EDITORIAL

HELICOBACTER PYLORI AND ALLERGIC DISEASE

Since 1983 when patients with type B gastritis were found to be infected by a hitherto unknown type of bacteria showing a certain similarity to Campylobacter*, the prognosis of this disease has changed considerably with the availability of highly effective antibiotics. The Gram-negative bacterium was identified as* Helicobacter pylori*, which causes most cases of gastritis and of peptic and duodenal ulcer. It has also been found in patients with gastric adenocarcinoma and B-cell lymphoma of mucosa-associated lymphoid issue (MALT) type*[1,2].

H. pylori *has certain properties than enable it to cross the gastric mucosa easily, stimulating the production of inflammatory cytokines due to the activity of urease, which is present in large quantities in the bacterial membrane. The mucosal lesion is mediated by mucinase and phospholipase, which alter mucous secretion, and the action of a vacuolizing cytosine.* H. pylori *also produces factors that stimulate interleukin-8 secretion, production of platelet-activating factor, which causes gastric acid hypersecretion, and programmed cell death of epithelial cells, all of which contributes to the epithelial lesion*[1].

Subsequently, H. pylori *infection was related to particular allergic diseases when Kolibasova et al*[3] *achieved remission of chronic urticaria in* H. pylori*-infected patients through antibiotics against this infection. Since then, numerous studies have been published confirming or disconfirming this initial experience. To date, the causative role of* H. pylori *in the etiology of chronic urticaria has not been confirmed and this criterion has predominated, despite clinical experiences relating chronic urticaria with* H. pylori *infection*[4-7]*. However, the possibility that an immunological mechanism related to* H. pylori *is involved in some cases of chronic urticaria has not been ruled out. Thus, Bakos et al*[8] *studied 33* H. pylori*-infected patients with chronic urticaria and mild gastric symptoms. All patients showed high concentrations of IgG antibodies against* H. pylori *and in 31 of these (93.9 %) specificity was to lipoprotein 20 (Lpp20; molecular weight: 19 kDa) of the bacterial surface. Thirteen patients also showed IgA antibodies against* H. pylori *and 6 of these (46.1 %) showed anti-Lpp20 antibodies. These results were compared with those in another group with chronic urticaria and without* H. pylori *infection who were seronegative. These authors suggest that there may be an underlying autoimmune mechanism that remains to be investigated.*

Other studies have concentrated on evaluating the association between food allergy and another dominant antigen on the surface of a particular strain of H. pylori-*CagA (cytotoxin-associated gene A), with different results. Thus, Figura et al*[9] *found anti-CagA antibodies in 65 % of a group of 38* H. pylori-*infected adult patients with food allergy but in only 28 % of* H. pylori-*infected controls without food allergy. Consequently, these authors deduced that infection by CagA-positive* H. pylori *increases the risk of developing food allergy. In contrast, Corrado et al*[10] *found no differences in anti-CagA IgG titers between children with atopic dermatitis as the sole clinical manifestation of food allergy and children with allergic asthma and without food hypersensitivity. In both studies, patients with food allergy showed IgG antibodies against* H. pylori. *However,* H. pylori *antigens are unlikely to induce allergy because the finding of* H. pylori-*specific IgE antibodies is not common*[11].

A different feature of Helicobacter *infection arousing greater consensus concerns the recently put-forward "hygiene hypothesis"*[12] *according to which the increased prevalence of allergic diseases is related to the lower incidence of infectious diseases in the most developed countries. This reduction is due to improved conditions of hygiene and the protection conferred by vaccines against numerous infections, which has shifted the balance in Th1 and Th2 responses in favor of Th2 responses, which, as we know, are involved in allergic reactions*[13]. *Several recent studies demonstrate the lower prevalence of allergic disease in individuals with antibodies to bacteria transmitted through the orofecal route (*H. pylori *and others) than in those who tested negative*[14,15].

As can be seen by this brief commentary, H. pylori *seems to be implicated in several aspects of allergic disease. Other aspects not discussed are its possible role in autoimmune processes, such as Sjögren's syndrome or autoimmune thyroiditis, and its involvement in many other processes not directly related to immune response disorders*[4,16].

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