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Research

Helicobacter pylori in Dermographic Urticaria

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Abstract

Introduction: *Helicobacter pylori* infection is thought to be a causal factor in urticaria. Dermographism is the most common type of physical urticaria in which the etiological factors are unclear.

Objective: We aimed to determine the etiologic role of *Helicobacter pylori* infection in dermographism.

Methods: We assessed the frequency of *Helicobacter pylori* infection in 22 patients with dermographic urticaria and 14 patients with *Helicobacter pylori* positivity were evaluated whether they have dermographism. The presence of *Helicobacter pylori* infection was determined by rapid urease test.

Results: Six of 22 patients had *Helicobacter pylori* infection. And 5 patients with *Helicobacter pylori* positivity have dermographism.

Conclusion: Our results indicate that the pathogenic role of *Helicobacter pylori* in dermographic urticaria is highly doubtful. It may be a triggering factor in patients with dermographic urticaria.

Introduction

Urticaria is a common clinical disorder with complex triggering factors. Current etiological mechanisms may be separated into physical and non-physical subgroups. Urticaria has provided evidence that enteric infection with Helicobacter pylori may induce the disease [**1, 2, 3, 4**].

Dermographic urticaria (DU) is one of the most frequent variants of physical urticaria. It may be characterized by a linear weal and flare reaction which occurs in response to rubbing or stroking of the skin. It usually resolves within 1 to 2 hours. Patients have no associated systemic signs and symptoms. It is generally accepted that *Helicobacter pylori* (H pylori) infection plays an etiologic role in the development of chronic urticaria. We designed a case-control study of a population-based sample to assess the prevalence of H pylori infection in patients affected by DU [**1, 5, 6, 7**].

Methods

Twenty-two patients with the presumptive diagnosis of DU underwent precise history taking, dermographism and endoscopy. Clinical diagnosis of dermographism (symptomatic) was performed by development of linear weal and flare induced by firmly stroking the skin of the back using the edge of a pencil. Symptomatic dermographism was differentiated from simple dermographism by the presence of itching and a tendency of easy-wealing by minor trauma. Patients having chronic idiopathic urticaria and other types of physical urticarias were excluded by appropriate tests. Challenge tests for cold, heat, cholinergic, and delayed-pressure urticaria yielded negative results.

Presence of any concomitant or previous gastrointestinal disease or complaints and drug intake were

recorded. After detailed physical examination each patient was studied according to routine biochemistry. Patients were not on any continuous treatment. Six patients were receiving H1 antihistamines during the symptomatic phase. They have stopped antihistamine therapy for at least 48 hours.

Fourteen patients with helicobacter positivity (9 women and 5 men; mean age 36.6 years) were used as control group. Presence of any concomitant or past gastrointestinal disease was recorded. They underwent a full physical examination. In this group dermographism positivity is defined as an abnormal wealing response of the skin to moderate local trauma with the presence of itching. Informed consent was obtained from all patients and controls.

H pylori infection was identified by the same method in patients and controls. Patients undergoing elective endoscopy were routinely tested for H pylori using the rapid urease test (CLOtest). At endoscopy an antral biopsy was immediately inserted into the gel of a prewarmed CLOtest and then kept at 37°C on a warmer. CLOtests were examined for a characteristic color change from yellow to red at the end of the endoscopy session. If the CLOtest was red, it was recorded as positive.

A χ^2 exact test was used for comparison between those with and without symptomatic dermographism and the presence or absence of *H. pylori* infection.

Results

Twenty-two patients (14 women and 8 men) diagnosed with DU were enrolled in the study. The patients' ages ranged from 18 to 52 years (mean, 28.6 years). The mean age of onset was 21.6 years (range: 12 years to 51 years). The peak age of onset was during the second decade. The duration of disease extended from 6 months to 12 years (mean: 14 months). The mean duration at last follow-up was 9 months. The duration was longer than 5 years in 4 patients and longer than 10 years in 2 patients. The symptoms were seen in all regions of the body with a less frequent swelling of mucosa. The scalp and genitalia were less often affected. Five patients mentioned present or past gastrointestinal symptoms. Laboratory tests revealed no significant abnormalities. A moderate elevation of total IgE was recorded in six patients (atopic ones and the other patients). According to physical examination 3 patients were recorded as atopic. There was no temporal relationship between the onset and exacerbation of atopic complaints and DU. No malignant or other systemic diseases were found in patients with dermographism.

Six patients of 22 had H pylori infection (**Table 1**). Two patients infected with H pylori and 3 of 16 not infected, had a history of gastric symptoms. In the control group these symptoms were noted in 4 patients who have no dermographism. Five patients with H pylori positivity have dermographism (**Table 2**). Endoscopic examination revealed slight gastric erosions in 6 patients. As we compaired the groups in each other conversely there were no statistically significant differences between the DU patients and the controls ($p>0.05$).

Discussion

Dermographism occurs in 25% to 50% of the normal population, yet only 5% of patients are highly symptomatic and generally it is defined as 'idiopathic' [**8**]. In the literature, there are no detailed or classified data on etiologic factors in DU. The etiologic role of H pylori in urticaria particularly in DU is an unknown point. In our study we aimed to evaluate the relationship between DU and H pylori infection. There are numerous invasive and noninvasive tests available for the diagnosis of H. pylori infection [**9**]. When endoscopy is clinically indicated, it has been recommended that invasive techniques should be used. We used one invasive method of diagnosis—the rapid urease test (CLOtest). It gives a relatively quick diagnosis, and is less expensive than other invasive diagnostic techniques (histology, microbiology).

We found the peak age of onset of dermographism to be in the second decade, and the mean at last duration of 9 months. The peak age of onset was during the third decade and the mean duration at last follow-up was 5.1 years in a study. The discrepancy between our results and those of the literature [**8**], in terms of duration and peak onset are probably due to the patient selection.

Histamine is likely the primary mediator, as antihistamines are effective in treatment. The role of other mast cell mediators, such as prostaglandins, leukotrienes, platelet-activating factor, serotonin, and chemotactic factors for

Table 1. *Helicobacter pylori* Positivity in Dermographic Urticaria

	H. pylori (+)	*H. pylori* (-)	Total
Patients	6	16	22

Table 2. Dermographism in Patients with *H. pylori* Positivity

	Dermographism (+)	Dermographism (-)	Total
Controls	5	9	14

eosinophils and neutrophils, are not as clearly defined [**5, 6, 10, 11**]. In some cases the passive transmission of dermographism to the skin of healthy individuals may suggest an IgE-mediated immune reaction [**8, 12**]. It is hypothesized that IgE-sensitized mast cells react to an unidentified antigen induced by stroking of the skin. The possible role of H pylori as an etiologic agent in urticaria seemed a reality. The mechanisms that might explain such an association were related to a specific immunoglobulin E (IgE) immune response to antigens of H pylori by cross-reaction between H pylori antigens and gastric parietal cells. Furthermore it may cause inflammation in the gastrointestinal tract, which may facilitate the absorption of antigens or unmask existing antigens [**1, 2, 4, 5, 6, 13, 14**]. Although mast cell activation can also occur indirectly secondary to an IgE-directed mediator release, our study results do not support this idea. Because a moderate elevation of total IgE was recorded in only six patients.

It was reported that atopic disorders were not seen high in DU [**6**]. We recorded three patients with atopy and didn't find any temporal relationship between the onset or exacerbation of atopic complaints and DU.

Our results indicate that the pathogenic role of H pylori in DU is highly doubtful. Even tough H pylori may have a role in some cases of dermographism, it appears that it may only be responsible for a minority of cases. Because H pylori is so common infection seen in the population. And also dermographic urticaria is one of the most seen physical urticaria. Even in the presence of active infection dermographism will have causes unrelated to H pylori. It may be a trigger factor in patients with DU.

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