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ARTICLE

Eosinophils and Mast Cells in *Helicobacter Pylori* Infected Gastric Mucosa*

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Although Helicobacter pylori (HP) is frequently associated with chronic active gastritis and peptic ulcers, its exact pathogenic role or the pathomechanism is still unclear. Here, we describe a striking, statistically significant increase of eosinophils in HP infected gastric mucosa compared to HP negative gastritis with similar activity. In both cases, the mean number of the mast cells in the mucosa was comparable, although the individual values showed wide distribution. The source and role of eosinophilia in HP infected mucosa, the potential link between the degree of eosinophilia and the clinical progression, as well as between eosinophils and mast cells require further study. (Pathology Oncology Research Vol 2, No 4, 237–238, 1996)

Key words: eosinophilia, gastritis, Helicobacter pylori

Introduction

Although, there is no doubt that *Helicobacter pylori* (HP) infection can cause gastritis and peptic ulcers, the exact pathomechanism is still not fully understood.[1,2] Certain studies suggest the involvement of allergic components showing specific anti-HP antibodies in the sera and bound to basophils in HP infected patients.[3] In HP induced chronic, active gastritis, the inflammatory cells are mainly plasma cells and lymphocytes, mixed with fewer macrophages and polymorphonuclear leukocytes. But, if the allergic mechanism contributes to the inflammation,[4-7] the presence and activity of mast cells, as well as eosinophils, is expected. The aim of this study was to quantify those cells in HP positive and HP negative gastric mucosa.

Materials and Methods

Twenty-three HP positive (histologically confirmed) and 17 HP negative gastric biopsies were evaluated. The severity of inflammation, at least moderate, was comparable in the two groups. Sections from formalin-fixed and paraffin-embedded materials were stained with HE and toluidine blue, and were also used for immunohistochemistry (monoclonal mouse anti-human mast cell tryptase, Dako; diluted 1:50). Cell counting, performed with ocular micrometer, was restricted to the stromal area (0,3 mm^2/sample) of the tunica propria mucosae thus avoiding the difference in stroma/gland ratio between the samples. For statistical evaluation, t-paired test was used.

Results

Fig. 1 shows the individual cell numbers in each evaluated case. The mean value of the number of mast cells was 107.1 in the HP positive, and almost identical, 105.9 in the HP negative groups. Obviously, there was no statistical difference ($p = 0.950$), but there were large variations within the groups. (These values represent the result of immunohistochemistry since it proved to be more sensitive than the toluidine blue staining).

In contrast, the mean number of eosinophils was more than ten times higher in HP positive (166.0 ± 66.1) than in the HP negative group (13.7 ± 12.4) ($p < 0.001$). The individual values showed wide distribution in the HP positive group, but even the lowest value surpassed the highest level found in HP negative cases.

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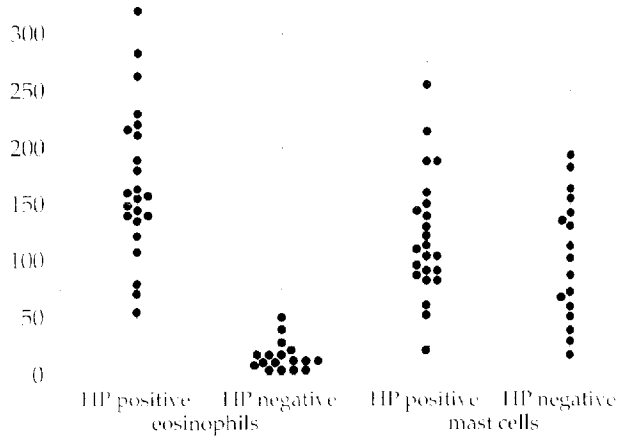

Figure 1. *Number of eosinophils and mast cells in HP positive and HP negative gastric mucosa. The vertical scale shows the number of cells/0.3 mm² stromal area.*

Discussion

It seems that HP infection does not alter the number of mast cells in gastric mucosa. However, the quantitative similarity does not rule out the possibility of the HP mediated degranulation of mast cells because the identification of degranulated mast cells in sections is rather difficult, even with immunohistochemistry. (The available anti-mast cell antibody recognizes a component of the granulum.)

The dramatic and significant increase in the number of eosinophils in HP infected gastric mucosa is slightly astonishing, since tissue eosinophilia occurs primarily in helminthiasis, in allergic diseases, and in certain neoplasias (e.g. Hodgkin's disease), but not, or seldom, in bacterial infections. We assume, that this increase is not a pure coincidence but represents the pathogenetic role of eosinophils in HP infection. The remaining question is: how does it work? Theoretically, either HP has a direct chemotactic effect on eosinophils, or the mucosal eosinophilia is a consequence of other cells' activities. E.g. there is evidence on the potential interactions of eosinophils and mast cells. The main chemotactic factors for eosinophils are the eosinophil chemotactic factor (ECF) and interleukin 5 (IL-5). The former is produced by parasites (ECF-P) and also by mast cells (ECF-A). Therefore, the accumulation of eosinophils in HP infected samples could result either from the direct chemotactic effect of HP, or from the ECF-A. IL-5 can come from T lymphocytes or, again, from mast cells. As we stated above, despite the unchanged number of mast cells, their increased activity could not be ruled out.

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