CORRESPONDENCE

Aspirin Sensitive Asthma

by Prof. Dr. med. Winfried J. Randerath, Dr. med. Wolfgang Galetke,
in volume 46/2007

Provocation Tests Do Not Work in Such Patients

In response to the impressive scientific review article by Randerath and Galetke, we want to share our own experiences with the treatment of asthma and nasal polyps, in these times of increasing funding shortages, to show that there are simpler solutions to the problem. Small practices cannot present high case numbers or evidence-based study designs. We tested the tolerability of salix (willow) and acetylsalicylic acid (ASA) in 536 patients with allergies using electroacupuncture according to the method of Voll (EAV) in 115 patients with rhinoconjunctivitis and in 65 asthma patients with concomitant migraine or lipid metabolism disorders (1). Of those 180 patients 11 were affected, of whom two reacted to willow and ASA. They were successfully desensitized by using destructive interference (3). This method is used similarly for stimulants such as tree or grass pollen, house dust mites, eiderdown, and synthetic fibers, to prevent nasal polyps and subsequent aspirin sensitive asthma successfully.

The author has been using ASA at low dosages since 1985 after testing for thrombocyte hyperaggregability (2). Platelet agglutinating factor (PAF) is released in every allergic reaction and causes microcirculatory disruptions that may manifest as pain or shortness of breath.

Mast cells are activated not only by IgE but also by type III reactions (IgG). Immunocomplexes develop that activate complement pathways, thereby releasing slow reacting substances or leukotrienes. The simultaneously released heparin and similar glycosaminoglycans stimulate the matrix to release large basic proteins and arginine and lysine from eosinophils, which provides a stimulus to attract more eosinophils. These processes are disrupted by destructive interference, and the mast cell system can calm down.

We cannot accept Randerath and Galetke's statement: "Currently there is no reliable in-vitro method to diagnose aspirin sensitive asthma, so that provocation tests are vital". Our own clinical practice has shown that provocation tests do not work in such patients. We therefore need leukotriene antagonists for such patients only in the initial phase of their treatment. ASA does not have to present a real problem in practice.

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Conflict of interest statement

The author declares that no conflict of interest exists according to the guidelines of the International Committee of Medical Journal Editors.

A Suitable Method Is Required

The authors classify aspirin sensitive asthma in connection with eosinophilia in the polyps themselves and in the tissues of the lower respiratory tract and with the obviously lacking formation of specific IgE antibodies as a non-allergy related process. Rather, they say that aspirin sensitive asthma is due to genetic disruptions of the arachidonic acid metabolic pathway, which results in overproduction of cysteinyl leukotrienes with subsequent overproduction of inflammatory mediators. In this process, the eosinophils are included only as the source, especially for LTC₄ synthase; this does not do justice to the position of eosinophils in the immune system as per current knowledge. To detect eosinophils in tissue and peripheral blood circulation, the histocytochemical test for the enzyme phenoloxidase, after treatment with methanol, has proved to be suitable. Morphologically, the involvement of eosinophil granulocytes in cytotoxic as well as antibody forming reactions can be proved. Both types of reaction can occur simultaneously and can be triggered by medical drugs (1, 2, 3).

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In Reply:

We thank the correspondents commenting on our article on aspirin sensitive asthma. Dr Schmidt points out the option of detecting eosinophils in tissues and blood by using histocytochemical testing with the enzyme phenoloxidase. The finding could prove the involvement of eosinophil granulocytes in cytotoxic or antibody forming reactions, also in association with medical drugs. We thank the author for pointing this out. However, thus far the literature does not include

reports of immune reactions on a cellular or serological basis in aspirin sensitive asthma. Specific IgE or IgG antibodies have not been found. The pathophysiological factors known so far include polymorphisms of key enzymes in the synthesis of leukotriene and the cysteinyl leukotriene receptors (1). Cyclo-oxygenase inhibition by using non-steroidal anti-inflammatory drugs results in excess production of cysteinyl leukotrienes (2), whose unfavourable effect is particularly pronounced owing to increased sensitivity of the mucosa. An accumulation of eosinophils in the tissue does not only mean increased production of cysteinyl leukotrienes but also of chemotactic factors and a delay in apoptosis, which substantially contributes to polyp formation and recurrence. The eosinophils therefore have a central role in the pathology (3).

Frick's reports on electroacupuncture according to Voll do not currently provide a prompt for us to change the methods described in our article. We have not found any further reports in the literature. A Medline search (19 December 2007, search terms "electroacupuncture" and "asthma") did not yield any articles on aspirin sensitive asthma. The data on the reaction to aspirin sensitive asthma as presented by Frick should be compared with the current diagnostic gold standard

(provocation tests). Whether, in addition, destructive interference has an effect that is equal to that of adaptive deactivation will need to be investigated in a prospective study. DOI: 10.3238/arztebl.2008.0221

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As a member of the National Advisory Board-MSD Prof. Randerath receives honoraria for lecturing and reimbursement of travel expenses.