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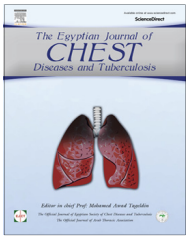

ORIGINAL ARTICLE

Study of the *Helicobacter pylori* infection in chronic obstructive pulmonary disease

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KEYWORDS

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Abstract *Background:* COPD may be associated with other systemic diseases including cardiovascular diseases, diabetes, osteoporosis and peptic ulceration and *Helicobacter pylori* infection seems to be the main cause of PUD.

Aims: The aim of this work is to study the association of *H. pylori* infection in chronic obstructive pulmonary disease (COPD).

Subject and methods: This study was performed on 80 subjects. They were classified into two groups: **Group I:** 65 COPD patients. **Group II:** 20 healthy control subjects. COPD was diagnosed according to the Global Initiative for chronic obstructive pulmonary disease admitted at chest department of Benha University Hospital.

Results: It shows that seropositivity of anti-*H. pylori* IgG and anti-CagA IgG was higher in COPD patients than in controls with a highly statistically significant difference ($p = 0.009$ and 0.047 respectively). Also the IgG level of *H. pylori* positive cases and CagA positive cases was higher in the COPD group than in the control group with a highly statistically significant difference ($p = 0.027$ and 0.0001 respectively).

Conclusion: The present study suggests that patients with COPD have an increased seroprevalence of Hp infection.

Introduction

Chronic obstructive pulmonary disease (COPD) is a common preventable and treatable disease. It is characterized by persistent airflow limitation that is usually progressive. It is associated with abnormal chronic inflammatory response to noxious particles or gases. Exacerbations and co-morbidities contribute to the overall severity in individual patients [1].

Helicobacter pylori is a slow growing, microaerophilic, gram-negative spiral shaped bacterium. It colonizes gastric mucosa and elicits both inflammatory and lifelong immune responses with release of various bacterial and cytotoxic

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substances [2]. An increased seroprevalence of *H. pylori* has also been reported in various extragastrointestinal disorders including skin, vascular, and autoimmune disorders, as well as in some respiratory diseases such as bronchial asthma, bronchiectasis, chronic bronchitis, and lung cancer [3].

COPD had been associated with gastroduodenal ulcer, many years before the identification of *H. pylori* infection as a cause of peptic ulcer disease [3]. Despite the fact that the role of *H. pylori* in pathogenesis of COPD remains controversial the activation of inflammatory mediators by *H. pylori* infection and during the course of COPD may explain the potential pathogenetic role of *H. pylori* [4]. Data in the literature on the relationship between *H. pylori* infection and chronic obstructive pulmonary disease (COPD) are poor [3].

Subjects and methods

The present study is a cohort prospective study. It was conducted in the period extending from January 2014 to January 2015 after obtaining informed patients' consent.

Subjects

This study was performed on 85 subjects. They were classified into two groups:

- **Group I** included 65 COPD patients.
- **Group II** included 20 healthy control subjects.

Inclusion criteria: COPD was diagnosed according to the Global Initiative for chronic obstructive pulmonary disease (GOLD) admitted at chest department of Benha University Hospital. Severity of COPD will be classified by spirometric data according to guidelines of Global Initiative for chronic obstructive lung disease [1]. The studied groups were complaining of dyspeptic symptoms (as abdominal pain related to meals, fullness, early satiety and nausea) or history of peptic ulcer.

Exclusion criteria: Patients with exacerbation of COPD in the preceding month, as in those cases pulmonary function does not represent baseline levels, prior *H. pylori* eradication therapy, history of taking of acid suppressive drugs or antibiotics in the preceding 6 months, and history of vagotomy or operation on the upper gastrointestinal tract.

All patients were submitted to:

(1) Full history and thorough clinical examination (stress on dyspeptic symptoms and history of peptic ulcer).
(2) Radiological examination: plain postero-anterior and lateral chest X-ray.
(3) Ventilatory function test (spirometry) before and after bronchodilatation.
(4) *H. pylori* antibody level positive detection was measured by ELISA using specific kits for anti-*H. pylori* IgG (Catalog No. E-HL G-K08) and anti-CagA *H. pylori* IgG [DIA. PRO (Diagnostic Bioprobes) SrlVia G. Carducci no 27 20099 Sesto San Giovanni (Milano)- Italy]. Purified antigens are coated to a microwell plate. Antibodies in the patient samples bind to the antigens and were determined during the second incubation step using enzyme-labeled antihuman antibodies (the conjugate). All unbound materials are removed by washing. The bound enzyme converts the colorless substrate (H_2O_2/TMB) to a blue end product.

Statistical analysis

Data obtained from the present study were computed using SPSS versions 17 under the platform of Microsoft Windows XP, Professional Edition. Continuous data were expressed in the form of mean ± SD while categorical data were expressed in the form of count and percent. Comparison of continuous data was performed utilizing student *t* test, while categorical data were done using Chi-square test. *P* value less than 0.05 was considered statistically significant.

Results

Table 1 shows a highly significant difference between them as regards smoking ($p = 0.0001$).

Table 2 shows that seropositivity of anti-*H. pylori* IgG and anti-CagA IgG were higher in COPD patients than controls with a highly statistically significant difference ($p = 0.009$ and 0.047 respectively). Also IgG level of *H. pylori* positive cases and CagA positive cases were higher in the COPD group than in the control group with a highly statistically significant difference ($p = 0.027$ and 0.0001 respectively).

Table 3 shows no statistically significant differences between COPD patients with anti-*H. pylori* +ve and −ve IgG regarding pulmonary functions.

Table 4 shows there is significantly lower FEV1 in anti-CagA +ve patients when compared with anti-CagA −ve patients with a statistically significant difference and ($p = 0.011$).

Table 5 shows the majority of anti-CagA +ve patients had a significantly higher frequency of severe disease 26 (72.2%) with a highly statistically significant difference between 2 groups ($p = 0.02$).

Table 6 shows a statistically significant correlation between anti-CagA seropositivity and both disease severity and FEV1.

Discussion

The pulmonary component of chronic obstructive pulmonary disease (COPD) is characterized by airflow limitation that is not fully reversible [5]. *H. pylori* is a major causative agent of peptic ulcer disease and a risk-factor for gastric cancer [6]. It is believed that the release of proinflammatory cytokines stimulated by *H. pylori* may play a role in chronic inflammation of bronchi. Cytotoxin-associated gene-A (CagA) is the most important virulence factor for *H. pylori* that affects

Table 1 Demographic data in COPD patients and controls.

Variables		Group (I) (COPD patients) $n = 65$	Group (II) (Controls) $n = 20$	*P* value
Age (years)		62.2 ± 9.3	60.6 ± 6.4	0.22
Gender	Male	63 (96.9%)	18 (90%)	0.31
	Female	2 (3.1%)	2 (10%)	
Smoking		50 (76.9%)	5 (25%)	0.0001*

* Means significant.