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Original Article

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A Study to Analyse Association of H.Pylori Infections in Patients of Obstructive Sleep Apnea (OSA) Presenting with Symptoms of Gerd

Dr Munis Zehra[1]; Prof SF Haque[2]; Dr S.Haider MehdiHusaini[3]; Prof. Kafil Akhtar[4]; Prof M.Shameem[5]

[1] Junior resident , Department of Medicine
[2] Professor Department of Medicine
[3] Assistant Professor Department of Medicine
[4] Professor Department of Pathology
[5] Professor Department of TBRD

ABSTRACT

Introduction: Several studies identified a link between gastroesophageal reflux disease (GERD) andobstructive sleep apnea (OSA). GERD is a condition in which acid reflux from the stomach to theoesophaguscauses troublesome symptoms. On the other hand, OSA is defined as a sleep-related breathing disorder inwhich airflow significantly decreases or ceases due to upper airway obstruction, leading to arousal fromsleep. OSA was found to be associated with GERD. In this study ,we aim to analyse the association of H.pylori infection in patients of obstructive sleep apnea presenting with symptoms of GERD .

Methods:40 patients with symptoms of obstructive sleep apnea and gastroesophageal reflux diseases were enrolled . They underwent polysomnography and esophageogastroduodenoscopy .biopsy samples were taken and sent for histopathological examination with giemsa stain .

Results: Out of 40 patients, 34 patients have giemsa positive h.pylori gastritis on biopsy (p=0.001)prevalence of h.pylori infection increases with severity of obstructive sleep apnea. Inmoderate osa (17.65%) , severe osa (82.35%)(p=0.004)

Conclusion: This study concludes that there is significant association of h.pylori infection with OSA presenting with symptoms of GERD. Also, prevalence of h. pylori infection increases with severity of OSA.

Key Words: *H.Pylori Infections;*

***Corresponding Author**
Dr Munis Zehra
Junior resident , Department of Medicine

INTRODUCTION

Obstructive sleep apnea/hypopnea syndrome (OSAHS) is characterized by recurrent episodes of partial or complete upper airway collapse during sleep which is highlighted by a reduction in, or complete cessation of airflow despite ongoing respiratory efforts. An Apnea Hypopnea index≥5 events/hr is commonly used to define OSA, with obstructive or mixed (rather than central) events comprising more than 50% of the total with persistent complaints of excessive daytime somnolence, unrefreshing sleep or fatigue[1]. Based on AHI, OSA is categorized as Mild(5-15 events/hour),Moderate (15-30 events/hour),Severe(>30 events/hour).Risk factors for the development of OSA are obesity, male sex, age, alcohol use, adenotonsillar hypertrophy, and craniofacial abnormalities. Besides its best-known manifestations, which include snoring and excessive daytime somnolence, OSA induces several chronic health concerns related to the cardiovascular, cerebrovascular, pulmonary, digestive, metabolic, autonomic nervous and psycho-physiological systems. Many previous studies have demonstrated a higher prevalence of gastroesophageal reflux disease (GERD) among patients with OSA[2,3].

Worldwide, Helicobacter pylori infection (Hp-I) is very common. H. pylori seroprevalence has been observed to be higher in OSAHS despite its high prevalence [4,5]. This is due to inflammation produced by H.pylori which has impact on autonomic reflexes, upper airway collapsibility, and lead to inspiratory pharyngeal muscle dysfunction[6].

Few studies suggested that H. pylori infection can make GERD worse[7–9]. The severity of GERD may increase or decrease depending on whether H. pylori infection increases or decreases stomach acid secretion[10].

AIMS AND OBJECTIVES

1. To study the prevalence of H.pylori infection in patients of Obstructive sleep apnea having symptoms of GERD.

2. To establish a co-relation between H.pylori infection and OSA.
3. To establish a co-relation between H.pylori infection and severity of OSA.

MATERIALS AND METHODS

This observational study was conducted at the Medicine OPD, Gastroenterology OPD and Endoscopy Lab, Department of Medicine and Histopathology Lab, Department of Pathology, of tertiary care centre in North India. The study was conducted between December 2020 to November 2022 after approval from Board of Studies and Institutional Ethics Committee, consisting of 40 subjects who presented to us with symptoms of OSA and GERD. After taking written informed consent, Polysomnography of the recruited subjects was done, and subjects who had moderate and severe OSA underwent esophagogastroduodenoscopy (EGD). During EGD rapid urease test was done, biopsy samples were taken and were sent for histopathological examination.

Inclusion criteria

- Age > 18
- Patients with diagnosed OSA on Polysomnography (AHI index > 15)
- Patients of OSA having symptoms persistent with GERD

Exclusion criteria

- Patient not givingconsent.
- Consumption of acid suppressive drugs or antibiotic in preceding 6 months.
- History of vagotomy or operation of Upper GI tract.

OBSERVATIONS AND RESULTS

Out of 40 subjects, 24 were male (60%), 16 were female (40%).In study subjects, 10 cases were in age group 18-39 years (25%), 23 cases were of age group 40-59years (57.5%), 7 cases were ≥60 years (17.5%).In our study most of the patients belong to age group 40-59years.this data is shown in Table 1.

Table 1: Showing Age Distribution

S.No.	Age Group	No. of Cases	(%)
1	18-39 Year	10	25
2	40-59 Year	23	57.5
3	≥ 60 Year	7	17.5
4	Total	40	100

Out of 40 patients, 34 were RUT positive (85%), 6 were negative (15%).it is shown in table 2.

Table 2: Showing distribution of RUT in study subjects

Rapid urease test	Frequency	Percentage
Negative	6	15%
Positive	34	85%
Total	40	100.00%

Out of 40 patients, 4 patients had oesophagitis (10%), 25 patients had antral gastritis (62.5%), 9 patients had pangastritis (22.5%), 2 patients had duodenitis (5%), hiatus hernia is present in 8 patients (20%).It is shown in Figure 1.

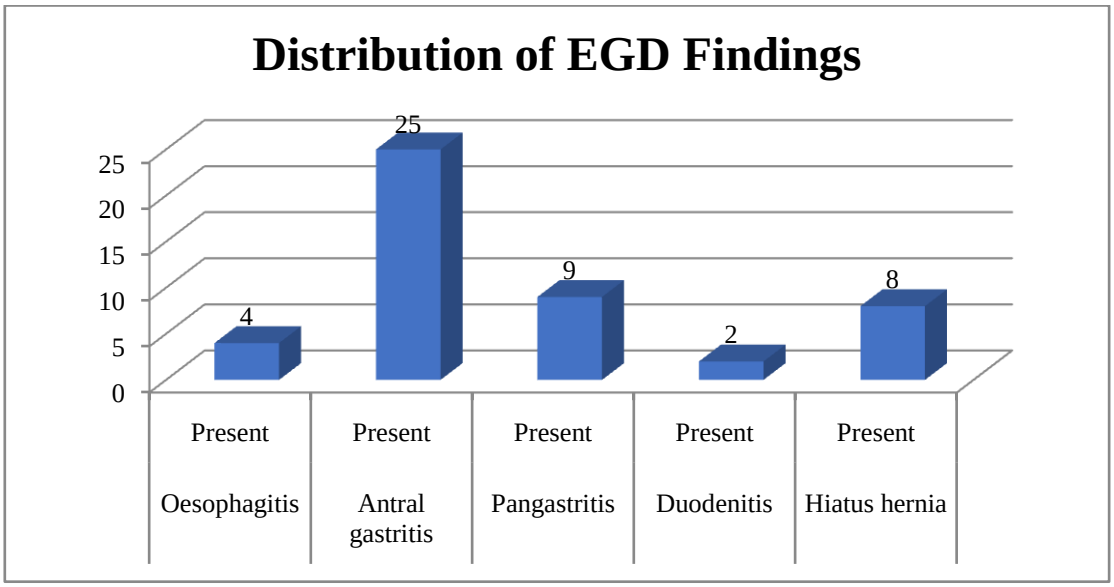


Figure 1: Showing Distribution of EGD findings in study subjects

In our study, 34(85%) patient were giemsa positive and all of them have H. Pylori induced gastritis, 6 patients were giemsa negative (15%), out of which 2 patients (5%) have nonspecific duodenitis, 4 patients have non specific gastritis (10%). It is shown in table 3.

Table 3 -showing distribution of biopsy findings of study subjects:

Giemsa Negative		Giemsa Positive
6 (15%)		34 (85%)
Nonspecific Duodenitis	**Nonspecific Gastritis**	**H.pylori induced Gastritis**
2 (5%)	4 (10%)	34 (85%)

In H.pylori positive group, 24 patients had BMI ≥30 (70.59%), 7 patients had BMI of 25-29.9 (20.59%), 3 patients had BMI of <25.(8.82%)

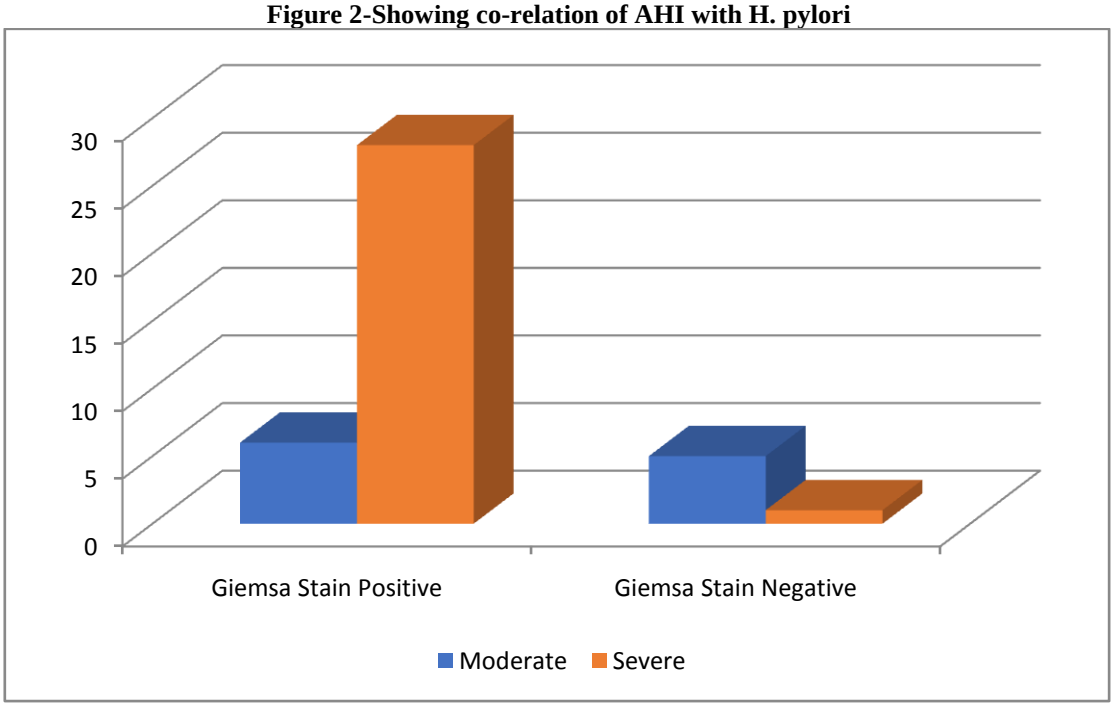
In H.pylori negative group, 3 patients had BMI of 25-29.9 (50%) , 3 patients had BMI <25 (50%) These results are statistically significant (p-value – 0.001)(spearman co-relation coefficient -0.483).It is shown in table 4.

Table 4-Showing distribution of BMI in H.pylori positive and negative groups

S.No.	BMI	Biopsy Findings						Spearman co-relation coefficient	P- Value
		Giemsa Stain Positive		Giemsa Stain Negative		Total			
		Cases	(%)	Cases	(%)	Cases	(%)		
1	< 25	3	8.82	3	50.00	6	15	0.483	0.001
2	25.0-29.9	7	20.59	3	50.00	10	25		
3	≥ 30	24	70.59	0	0.00	24	60		
4	Total	34	100.00	6	100.00	40	100		

In this study, out of 34H. pylori positive patients 28 had severe OSA (82.35%), 6 had moderate OSA (17.65%). In 6 H. pylori negative patients, 1 patient had severe OSA (16.67%), 5 patients had moderate OSA (83.33%). These results are statistically significant (p-value -0.004) (spearman co-relation coefficient -0.458).It is shown in figure 2 .

Figure 2-Showing co-relation of AHI with H. pylori



DISCUSSION

Data in the literature regarding H.pylori and OSA relationship is poor . Main finding of our study is that out of 40 recruited subjects of OSA with symptoms of GERD 34 are H .pylori positive (85%) , 6 are negative (15%) .Also the prevalence of H.pylori increases with the severity of OSA i.e. there is increased prevalence of H.pylori in severe OSA than in moderate OSA .These findings are in accordance with the findings of ***Ye et al 2008*** ,a cross sectional study on Chinese population recruited 236 subjects and found that H. pylori infection was more common in patients with OSAS (75.5%) than in controls (53.4%) and there was increased seroprevalence of H .pylori with increase in OSA severity .Regarding the etiopathogenetic role of H.pylori in OSA , H.pylori strains stimulate the host immune response and release of inflammatory cytokines like IL-1 (interleukin -1) , IL-8, TNF-α(tumor necrosis factor -alpha) [11,12] H.pylori can cause systemic inflammation .Inflammation impactautonomic reflexes, upper airway collapsibility, and inspiratory pharyngeal muscle dysfunction. Theseunfavorable processes may increase the severity of OSA .Another pathogenetic mechanism could be the spilling and inhalation of H.pylori and its exotoxins into the respiratory tract .

In our study, we found there is increased prevalence of H.pylori in OSA patients with symptoms of GERD . A few studies, however, suggested that H. pylori infection can make GERD worse[7–9]. H. pylori may result in (a) antral gastritis, which increases acid output and worsens GERD,(b) corpus or pangastritis, which causes milder GERD by reducing acid secretion[13]. Therefore, the site of infection affects the severity of GERD in people with H. pylori infection. This explanation could explain our findings as in our study most of the patients have antral gastritis.

There are a few studies about seroprevalence of *H.pylori*in patients with OSAS. These studies revealed that seroprevalence of *H.pylori* among patients with OSAS was higher thannormal subjects [17,18]. To the best of our knowledge, there is no study which studied the prevalence of H.pylori in OSA patients using gold standard test i.e. . biopsy with histopathological examination. ours is the first study.

Limitations of the study –

The study population was small. Our study is a single center study, and hence, the results cannot be generalized to general population; necessitating the need of further large-scale studies.

CONCLUSION AND SUMMARY

Our study suggest that there is increased prevalence of H.pylori in patients of OSA presenting with symptoms of GERD. Prevalence of H.pylori infection increases with severity of OSA .Our findings should be confirmed in a multicentric study with large number of participants. Further studies should be done to see the effect of H.pylori eradication on severity of OSA to make the pathogenetic mechanism behind the association more clear and to confirm our observed results.

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