Inflammation, Asthma and Tumor

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ABSTRACT

Inflammation is a risk factor for the development of many types of neoplasia, including skin, colon, gastric, and mammary cancers, among others. The role of inflammation in lung neoplasia is influenced by many other factors. Asthma is a very common chronic disease that occurs in all age groups. Some studies reported a positive association between asthma and lung cancer, even in never-smoking patients.

Key Words: Inflammation, lung neoplasia, Asthma

Received 02.03.2013 Accepted 09.04.2013

Inflammation is a risk factor for the development of many types of neoplasia, including skin, colon, gastric, and mammary cancers, among others. Chronic pulmonary diseases, such as chronic bronchitis and asthma, predispose to lung neoplasia (1, 2). The role of inflammation in lung neoplasia is influenced by many other factors, for example; genetic susceptibility, pharmacologic modulation of inflammatory pathways, pro- and anti-inflammatory pathways. Identification of molecular mechanisms that govern the association between inflammation and pulmonary neoplasia could provide novel preventive, diagnostic, and therapeutic strategies for a disease in which few biomarkers currently exist (3-5).

Asthma is a very common chronic disease that occurs in all age groups. Its high prevalence has significant health costs, which are even higher in the most severe disease forms. Lung cancer has the highest incidence of all cancers in the developed world and is an important cause of mortality. Atopic constitution, including different manifestations of allergy and asthma, are possible risk factors for lung cancer, above all in never-smokers. Given the high asthma prevalence and lung cancer incidence and mortality in developed countries, this association would have important public health implications. But the association and the underlying physiopathological mechanisms, however, seem to require further studies (6-8).

The high prevalence of asthma means it has significant health costs, increasing in the most severe disease forms. Lung cancer has the highest incidence of all cancers in the developed world and is an important cause of mortality. Patients with lung cancer are a big economic burden on health services, both in direct and indirect costs. Atopic constitution, including different manifestations of allergy and asthma, are possible risk factors for lung cancer, above all in never-smokers; other studies, however, do not find this association (9-11). Given the high asthma prevalence and lung cancer incidence and mortality in developed countries, an association between the two disorders would have important public health implications. Even if the association between allergy and cancer risk is based on epidemiologic studies, the exact nature of the association is still controversial. Some studies defend the allergy protective effect, based on the "enhanced immune surveillance theory", with stimulated immune systems being better able to detect and destroy malignant cells (12-15). Other works, based on the theory of chronic antigenic stimulation, which would be a stem-cell pro-oncogenic mechanism, suggest, on the contrary, that allergy is associated with a higher cancer risk. In atopic syndrome there is the persistence of a chronic inflammatory condition (16, 17). Chronic airway inflammation has been suggested to be a carcigenogenesis mediator; this could happen through different mechanisms. First in the inflammation cell damage and repair process seems to cause permanent abnormalities in
the damage of tissue. Second Chronic inflammation results in a high cell turnover rate with a higher possibility of spontaneous mutation and it can reduce the clearance of inhaled toxins and carcinogens that are found in the bronchoalveolar epithelium or it can cause greater susceptibility to oxidant-induced alterations. Also, antioxidant levels in the respiratory tract of patients with asthma are low so free radical-induced cell ageing and oxidants cause direct damage to Genom, which can contribute to lung carcinogenesis. Third IL-6 plays a key role in the host defence mechanisms; it is the main cytokine detected in lung tumor and inflammation tissue, and can be found at high levels in epithelial cells and peripheral blood of asthma patients. Fourth Asthma treatment with corticosteroids increases both the survival and the proliferation of basal epithelium cells, with high expression of Bcl-2 protein, an epithelial cell apoptosis inhibitor and this can suppress Immune system. Histamine, the main mediator in type 1 allergic reactions, seems to protect NK cells from oxidative reactions, as well as from inhibition and synergism with other cytokines to stimulate T-cell activity. The addition of histamine to certain cancer immunotherapy regimes has shown improved survival results and extended remissions. There are very little data for comparing lung cancer risk between patients suffering from allergic asthma and those suffering from non-allergic asthma (18–22). A study of a meta-analysis reported a positive association between asthma and lung cancer, even in never-smoking patients. Another potential confusion factor is the life style related exposures associated with asthma and lung cancer. However, they can have a lower effect than smoking, since patients with a history of asthma seem to tend to avoid other cancer-related exposures due to the symptoms, and the different preferences in food or lifestyle. Therefore a relation between allergy conditions and a higher incidence of lung cancer seems possible but need many other studies (23-25). Therefore, inflammation with trigger and induce of tissue, can be beginning change and remodeling of tissue and lead to transformation of Cells in long time and may be lead to neoplasia and tumor of tissue.

REFERENCES

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