Helicobacter Pylori and Chronic Urticaria

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Dr Mervi Liutu defended her thesis on February 8, 2002 at the University of Turku, Finland. Opponent was Docent Urpo Kiistala (*left*) from the University of Helsinki, and Chairman Professor Christer Jansen (*right*) from the University of Turku.

Chronic urticaria (CU) is a disease probably triggered by several, possibly simultaneously acting factors. The connection between Helicobacter pylori (H. pylori) and CU has been proposed, since the antibiotic therapy aimed at eradication of H. pylori may have beneficial effects on symptoms of patients with CU. This study was undertaken to establish the etiologic factors in CU and to clarify the role of H. pylori in CU symptoms.

Of the 107 Finnish patients with C U investigated in this study, pathologic findings were seen in 92 patients, including elevated serum total IgE levels, positive skin prick or scratch test results, concomitant diseases and infections. Results of routine laboratory investigations were within normal limits in all patients. Autoimmune reactivity (including hypothyroidism, insulin dependent diabetes mellitus, multiple sclerosis, ulcerative colitis and positive rheumatoid factor) was found in nine patients. Dental or respiratory infections were found in 16 patients. Four patients had lichen planus, two had psoriasis, and six had a previous history of atopy.

Over half (55) of the patients reported recent abdominal complaints, suggesting a role for the gastrointestinal tract in the pathogenesis of CU. Endoscopy was performed and biopsies were taken from 32 patients. According to the histopathologic examination, 30 patients had gastritis. Histologically, H. pylori was verified in 24 patients, two of them being H. pylori seronegative. Subtotal villous atrophy was found in one patient, and additionally, elevated antigliadin IgA antibodies were seen in four patients. Lactose intolerance was diagnosed earlier in two patients. Active gastritis might weaken the gastric barrier and thus increase the penetration of e.g. food allergens, histamine or H. pylori toxins to the vascular system, and by this means induce urticaria symptoms in some patients. On the basis of these observations, endoscopy is warranted as a basic examination of patients with CU, especially of patients with dyspeptic symptoms or elevated serum H. pylori IgG antibody levels.

The prevalence of H. pylori infection among patients with CU was of the same magnitude than the overall prevalence in the general population in Finland. However, after H. pylori eradication therapy, 22% of H. pylori-infected patients with CU became free of urticaria, and 56% felt their urticaria symptoms to be alleviated. This suggests that antibiotic therapy aimed at H. pylori infection may have beneficial effects on CU symptoms.

Overall, total concentrations of IgE were slightly above the normal range in the sera of H. pylori-infected patients, the level varying from 111

to 1140 kU/l. To evaluate the role of H. pylori specific IgE in CU, an immunoblotting analysis and H. pylori specific RAST were done, and the presence of IgE-containing cells was demonstrated by immunostaining in gastric biopsy specimens. In immunoblotting analysis, IgE-binding epitopes were found in the two H. pylori strains tested. However, the RAST assay revealed H. pylori-specific IgE only in one out of 34 patients with CU. In the gastric mucosa, H. pylori-infected patients had a significantly higher density of IgE-containing cells when compared to non-infected patients skin or allergic disease. Furthermore, no significant difference was found in the density of IgE-containing cells between H. pylori-infected and -non-infected patients with CU. These results suggest that there is no need to examine H. pylori-specific IgE in patients with CU. Total serum IgE, however, is a useful parameter to investigate in CU, as it suggests various IgE-mediated diseases, including food allergies, and parasite infections.

In conclusion, the results of this study suggest that patients with CU benefited from antibiotic therapy aimed at eradication of H. pylori infection. Serum IgE levels were overall slightly elevated in H. pylori-infected patients with CU, and H. pylori specific IgE response was verified, but the IgE-mediated connection between H. pylori and CU remained questionable.

List of original communications

I. Liutu M, Kalimo K, Uksila J, Kalimo H. Etiologic aspects of chronic urticaria. Int J Dermatol 1998; 37: 515–519.

II. Liutu M, Kalimo K, Leino R, Uksila J, Kalimo H. Chronic urticaria and Helicobacter pylori infection. J Dermatol Treat 1998; 9: 31–33.

III. Liutu M, Kalimo K, Uksila J, Savolainen J. Extraction of IgE-binding components of Helicobacter pylori by immunoblotting analysis in chronic urticaria patients. Int Arch Allergy Immunol 2001; 126: 213-217.

IV. Liutu M, Kalimo K, Kalimo H, Uksila J, Leino R. Mast cells and IgE-containing cells in gastric mucosa of Helicobacter pylori infected and non-infected patients with chronic urticaria. Submitted.