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***Yersinia enterocolitica*: another factor in the pathogenesis of chronic urticaria?**

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Chronic urticaria (CU) defined as recurrent weals for > 6 weeks is thought to have numerous causative factors including a large variety of infectious conditions, food intake and drugs, but in one-third of the cases the origin remains unclear.

We planned a study to investigate the influence of *Helicobacter pylori* infection on the treatment and outcome of patients with CU. This retrospective study comprised 74 patients with CU who had a positive urea breath test and 74 *H. pylori*-negative patients with CU. All patients underwent extensive diagnostic investigations to search for trigger factors, including serological testing for *Yersinia enterocolitica* IgA and IgG antibodies. Interestingly, *Yersinia* antibodies were found in 53.5% of all patients with CU. In the literature, the prevalence of *Yersinia* antibodies in healthy blood donors in Germany was found to be up to 43% and may indicate the widespread presence of subclinical *Yersinia* infections in healthy populations.¹

In our study, patients with proof of a former *Yersinia* infection had a significantly longer duration of symptoms compared with *Yersinia* antibody-negative patients (43.6 vs. 22.4 months, $P = 0.040$). The infection rate of male and female patients was identical, and the prevalence of *Yersinia* antibodies was not dependent on age. *Yersinia* antibody-positive individuals had slightly higher levels of total IgE (173.2 vs. 162.1 kU/L) and C-reactive protein (9.2 vs. 7.5 mg/L), but this difference did not reach significance. The presence of *Yersinia* antibodies was independent of proof of an active *H. pylori* infection.

One important trigger factor for CU is a pseudoallergic reaction to food. Normally, the intestinal barrier prevents the immune system from an immoderate contact with food allergens. The passive intestinal permeability of patients

with *Yersinia*-triggered reactive arthritis was studied by Lahesmaa-Rantala *et al.*,² using differently-sized polyethylene glycols. This controlled study was carried out at least 1 year after the onset of *Yersinia* infection, and patients had neither acute gastrointestinal nor joint symptoms. The passive permeability of the intestinal mucosa to the larger molecules in patients with a history of *Yersinia* infection was found to be increased for an unexpectedly long time after the acute *Yersinia* infection.² Buhner *et al.*³ were able to show that patients with CU also present with greater gastroduodenal permeability compared with healthy controls, and this effect was independent of *H. pylori* infection.

In conclusion, increased intestinal permeability due to *Yersinia* infection might be an additional trigger factor in the pathogenesis of CU. Prospective randomized controlled studies are necessary to further investigate the role of *Yersinia* infection in CU.

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