

EOSINOPHILIA IN ALLERGIC DISEASES

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Eosinophilia is an interesting confirmatory observation in the diagnosis of allergic disorders, but it is not pathognomic or invariably present. When eosinophilia is observed in an allergic subject, there is frequently a certain degree of proportionality between the number of the eosinophil cells and the clinical severity of the allergic disease. Most allergic patients exhibit an increase in the eosinophil count in the blood when exposed to substances to which they are allergic. This has been demonstrated by Dragsted (1951) in patients with hay fever by counting the eosinophil cells before and after exposure to mixed pollen. He found a significant increase in the eosinophil count at the same time as the patients developed clinical signs of hay fever.

In addition to the blood, pronounced eosinophilia may be found in the exudates and secretions which form and are possibly discharged in the course of allergic reactions.

According to Godlowski (1953), the explanation of eosinophilia under these circumstances is that the eosinophilic cells absorb the invading antigen and transport it away. In his opinion, there is an afflux of eosinophils, when a foreign protein is immediately destroyed. This theory is worthy of careful consideration, and it explains why substances such as adrenaline and adrenal cortical hormones are effective in allergic diseases, as both prevent the eosinophilic cells from flocking to the site, absorbing the antigen and storing it in the organism. Thus, the explanation of the high eosinophil counts in

exudates in allergic reactions is supposed to be that these cells contain antigen. In this manner, the body gets rid of a large quantity of antigen, so that the allergic reaction is weakened. This theory is supported by the fact that in most instances, improvement occurs when the allergic reactions have lasted for some time, viz. when the organism has got rid of a large quantity of antigen.

A possible competitive cause of eosinophilia in allergic subjects is the consequence of the chronic stress to which some of these patients are submitted. Patients of this category are apt to develop mild adrenal cortical insufficiency, that is to say only the glucocorticoid fraction is reduced, while the aldosterone production remains unchanged; in other words, the patient does not develop Addison's disease. Albeaux-Fernet et al. (1957) have pointed out that patients with chronic diseases and chronic asthenia exhibit a disturbance of adrenal cortical function, i.e. a reduction in the production of 17-hydroxyketosteroid (Compound F), whereas that of 17-desoxycorticosterone (Compound B) is increased. This alteration in cortical secretion is stated to be due to a disturbance of the enzymatic processes.

For a long time, investigation of the adrenal cortical function has been a link in allergic research. As early as 1949, Erikson-Lihr and her associates submitted their studies on adrenal cortical function in allergic patients. In a large number of cases they found signs of reduced adrenal cortical function.

In 1953 Andersson, investigating a series of 25 patients with severe bronchial asthma, found pronounced eosinophilia as well as reduced urinary excretion of 17-ketosteroids and corticoids in all cases.

These findings may be interpreted as a sign of reduced adrenal cortical function. In a further attempt to study the adrenal cortical function, Andersson carried out stress tests, using subcutaneous injection of histamine, 0.5 mg, and counting the eosinophils before and half an hour after the injection. These experiments included 50 patients with severe asthma and 50 normal subjects.

After the injection of histamine, the allergic patients exhibited an increased eosinophil count, whereas the normal subjects showed a fall or no alteration in the count.

Since histamine stimulates the hypophysis to produce ACTH, this can explain the fall in the eosinophil count in normals. This fall does not appear in allergic subjects whose adrenal cortical function is relatively reduced. The increase in allergic subjects is due to the mobilization of eosinophilic cells by histamine.

The investigations into the cause of eosinophilia in allergic patients has not yet passed the incipient stage, and they are based chiefly on working theories. Continued research within this field is required and may at the same time be expected to afford further penetration into the problems of allergy.

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