Cigarette smoking status and *Helicobacter pylori* infection in non-ulcer dyspepsia patients

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Non-ulcer dyspepsia; *H. pylori*; Smoking

**Abstract**  
*Background and objective:* There is strong, if not conclusive, evidence that *Helicobacter pylori* plays a role in duodenal ulcer disease, gastric ulcer disease, and gastric adenocarcinoma. The potential association with nonulcer dyspepsia, however, still remains unclear despite the large number of studies that have attempted to elucidate it, smoking has been found to be related to a higher incidence of *H. pylori* in non-ulcer dyspepsia patients. Therefore we aimed to find out the incidence of *H. pylori* infection in our group of non-ulcer dyspepsia patients and to study the relationship between smoking status of the patients and *H. pylori* positivity in them.

*Patient and methods:* All patients presenting with dyspepsia in whom upper gastro-intestinal endoscopy had ruled out acute or chronic peptic ulceration, esophagitis, gastric cancer, and other structural and metabolic causes were taken as patients of non-ulcer dyspepsia. Urea breath test was used for all patients. The history was reviewed; especially their smoking statuses were recorded.

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Introduction

Dyspepsia is defined as any pain, discomfort or nausea referable to the upper alimentary tract, which may be intermittent or continuous and has been present for at least one month [1]. Dyspepsia is a very common clinical problem accounting for two to three percent of primary care consultations and some 30–50 percent of cases of chronic upper abdominal complaints presented to the gastroenterologist [2]. A large proportion of these patients do not have recognizable organic disease and are commonly labeled as having functional dyspepsia. Although upper gastrointestinal endoscopy is the investigation of choice for the distinction between organic and functional dyspepsia, one must take into account that the percentage of relevant organic disease found by this invasive technique is in the range of 15–25 percent, whereby the lower limit refers to patients of the primary care setting [3,4].

Dyspepsia affects 20–40 percent of the population of the Western world [5–8]. Upper gastrointestinal endoscopy of patients with dyspepsia reveals no abnormality in the majority; such patients are considered to have non ulcer, or functional, dyspepsia [9]. The cause of non ulcer dyspepsia is unclear but is thought to be heterogeneous [10–12]. The recognition of the pathogenic role of *Helicobacter pylori* infection in patients with peptic ulcer and the benefits of eradicating the infection [13–15] have led to suggestions that the infection may also be the cause of dyspepsia in some patients with non ulcer dyspepsia. A higher prevalence of *H. pylori* infection has been reported in patients with dyspepsia than in those without it, but in some of these studies the patients were not matched for age or excluded if they had underlying ulcer disease [16–18].

Several studies have examined the effect of eradicating *H. pylori* infection on dyspeptic symptoms in patients with non ulcer dyspepsia, but the results have been conflicting and all such studies have been criticized because of design flaws [19]. In a recent review of these trials, Talley and Hunt concluded that "*H. pylori* has not been established, as yet, to play a definitive role in dyspepsia" [19].

Finally, Non-ulcer dyspepsia is one of the most frequent diagnosis made in patients who present with chronic dyspepsia. The pathogenesis remains poorly understood and little information on potential risk factors for this condition is available.

As has been previously reported by Rajashekhara et al. [20], both *H. pylori* infection and smoking are risk factors for acid peptic disorders. There is paucity of data on the relationship between smoking and *H. pylori* infection.

This study was carried out to find the incidence of *H. pylori* infection in patients presenting with non ulcer dyspepsia. Also, its relationship to the smoking status of the patients was studied.

Subjects and methods

We studied patients who presented with dyspepsia and requested for proved or excluded *H. pylori* infection using non invasive urea breath test because they had dyspepsia for at least 4 months. All patients had no endoscopic evidence of current or previous peptic ulcer disease. Dyspepsia was defined as intermittent or persistent pain or discomfort in the upper abdomen or lower part of the chest, heartburn, nausea, a feeling of postprandial fullness, or any other symptoms thought to be related to the upper gastrointestinal tract [21]. Patients were excluded if they had previously been found to have peptic ulcer disease, had endoscopic evidence of esophagitis, history or evidence of ischemic heart diseases, gallbladder diseases, were taking nonsteroidal anti-inflammatory drugs (other than low-dose aspirin), had undergone gastric resection, were pregnant, or had previously been treated for *H. pylori* infection.

At this visit, a standardized interview was used to determine each patient’s symptoms and smoking history.

After the interview and clinical assessment, the patients underwent a carbon-14 urea breath test to determine their *H. pylori* status [22], where patients swallow urea labeled with an uncommon isotope, radioactive carbon-14. In the subsequent 10–30 min, the detection of isotope-labeled carbon dioxide in exhaled breath indicates that the urea was split; this indicates that urease (the enzyme that *H. pylori* uses to metabolize urea) is present in the stomach, and hence that *H. pylori* bacteria are present.

The results of the test were considered positive if the value at 20 min was more than 30 (units equal the percentage of the dose administered per millimole of carbon dioxide expired times the body weight in kilograms times).

This study was conducted at Alborg laboratories Almadijah almunawwarah branch, Saudi Arabia, one hundred consecutive patients fulfilling criteria of non-ulcer dyspepsia searching for *H. pylori* attending Alborg lab were taken in this study.

Data collection

All the NUD patients who fulfilled the inclusion criteria underwent detailed history taking, including demographic data, result of urea breath test and the smoking history of their lifetime were analyzed by (SPSS version 17).

Results

Patients referred by their physician to the Alborg lab searching for *H. pylori* were enrolled into our non ulcer dyspepsia study if they met the our study criteria.
Cigarette Smoking Status and Helicobacter Pylori Infection in Non-Ulcer Dyspepsia Patients

We analyzed 100 of non-ulcer dyspepsia patient’s data. 58 patients (58%) were males and 42 patients (42%) were females. The age ranged from 15 to 75 years with a mean of 37.5 years (Table 1).

Nonparametric data were given as absolute numbers or percentages (Table 2).

The population—both genders individually and pooled—was stratified into sex subgroups: (Table 2, Fig. 1):

1. Patients who did not smoke and had a negative result for *H. pylori* by non invasive urea breath test.
2. Patients who did smoke and had a negative result for *H. pylori* by non invasive urea breath test.
3. Patients who were ex smokers and had a negative result for *H. pylori* by non invasive urea breath test.
4. Patients who did not smoke and had a positive result for *H. pylori* by non invasive urea breath test.
5. Patients who did smoke and had a positive result for *H. pylori* by non invasive urea breath test.
6. Patients who were ex smokers and had a positive result for *H. pylori* by non invasive urea breath test.

**Smoking and urea breath test**

The smoking habits were analyzed (Table 1, Fig. 1). Smokers were 11, out of which 7 (63.64%) had *H. pylori* positive result, among non-smokers 29 out of which 65 (44.62%) had *H. pylori* positive result, and among ex-smokers 7 out of which 24 (29.17%) had *H. pylori* positive result. The difference in incidence of *H. pylori* infection among smokers with NUD, non-smokers with NUD and ex smokers with NUD did not match with *P* values > 0.1.

**Discussion**

Dyspepsia is remarkably common in the general population. Only a minority of subjects with dyspepsia seek medical care, or in other words become patients. While dyspepsia and non-ulcer dyspepsia are common conditions, the natural history remains relatively poorly defined. There is a turnover of symptoms in the population, leading to a stable prevalence by a similar number of other subjects who experience the symptoms in the population, resulting in a stable prevalence from year to year [23].

In the present study, we explored the probable interaction between of smoking status and *H. pylori* positivity in non-ulcer dyspepsia patients and to study the relationship between in the middle-aged and elderly population with non-ulcer dyspepsia.

In our study we found that the incidence of *H. pylori* positivity was 43% and there was no statistically significant difference between smokers, non-smokers and ex smoker in the positivity rate of *H. pylori* and these agree with the study of Sharma et al. [24] where they found that the incidence of *H. pylori* positivity was 59% and there was no statistically significant difference between smokers and non-smokers in the positivity rate of *H. pylori* but findings were contrary to the findings of Rajashekhar et al. [20] who had found an association of smoking with *H. pylori* positivity in NUD patients.

In our study we found that 36 out of one hundred non ulcer dyspepsia (36%) patients were non smokers and proved negative for urea breath test. There are only limited data from the literature available to show to what degree of the cigarette smoking with *H. pylori* incidence as risk factors in non-ulcer dyspepsia patients and can be considered as guidelines in helping the primary care physician to preselect patients at risk for organic and non organic dyspepsia with a reasonable confidence, and thus to help reduce unnecessary endoscopies. There is a considerable overlap for each factor between patients suffering from organic and functional dyspepsia.

It is of particular interest to evaluate the additive discriminate power of more than one risk factor. It is the aim of this review paper to discuss the results of a study of our group, where these parameters were analyzed, and to compare them with the few published sampled data.

The cause and pathophysiology of non-ulcer dyspepsia remain inadequately defined. It is likely to be a multi-factorial condition. However, new pathophysiological information has come to light in recent years that has provoked changes in patient management. Various causes like gastric acid hypersecretion, abnormal gastric sensations, disturbed gastric emptying, abnormal relaxation of gastric fundus, psychological stress, diet, smoking, alcohol, etc., have been implicated with varying results in different studies. One important factor which has been widely studied is *H. pylori* status in NUD. A major role for *H. pylori* gastritis in non-ulcer dyspepsia (NUD) is controversial. Gastro-duodenal dysfunction may be associated with *H. pylori* infection, but there is little evidence for a causal link with dyspepsia. Population-based studies with appropriate methodology have generally failed to confirm an association between *H. pylori* and NUD. Furthermore, no definite association between sub-groups of NUD (ulcer-like, dysmotility-like, reflux-like, and non-specific) and *H. pylori* has been identified. However, the sub-groups have been defined, and no specific symptom pattern characterize patients with *H. pylori* infection. Whether *H. pylori*-induced alterations of gastric

<table>
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<tr>
<th>Table 1</th>
<th>Distribution of all (100) patients according to age, gender, smoking habit and urea breath test for <em>H. pylori</em>.</th>
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<tbody>
<tr>
<td>Urea breath test for <em>H. pylori</em></td>
<td>Positive</td>
</tr>
<tr>
<td>Smoking</td>
<td>Non smokers</td>
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<tr>
<td></td>
<td>Ex smokers</td>
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<td>Age</td>
<td>Male only</td>
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physiology can explain NUD remains open to debate while we await the results of more specific experiments [25]. In a cross-sectional survey of 1060 consecutive patients presenting with dyspepsia at the Endoscopic Unit, University Hospital, Kuala Lumpur, Malaysia, the 

**H. pylori** prevalence in non-ulcer dyspepsia (NUD) was 31.2%. The prevalence among the races was as follows: Malay 16.4%; Chinese 48.5%; and Indians 61.8% [19]. Thus, 

**H. pylori** infection (and the associated histological gastritis) occurs in up to 50% of patients with non-ulcer dyspepsia, although the prevalence depends on age, socioeconomic status, and ethnic background [25].

**H. pylori** infection is more common in smokers with NUD than in non-smokers. However duration and amount of smoking have no relationship with 

**H. pylori** positivity [26].

### Limitations of the study

Several limitations must be recognized when interpreting the results of this study. First, this was a cross-sectional analyses, and therefore conclusions about causal associations cannot be made. We intended to assess the relationship of cigarette smoking with 

**H. pylori** incidence in non-ulcer dyspepsia patients, but we found that all the female groups were non-smokers in our study done in Almadinah almunawwarah, Saudia Arabia, where females were usually non-smokers according to their environmental and religion aspects. Second, It is a non selected analytical study so the number of smokers, non-smokers and ex smokers did not match. The strengths of this study are (1) the data are from a well-designed representative survey; (2) the laboratory measurements are validated and reliable; and (3) we have adjusted for two of known risk factors including 

**H. pylori** infection and cigarette smoking.

### Conclusion

In our study we found that the incidence of **H. pylori** positivity was 43% and there was no statistically significant difference between smokers, non-smokers and ex smokers in the positivity rate of 

**H. pylori** in NUD patients.

### Recommendation

Longitudinal research is needed to be done in a large number of population samples with matching groups according to age, sex, and smoking habit to study the sensitivity and specificity of different laboratory methods using for the detection of 

**H. pylori** in non ulcer dyspepsia patients.

### Conflict of interest statement

We declare that we have no conflict of interest.

### References

