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RESEARCH ARTICLE

Evaluation of anti-*Helicobacter pylori* IgG level in the serum of patients with Rheumatoid arthritis

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Abstract

Rheumatoid arthritis is an inflammatory chronic disease with an autoimmune pathogenesis. To determine the role of *Helicobacter pylori* as a trigger agent, twenty five patients with rheumatoid arthritis of ages (15-47) years have been investigated and compared with twenty healthy individuals. All the studied groups were carried out to measure the rheumatoid arthritis (RA) IgM, anti-CCP antibody IgG and IgA by ELISA test and by measured anti-IgG antibody level of *H. pylori* by using ELISA and IFAT techniques. The present study showed significant differences (P< 0.05) of anti-*H. Pylori* in sera of RA patients than control group, this lead to suggest that *H. pylori* had a role in pathogenesis of RA.

Introduction

Rheumatoid arthritis is an inflammatory chronic disease with an autoimmune pathogenesis whose aetiology and progression are multifactorial, including a range of immune, genetic, neuroendocrine, environmental and psychosocial factors. An infectious aetiology of rheumatoid arthritis has also been postulated, but, although many germs have been proposed as the triggering agent, none has been identified. [1,2]. Due to a number of factors, *Helicobacter pylori* infection is increasingly recognized as highly prevalent in many populations and of increasing health concern [3]. The role of *Helicobacter pylori* infection is explored in more and more extragastric diseases including rheumatic disorders [4, 5]. Although animal experiments and clinical investigations have suggested a synergistic effect of *H. pylori* on non –steroid anti-inflammatory drugs (NSAIDs) induced gastropathy [6, 7]. Patients with rheumatoid arthritis (RA) often develop ulcers induced by NSAID that used, and *H. pylori* may play a more minor role in inducing gastro duodenal ulcers in patients with RA than in patients without such a disease [8,9]. Proposed mechanisms underlying extragastric pathogenicity associated with *H. pyloi* include a direct effect of the bacterium :activation of inflammatory processes, with a release of cytokines and flogistic mediators with subsequent system effects responsible for the remote manifestations of the disease and finally mimicry between bacterial and host antigens [10,11,]. The aim of the present study was to evaluate seroprevalence of H.pylori IgG antibodies in patients with Rheumatoid arthritis.

Materials and Methods

The study included (25) patients with Rheumatoid arthritis of ages (15 - 47) years from Baghdad Teaching Hospital/Ministry of Health ,those who were diagnosed by Rheumatoid factor (RF) IgM, and anti-CCP antibody IgG and IgA by ELISA technique according to Euroimmune Company (Germany), and (20) health blood donors taken as a healthy control group. All the study groups carried out to measured anti- IgG Ab level of *Helicobacter pylori* by using ELISA and IFAT test according to Euroimmune Co. (Germany).

Statistical analysis:

Comparison of paired data from the groups of subjects was done using T–test, while correlation between groups were analyzed using Person Chi-Square. The computer program which used was SPSS v. 20 [12].

Results and discussion

The present study showed significant differences (P<0.05) in anti-CCP antibody IgG concentration and RF IgM with RA patients than control group as in figure (1 and 2 respectively), also there was significant differences (P<0.05) with the concentration of *H. pylori* IgG in patients group than control group by ELISA technique as in figure (3), and there was twenty percentage of anti-*H. pylori* IgG and IgA positive by IFAT test as in figure (4), and negative anti-*H. pylori* IgG and IgA by IFAT as in figure (5).

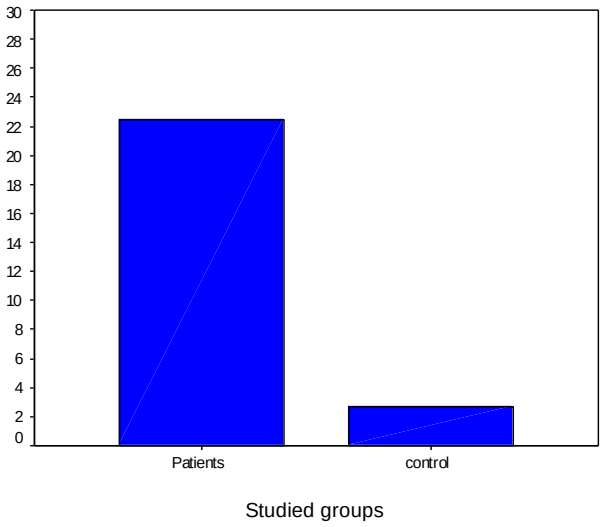

Figure (1): Anti-CCp antibody IgG in studied groups

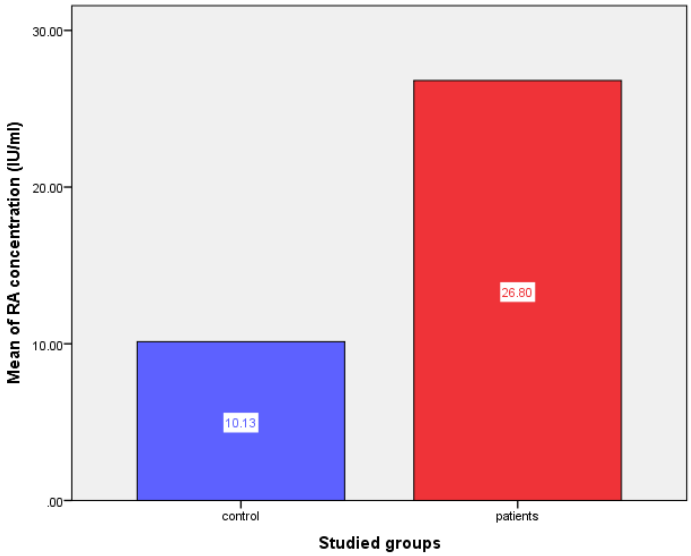

Figure (2): Concentration of Rheumatoid factor IgM in studied groups

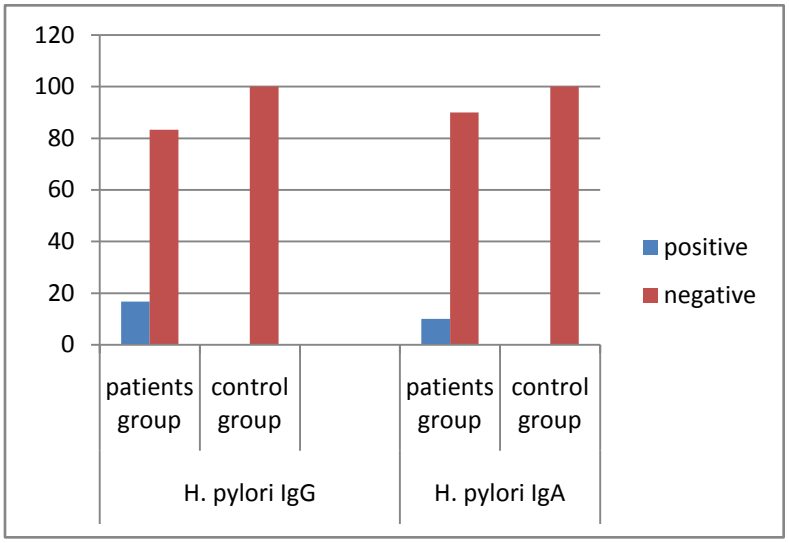

Figure (3): Anti-*H. pylori* IgG concentration in studied groups

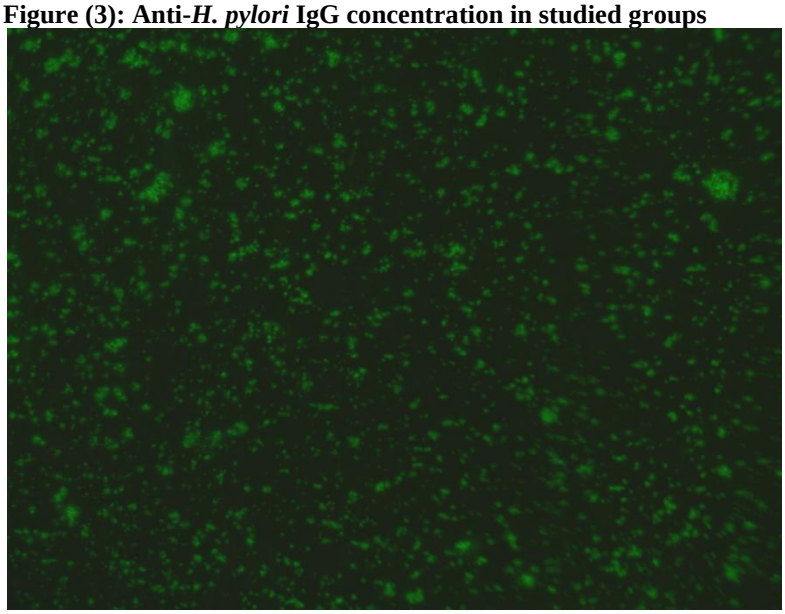

Figure (4): Positive anti *H. pylori* IgG and IgA

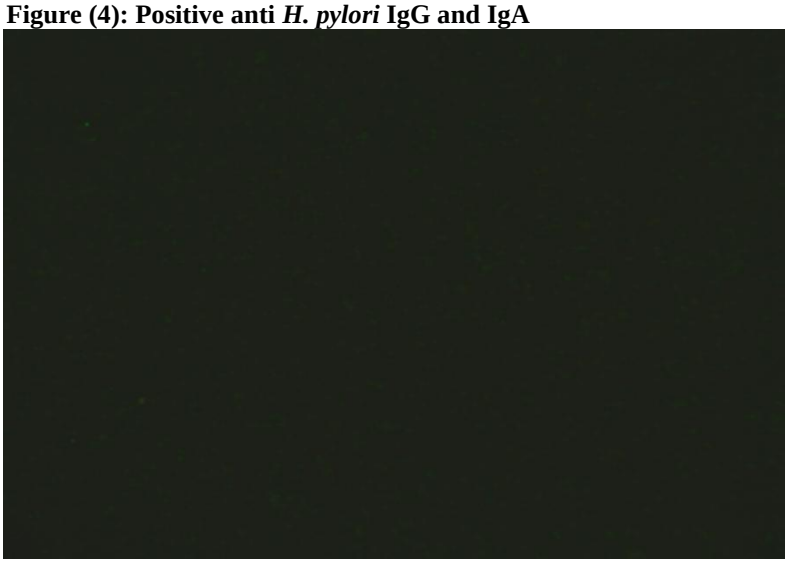

Figure (5): Negative anti-*H. pylori* IgG and IgA

Our study showed agreement with other previous studies that showed the anti-CCP antibody is present in the earlier stage of disease [13]. Previous study showed that anti-CCP combing with RF IgM testing has additional value over IgM-RF testing alone in patients with early undifferentiated oligo and polyarthritis [14]. The prevalence of *H. pylori* infection continues to vary markedly between developing and developed countries and ranges from 3.3%-75.4% [15]. In one study the prevalence of *H. Pylori* infection was (61.4%). [16]. Some studies showed that patients with rheumatoid arthritis had a significantly higher *H. Pylori* seropositive than volunteers [17]. On the other hand, other investigators reported that the prevalence of *H. pylori* was not greater in adult RA patients than normal subjects of the same age in western countries [18].

The above findings seem to indicate the existence of pathogenic role of *H. pylori* infection in rheumatoid arthritis. The bacterium is detected in only one-half of patients with this disease, but, when present, is associated with a higher disease activity, which decline progressively and significantly after eradication. Obviously, the infection does not have an aetiological role, but it may contribute to maintain an inflammatory status in response to the continuous antigenic stimulus induced by chronic infection. The immunological and inflammatory response by the host against the bacterium is determined by the direct or indirect production of various cytokines [19, 20, 21]. The most important cytokine is interleukin-8, which has a potent chemotactic activity for neutrophils and T lymphocytes. Indeed, the increased expression of interleukin-8 leads to the activation an accumulation of polymorphonuclear cells and monocytes in the gastric mucosa with the production of cytokines, such as interleukins-1, 6, 7 and 10 and tumor necrosis factor α, all substances involved in the inflammatory response. It is possible that these factors act not only at a local level, but also in extra digestive areas, thus increasing the expression of inflammatory status in rheumatoid arthritis. An alternative pathogenic hypothesis could be the production of autoantibodies induced by the bacterium through across-reaction mechanism which can act against gastric epithelial cells and other host tissues [22, 23, 24]. The present study leads to suggest that *H. pylori* may play a role as triggering factor of RA

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