Sarcouncil Journal of Internal Medicine and Public Health

ISSN(Online): 2945-3674

Volume- 03| Issue- 02| 2024

Research Article **Received:** 22-01-2024| **Accepted:** 10-02-2024 | **Published:** 25-03-2024

Asthma is Inversely Associated with Helicobacter Pylori Status in Al-Kadhimia Teaching Center

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Abstract: **Background:** - Microbial exposures have been suggested to confer protection from allergic disorders, and reduced exposures to gastrointestinal microbiota have been proposed as an explanation for the increase in asthma prevalence. Since the general prevalence of Helicobacter pylori has been decreasing, we hypothesized that H. pylori serostatus would be inversely related to the presence of asthma. **Methods:** Adults were recruited to participate in the Al-Kadhimiah Teaching Center. Adult asthma cases (N = 64) and controls (N = 40) were identified, and serum IgG antibodies to H. pylori whole-cell antigens was measured. **Results:** As expected, the asthma cases and controls differed with respect to atopy and lung function. Seropositivity to H. pylori was present in 10.9% in asthma groups, while in controls was 92.5% of the study population. Asthma was inversely associated with H. Pylori IgG. **Conclusion:** These data are consistent with the hypothesis that colonization with H. pylori is inversely associated with asthma and is associated with an older age of asthma onset in the population. The data suggest H. pylori as a marker for protection.

Keywords: Helicobacter, Atopy and Asthma, Al-Kadhimia Teaching Center

INTRODUCTION

The prevalence of atopy and asthma has increased worldwide [Anderson, H. R, 2005]. The "hygiene hypothesis," that reduced childhood exposure to microorganisms modifies polarized Th1/Th2 responses leading to more allergic disorders, has been proposed to explain this increase [Strachan, D. P, 2000]. Relevant microbial exposures may include gastrointestinal biota. Intestinal microbiota differs between healthy infants in countries with low or high allergy prevalence, as well as between allergic and no allergic infants [Sepp, E. *et al*., 1997]. Early exposure to orofacial microbes, such as Hepatitis A, appears to protect against allergen sensitization [Bjorksten, B, 2004- Matricardi, P. M. *et al*., 2002], and in Italian military recruits and Danish adults, HAV, Toxoplasma gondii, and Helicobacter pylori are inversely associated with atopy [Matricardi, P. M. *et al*., 2000; Linneberg, A. *et al*., 2003]. Ulcer disease, associated with gastric carcinoma, low guide MAL Tomas, but a role in gastro-esophageal reflux and non-ulcer dyspepsia has not been established. Immunoglobulin G serological test is the non-invasive test of choice for H. Pylori infection in treated patients. We hypothesized that the presence of H. pylori antibodies would be inversely related to asthma, with H. pylori-positive IgG would have a more pronouncer! Inverse relationship with asthma. We found inverse associations of H. pylori status with childhood-onset asthma and allergic disorders [Dooley, C. P. *et al*., 1989]. Helicobacter Infection occurs worldwide; it is much more common in developing countries (up to 80%), were most infections occur in childhood. Is much less common in developed countries. Were childhood infection is low (5-10%), and new adult infection is uncommon. The most common route for infection is the fecal-oral route. Helicobacter Pylori is the most important cause of peptie because the antibody may persist for years; serological analysis is not useful as a document for cure from infection [Kosunen, T. U. *et al*., 1997; Blaser, M. J. *et al*., 2004; Rowland, M. *et al*., 2006; Perez-Perez, G. I. *et al*., 2002]. Also, the urea breath test is the non-invasive test of choice to document the successful eradication of Pylon infection [Blaser, M. J, 2005; Odenbreit, S. *et al*., 2000]. Stool nitrogen test endoscopy and mucosal biopsy are algo important diagnosis tests. Treatment is by a combination of antibiotics and proton pump inhibitors.

STUDY DESIGN

This study is carried out during the period of October 2010 to April 2011. The design of the study is a selection of asthma cases number = 68, from which 24 cases were males and 40 females, and control cases 40, from which 15 cases were males and 25 cases were females; all these cases were admitted to Al-Kadhimia Teaching Hospital for evaluation.

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DOI- https://doi.org/10.5281/zenodo.10870530

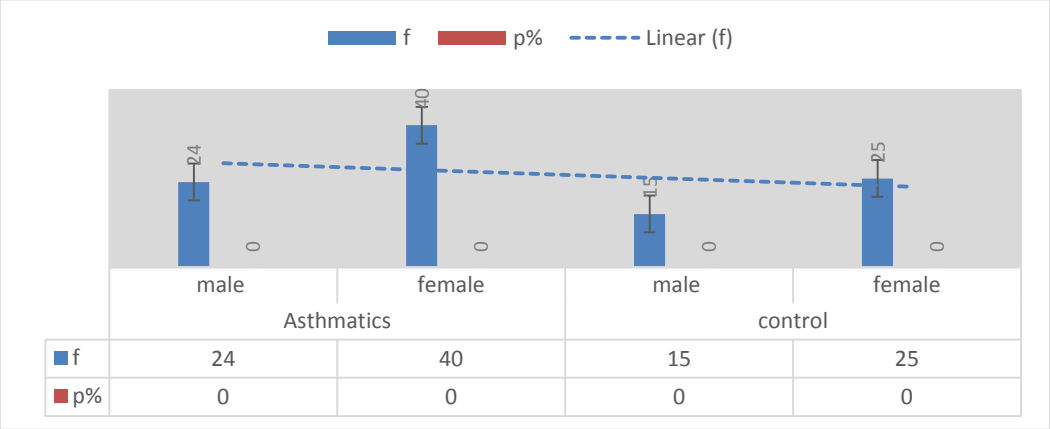

Fig 1: Distributed of cases according to sex

Exclusion Criteria

- Age, all cases, and control were excluded if they are below 18 years old or more than 65 years old.
- The current smoker has a history of more than ten packs and a year of tobacco Use.
- Unstable cardiac disease.
- Uncontrolled hypertension.
- Lung disease other than asthma.

Serum Antibody analysis

Serum anti-H. Pylori IgG levels were determined by ELISA using whole-cell antigens [Everhart, J. E. *et al*., 2002], with the absence of H. Pylori as negativity in whole-cell antigens, *et al*.,[29]

Spirometry

Pre- and post-bronchodilator spirometry was performed according to American Thorasic Society guidelines [28]. Normal values were obtained from Hankinson, *et al*., [29].

STATISTICAL METHOD

The T-test was used for comparisons of categorical variables. In multivariable analyses, generalized estimating equations (GEE) were used to confirm the findings because of the presence of matched sets of individuals that occurred when asthma cases referred family members to the study.

RESULTS

Characteristics of the study groups Characteristics of the asthma cases and non-asthma controls are shown in Table 1. Cases and controls were similar in age and gender. Lung function parameters, include the post-bronchodilator FEVI and post-bronchodilator FVC, were reduced in the cases compared to controls. These characteristics are consistent with expectations for an asthma population compared to a control population.

Table (1): - Characteristic of the Case-controlled Study population

Characteristics	Asthma Cases No.	Controls No.
Age - Year	34 (18-64)	38 (18-64)
Sex		
Male	24	15
Female	40	25
Spirometry- % predicted		
POST bd FEV1	85 (75-97)	92 (85-101)
POSTbd FVC	88 (8-98)	91 (82-100)
POST bd FEV1/FVC	81 (75-85)	85 (81-87)

Table (2): - The paired t-test between spirometry of controls andasthmatics

	Samplesize (n)	Mean	Standarddeviation	Standarderror of mean	T. value	P-value
Control	40	83.45	2.136	0.551	-	-
Asthmatic	64	80.08	3.144	0.393	-5.547	<0.001*

*P-value < 0,05 is significant.

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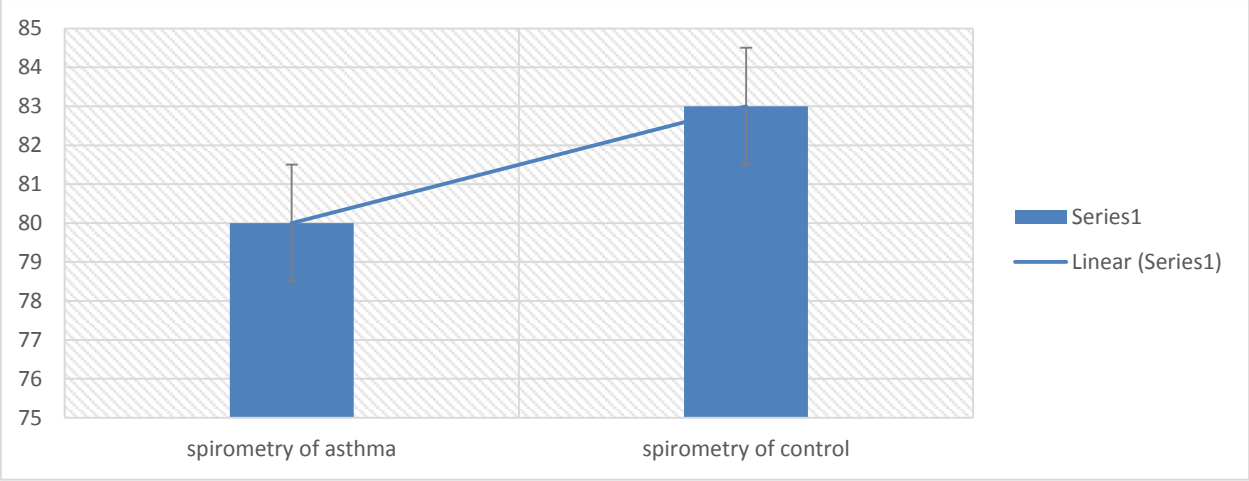

Fig 2: Spirometry of asthmatic versus control

DISCUSSION

Reduced exposure to orofecal organisms has been suggested as an explanation for the increasing prevalence of atopy and asthma, but studies of multiple organisms have had conflicting results [Suerbaum, S. *et al*., 2002].

H. pylori seroprevalence was common in both cases and controls; in accordance with national estimates [33], the analysis identified a trend toward an inverse association between H. pylori and asthma that became significant when we examined individuals who carried IgG + pylori strains.

Our data provide evidence that asthma is inversely associated with serologic evidence of the presence of IgG+ H. pylori strains, we used several different analytical approaches, and analytical results were highly consistent.

Our findings support and expand upon our recent cross-sectional study of separate populations of individuals with an asthma diagnosis in the NHANES III and NHANES IV populations [Kuipers, E. J. et al., 1995] and now include subjects with spirometry.

Our analysis of the age of disease onset in an adult population, in which we detect a delay on the onset of asthma in IgG+ individuals, is consistent with the age relationships reported in the two NHANES populations. Interestingly, although we detected a delay in the onset of asthma associated with the presence of IgG serology, FEV1 was reduced in this group. The finding of reduced lung function in individuals with a later age of onset of asthma is consistent with those recently reported (34-361)

Potential mechanisms by which H. pylori could aster asthma presentation include immune modifications or an effect on gastroesophageal reflux disease (GERD). Although H. pylori colonization recruit's neutrophils and lymphocytes and macrophages to the stomach [Suerbaum, S. et al., 2002], H. pylori generally persists for the host's lifetime [Everhart, J. E. et al., 2002], reflecting immune evasion and modification of host inflammatory. Innate and adaptive immune responses [Dooley, C. P. et al., 1989; Blaser, M. J. et al., 2004, 38,39].

H. pylori may alter the polarized Th1/Th2 T cell response through dendritic cell-mediated T-cell expression of IL-12, TNF-a, and IFN-e [40-42]. H. pylori colonization induces regulatory T cells, including CD4+CD25 T cells that express the forkhead box P3 transcription factor (Foxp3) [Thjodleifsson, B. et al., 2007] and also induces indoleamine 2,3- dioxygenase, mechanisms that suppress T cell function IL-10 expression is increased in the gastric mucosa of children carrying H. pylori Such immunosuppressive and immunomodulatory effects have the potential to alter the effector phase of asthma, as recently shown in murine models of helminth infection.

Helicobacter pylori upregulates Toll-like receptor 4 (TLR4), and although results are complex, human and murine studies suggest that activation of TLR4 may be protective against allergic asthma [50].

Associations between GERD and asthma also are well-established [51-53]. Longitudinal studies show that asthma is a risk factor for the development of GERD. And that GERD can trigger asthma.

H. pylori, especially cagA+ strains, are inversely associated with GERD [Blaser, M. J. et al., 2004,54]. Although we did not specifically assess

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for GERD in this study, the possibility exists that the inverse association between H. pylori and asthma reflects protection from GERD.

An alternative explanation for our findings is that H. pylori seronegativity is a surrogate for other phenomena, such as the presence or absence of other indigenous biota, or merely reflects cumulative early life exposure to antibiotics, identified as a risk factor for asthma.

Although possible, the specificity of the relationship to cagA+ strains argues against this point. There are some potential limitations to this study. Although we do not know the age at which H. pylori was Acquired in the cases or controls, multiple studies have demonstrated that nearly all acquisition that occurs does so at an early age, usually before the age of five, and H. pylori antibodies reflect the present carriage of H. pylori, and its prior elimination due to antibiotic exposure could lead to seronegativity. Thus, the current serostatus could under-estimate H. pylori acquisition but not persistence, since with long-term carriage of H. pylori, antibody levels are stable; one potential confounding factor could be greater antibiotic use in asthma cases than in controls, which would bias toward consequent elimination of H. pylori. The association of inverse association with childhood-onset but not later-age onset asthma in this study (Figure 1) and two other recent studies, as well as the specificity of the effect with IgG+ positivity, argues against that point. However, prospective studies will be needed to clarify this question. Because adults were studied, the association of H. pylori with delay in asthma onset may be confounded by recall bias or delay in doctor diagnosis. Although these issues suggest the need for future prospective studies, our findings support those of our recent cross-sectional studies of NHANES populations.

Table (3): - The paired t-test between H. Pylori IgG of controls and asthmatics.

	Sample size (n)	Mean	Standard deviation	Standard error of mean	T. value	P-value
Control	40	53.98	16.566	2.619	-	-
Asthmatic	64	24.73	15.514	1.939	-7.906	<0.001*

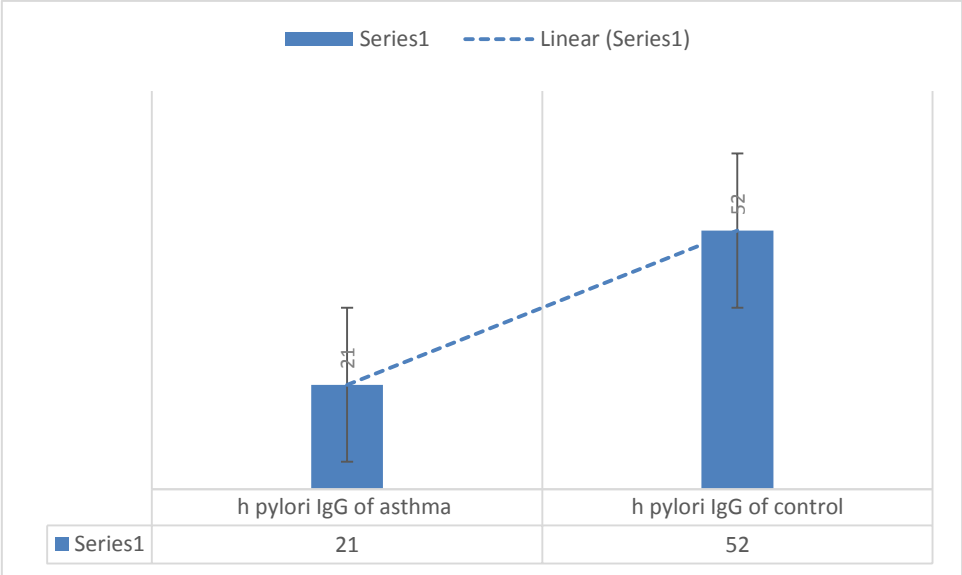

Fig 3: H. pylori IgG for Asthmatics and Control

CONCLUSIONS

Our data suggest that H. pylori and, specifically, IgG positively is inversely associated with asthma and with a delay in the onset of asthma. That the association was strongest with IgG + H. pylori strains suggests that the more intensive host interaction of these organisms may influence disease expression.

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Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Abed, H.D., Kadhim, S.J. and Asal, A.A. "Asthma is Inversely Associated with Helicobacter Pylori Status in Al-Kadhimia Teaching Center." *Sarcouncil Journal of Internal Medicine and Public Health* 3.2 (2024): pp 11-15.

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