status.

The clinical response was seen in all patients who were treated with low-intensive laser radiation: the general condition improved, the frequency of asphyxic episodes decreased from six to one-two a day, dependence of inhaled broncholytics decreased, dyspnea severity ameliorated starting from the third laser treatment, and bronchial secretion release improved. Auscultation and spirometric findings significantly improved, and the incidence of rale decreased after LILR.

Discussion

The study has shown that an abnormal immune status in patients with bronchial asthma. Conventional drug therapy did not improve it. A higher T lymphocyte helper function, B lymphocytosis, depressed T lymphocyte helper function and a high immunoregulation index (T helper/T suppressor ratio) persisted in patients at two weeks following the start of basic therapy plus broncholytics.

LILR had a modulating effect on the immune status of the patients seen a significant change in lymphocyte subpopulations and a nonspecific systemic response.

An important result of LILR therapy was correction of the T helper-T suppressor regulatory system which controls intensity of the specific immune response. The efficacy of laser therapy was suggested by a 29 percent increase in counts of suppressor T lymphocytes which can blunt the immune response and hence trigger autoaggression in the presence of autoimmune tissue damage.

Another important finding was reduction in the immunoregulatory T helper/T suppressor index from 5.7 before laser therapy to 3.1 after it. An increase in the T helper/T suppressor ratio, which is related mostly to a deficient T suppressor count, is ubiquitous in diseases with an autoimmune determinant, and is usually seen at the exacerbation-related peak of their activity. Therefore, the decrease in the ratio after laser therapy was indicative of a lower activity of immune mechanisms which can cause tissue damage.

CONCLUSIONS

The use of LILR in patients with bronchial asthma produced a clinical response which presented as an improved general condition, a decrease in asphyxia attacks from five-six to one-two a day, a lower requirement for broncholytics, a lower rate of dyspnea episodes and better bronchial secretory output.

LILR improved the immune status: the lymphocyte helper activity decreased by 24 percent, the suppressor activity grew by 29 percent, B lymphocyte counts decreased by 18 percent and the immunoregulatory T helper/T suppressor index declined from 5.7 to 3.1.

REFERENCES

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